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# Sanitation and Disease

## Health Aspects of Excreta and Wastewater Management

**Richard G. Feachem · David J. Bradley · Hemda Garelick · D. Duncan Mara**



**World Bank Studies in Water Supply and Sanitation 3**

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## *Health Aspects of Excreta and Wastewater Management*

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# Preface

MANY IMPORTANT infectious diseases are associated with human excreta. The most common association is that the pathogens causing the disease leave an infected person by way of the feces or urine. Excreta are thus the direct source of these infections. Less commonly and directly, excreta may be associated with the breeding of insects that are vectors of disease. The hygienic management and disposal of human excreta is thus of central importance in the control of these associated diseases. This is true in both poor and rich countries and across all climatic zones.

Most people in the developing countries do not have adequate disposal systems for human wastes. A survey of developing countries by the World Health Organization (WHO) in 1975 indicated that 75 percent of urban dwellers did not have sewerage (that is, sewers for disposal of excreta) and that 25 percent had no disposal system of any kind. In rural areas, 85 percent lacked any adequate excreta disposal facility. Major national and international initiatives are clearly required if any substantial improvement in sanitation systems in the developing world is to be made in the next few decades.

In the more wealthy and industrialized countries, most people have adequate excreta disposal arrangements in their homes and places of work. The treatment and disposal of human wastes, however, pose enormous problems for the responsible agencies. Large cities produce such volumes of sewage and such quantities of sludge that the infrastructure for the safe disposal of these wastes may be stretched to the limit. It is in this context that decisions about pathogen destruction in sewage and sludge and about the risks to public health of various treatment and disposal options become of the utmost importance.

In all countries, public health is of central importance in the design and implementation of excreta disposal projects, and better health is the main social and economic benefit that planners and economists hope to gain by investing in excreta disposal systems. To achieve this gain as much information as possible is needed about the interactions between excreta and health—information not only about broad epidemiological issues of disease prevention through improved excreta disposal, but also about the effect of particular excreta disposal and reuse technologies on the survival and dissemination of particular pathogens.

## Scope and Organization

This book sets out to provide such information for a broad readership. It is intended for the wide spectrum of professionals concerned with sanitation and public health: those who control—such as health planners, economists, and public health administrators; those who implement—such as environmental hygienists, sanitary engineers, public health workers, and health educators; and those who study and advise—especially epidemiologists, microbiologists, and parasitologists. The book has been written with a minimum of technical jargon so that it can be readily absorbed by people from different professional backgrounds; technical terms are defined when they are used, and acronyms and abbreviations are listed on page 22.

The book has two parts. Part One, entitled "The Health Hazards of Excreta: Theory and Control," presents a distillation of available knowledge about excreta, night soil, and sewage and their effects on health. The emphasis is on presenting the complex, and sometimes contradictory, evidence as clearly and concisely as possible. The source for Part One is largely, but not entirely, the literature. On occasion, we have gone beyond the literature to state what we anticipate to be the case; this theoretical content is based on a fundamental understanding of the particular disease or pathogen. Inevitably, the need for

clarity and the demands of limited space have necessitated some oversimplification.

Part Two, entitled "Environmental Biology and Epidemiology of Specific Excreted Pathogens," contains twenty eight chapters, each describing the environmental properties of a specific excreted pathogen or group of excreted pathogens and the epidemiology and control of the infections these pathogens cause. Emphasis is placed on the occurrence and survival of the pathogen in the environment and on the efficacy of various waste treatment processes in reducing or eliminating the pathogen. For ease of reference, the chapters of Part Two are grouped by biological class of pathogen in five sections—the excreted viruses, bacteria, protozoa, and helminths, and the excreta-related insect vectors of disease. As in Part One, the material in Part Two is derived from the literature. Where documentation is ambiguous or contradictory, we have attempted to give a conservative opinion—overestimating, for example, the ability of a pathogen to survive hostile environmental conditions.

Each chapter in Parts One and Two has its own list of literature cited. The several hundred papers and publications cited were selected from among a total collection of several thousand items assembled during the writing of this book. The literature searches for the various chapters were ended between late 1980 and mid-1981. The literature throughout has been selected from international sources (a considerable number of Czech, French, German, Japanese, Korean, Russian, Spanish, and other non-English language publications have been used).

Despite its division into two parts, the book is meant to be used as a unit. Readers desiring elaboration or support of statements made in Part One must refer to Part Two.

## Origins and Related Publications

This book arises out of a World Bank research project in appropriate technology for waste disposal that was initiated in 1976 by the Bank's chief water and wastes adviser, Mr. John M. Kalbermatten. The results of this research are published in three books, under the series title "World Bank Studies in Water Supply and Sanitation." Numbers 1 and 2 of this series were published in 1982 by the Johns Hopkins University Press and are entitled *Appropriate Sanitation Alternatives: A Technical and Economic Appraisal* (by J. M. Kalbermatten, D. S. Julius, and C. G. Gunnerson) and *Appropriate Sanitation Alternatives: A Planning and Design Manual* (by J. M. Kalbermatten, D. S. Julius, C. G. Gunnerson, and D. D. Mara). In addition, the Transportation and Water Department of the World Bank issues a series of reports—under the main title *Appropriate Technology for Water Supply and Sanitation*—available from the Bank's Publication Unit (for information on obtaining these and related World Bank publications, see the last page of this book). Twelve reports have been published in this series so far.

## Contributors

The book has been pepared by the Ross Institute of Tropical Hygiene from the work of a group of bacteriologists, engineers, entomologists, epidemiologists, parasitologists, and virologists from the London School of Hygiene and Tropical Medicine and elsewhere. Contributing specialists, their affiliations, and the chapters to which they contributed are:


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* Sadly, Dr. Donald Mackay died while this book was going to press. He made a substantial contribution to it, and we trust that he would have been pleased by the final result.

The book evolved, the group of contributors was convened, and the chapters on specific diseases initially each comprised a short general account followed by abstracted references. Following the review of this draft, Dr. Feachem rewrote these chapters, incorporating additional material, to form a continuous account. The chapters in their final form were then reviewed by their original authors.

## Acknowledgments

This review of sanitation and disease was originally commissioned by The World Bank, and the Bank covered a proportion of the costs involved in the preparation of the manuscript. We are indebted to the Bank's staff for their support and encouragement. We particularly acknowledge the assistance of Mr. John Kalbermatten, Mr. Charles Gunnerson, Dr. DeAnne Julius, and Mr. Richard Middleton. The Publications Department of The World Bank was instrumental in guiding this book from its first manuscript in 1978 to its final manuscript in 1981, and so to the printed book in 1983. We are especially indebted to Mr. James McEuen for his major contribution to the structure and content of the book. Jamie Cameron, Ian McIntosh, Lyn Udall and their colleagues at John Wiley & Sons, UK, ensured the rapid and efficient publication of the final manuscript.

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# Acronyms and Abbreviations

| | |
|---|---|
| BOD | Biochemical oxygen demand |
| $BOD_5$ | Biochemical oxygen demand by the standard test (5 days at 20°C) |
| °C | Degrees Celsius |
| DNA | Deoxyribonucleic acid |
| DVC | Double-vault composting [toilet] |
| EIEC | Enteroinvasive *Escherichia coli* |
| ELISA | Enzyme-linked immunosorbent assay |
| ENT | *E. coli* enterotoxin plasmids |
| EPEC | Enteropathogenic *E. coli* |
| ETEC | Enterotoxigenic *E. coli* |
| $ID_{50}$ | Median infective dose |
| IEM | Immuno-electronmicroscopy |
| LT | Heat-labile [*E. coli* enterotoxin] |
| PFU | Plaque-forming unit |
| PVC | Polyvinyl chloride |
| RNA | Ribonucleic acid |
| ROEC | Reed Odorless Earth Closet |
| ST | Heat-stable [*E. coli* enterotoxin] |
| $t_{90}$ | Time at which 90 percent reduction [in number of excreted pathogens] is achieved |
| TAB | *Salmonella typhi* and *S. paratyphi* A and B [vaccine] |
| $TCID_{50}$ | Median tissue culture infective dose |
| VIP | Ventilated improved pit [latrine] |
| WHO | World Health Organization |

# *Part One*

# *The Health Hazards of Excreta: Theory and Control*

# 1

# Elements and Health Risks of Excreta and Wastewater

IN THIS OPENING CHAPTER the nature and health risks of excreta, sewage and sullage are examined. Attention is given to both the composition and likely pathogen content of human wastes, the quantities of excreta and sullage produced in different countries of the world, and the hazards posed to public health by the microbes, parasites, and insects implicated in the spread of excreta-related human infections.

## Excreta and Health

This book is about human excreta and disease. Excreta are defined here as human feces and urine. Many infections, in excess of fifty even if the different numbered types of viruses and serotypes of enteric bacteria are ignored, are transmitted from the excreta of an infected person to the mouth of another. The disease-causing agents (the pathogens) of these infections travel from anus (or, rarely, bladder) to mouth by a variety of routes—sometimes directly on contaminated fingers and sometimes on food, utensils, in water, or by any other route which allows minute amounts of infected excreta to be ingested. Some of these pathogens may reinfect, not only through the mouth, but by inhalation of dust or aerosol droplets. There are also a few infections (notably hookworms and schistosomiasis) that can penetrate through the skin.

Human excreta are the principal vehicle for the transmission and spread of a wide range of communicable diseases. Some of these diseases rank among the chief causes of sickness and death in societies where poverty and malnutrition are ubiquitous. Diarrheas, for instance, are—together with malnutrition, respiratory disease and endemic malaria—the main causes of death among small children and infants in developing countries. Cholera, whether endemic or epidemic in form, is accompanied by numerous deaths in all age groups—although under endemic conditions, it is children who suffer the most fatalities. Other diseases, such as hookworm infection and schistosomiasis, cause chronic debilitating conditions that impair the quality of life (however defined) and make the individuals more liable to die from superimposed acute infections.

These diseases, and the many others discussed in this book, start their journey from an infected individual to a new victim when the causative agent is passed in the excreta. Therefore the collection, transport, treatment, and disposal of human excreta are of the utmost importance in the protection of the health of any community. They become even more important in those societies which recognize the value of human excreta in agriculture, aquaculture or gas production and therefore reuse, rather than dispose of, their raw or treated wastes. Such reuse systems have a positive role in supporting economic activity and food production and are often cheaper than alternative methods of disposal. However, reuse systems present a challenge to the public health engineer to design and develop technologies that will not pose unacceptable risks to health.

Around the world, and in most countries, there are millions of people who lack any hygienic and acceptable method of excreta disposal. There are also governments and international agencies spending, or preparing to spend, large sums of money to improve this situation. If these governments and agencies could arrange, by massive investment and miraculous social and economic transformation, that everyone be provided with a modern house with water and sewerage connections, the health dimensions discussed in this book would be less relevant. But change will not

come in this way. Change will come slowly and unevenly, and resources of money, manpower, and institutions will often be very scarce. The recipients of new excreta disposal technologies may be unable to pay completely for them, or they may lack the necessary experience and education to use them effectively. Always there will be many constraints, and with these constraints will come difficult choices.

Choices need to be made about all aspects of excreta disposal. There will be choices about technology, about ultimate disposal, about reuse, about sullage, about payment, about management, and about all the other elements that make up a sanitation system. A number of factors will influence these choices, but one central factor is *health*. Since a primary motivation for investing in excreta disposal is improved health, decisionmakers will need to understand the health implications of the various choices. The more limited are the resources, the more difficult the choices and decisions become—and the more it is necessary to understand precisely and in detail the relationships between excreta and health.

## Characteristics of Excreta and Sewage

Feces not only are malodorous and considered esthetically offensive in most societies, but they may contain an array of pathogenic viruses, bacteria, cysts of protozoa, and eggs of helminths (the collective term for worms parasitic to man) that may cause disease in a new host. Feces are therefore the beginning of the transmission routes of the diseases considered in this book; the objective of improving excreta disposal facilities is to intercept these routes at their point of origin.

### *Quantities*

There are marked differences in the volumes of excreta and sewage produced by different communities. Volume, composition, and consistency of feces depend on such factors as diet, climate, and state of health. Individual wet fecal weights vary from under 20 grams per day to 1.5 kilograms per day. When national or regional averages are considered, however, Europeans and North Americans produce daily between 100 and 200 grams, whereas people in developing countries have average daily wet fecal weights of 130–520 grams. Vegetarians generally have higher fecal weights than other groups, and fecal weights in rural areas are higher than in towns. Children, adolescents, and the elderly produce lower fecal weights than others. The data in table 1-1 show wet fecal weights reported by various authors from several countries.

The water content of feces varies with fecal weight. In a community with an average wet fecal weight of 100–150 grams per day, for instance, the water content will be around 75 percent. As fecal weight increases, so does the proportion of water: at a fecal weight of 500 grams per day, the water content of the stool may be about 90 percent. The frequency of defecation also varies with fecal weight. In Europe and North America, where fecal weights are generally under 200 grams per day, the average frequency is one stool daily. In rural areas of developing countries, especially where diet is vegetarian and fecal weights are high, a daily frequency of two or three stools is common.

Most adults produce between 1.0 and 1.3 kilograms of urine per day, but this depends on how much they drink and sweat, and this—as with fecal output—in turn depends on diet, occupation, climate, and other factors. If possible, local data should be consulted in designing a night soil system. In the absence of such data, a working assumption in a developing country is that adults will produce daily about 350 grams of feces and 1.2 kilograms of urine in rural areas, and 250 grams of feces and 1.2 kilograms of urine in urban areas.

Volumes of night soil produced for cartage and treatment may be computed from the sum of the per capita contribution of feces and urine plus any water used for ablution or for cleaning the toilet area. Daily night soil volumes are typically in the range of 1.5–2.0 liters per capita. Data from Kiangsu Province, China, show that a bucket-latrine system produces 2 liters of waste per capita daily, including the bucket wash water (McGarry and Stainforth 1978).

Volumes of domestic sewage depend on quantities of water used in the home. Houses connected to sewers must also be connected to water systems and usually have comprehensive plumbing fittings. Such houses may, rarely, use as little as 30 liters per capita daily (White 1977). If daily use falls below 50 liters per capita, however, the sewers can lose their self-cleansing flow and become blocked. At the other extreme, households with many water-using appliances (such as washing machines and dishwashers) may use 300 or more liters per capita daily.

The consistency or solids content of night soil may be calculated from these figures. Assume a daily fecal weight of 250 grams per capita, with a water content of 80 percent. Further assume a daily urine production of 1.2 liters plus 0.35 liters of water for anal cleansing per capita. The night soil of one individual then, will contain 50 grams of solids in 1.8 liters of night soil; in

Table 1-1. *Fecal weights around the world*

| Country | Population[a] | Number of subjects | Daily wet fecal weight (grams) | | Source[b] |
|---|---|---|---|---|---|
| | | | Average | Range | |
| India | Nurses | 13 | 155 | ND | Burkitt, Walker and Painter (1972, 1974) |
| | Less than 15 years old in New Delhi | 36 | 374 | 50–1060 | Tandon and Tandon (1975) |
| | More than 15 years old in New Delhi | 514 | 311 | 19–1505 | Ibid. |
| Kenya | Hospital staff in rural area | 16 | 520 | 300–>500 | Cranston and Burkitt (1975) |
| Malaysia | Chinese | | | | |
| | Urban | 1 | 227 | 180–270 | Balasegaram and Burkitt (1976) |
| | Rural | 10 | 489 | 386–582 | Ibid. |
| | Malays | | | | |
| | Rural | 10 | 465 | 350–550 | Ibid. |
| | Indians | | | | |
| | Urban | 5 | 170 | 110–240 | Ibid. |
| | Rural | 8 | 385 | 255–520 | Ibid. |
| | Doctors | | | | |
| | Urban | 6 | 135 | 40–300 | Ibid. |
| Peru | Rural Indians | 20 | 325 | 60–650 | Crofts (1975) |
| South Africa | Rural | | | | |
| | Schoolchildren (black, age 9–12 years) | 32 | 16 (dry weight) | ND | Walker (1975) |
| | Schoolchildren (black) | 500 | 275 | 150–350 | Burkitt, Walker and Painter (1972, 1974) |
| | Urban | | | | |
| | Schoolchildren (black) | 500 | 165 | 120–260 | Ibid. |
| | Tertiary students (white) | 100 | 173 | 120–195 | Ibid. |
| Uganda | Teenage boarding school pupils | 27 | 185 | 48–348 | Burkitt, Walker and Painter (1972, 1974) |
| | Villagers | 15 | 470 | 178–980 | Ibid. |
| United Kingdom | Naval recruits and wives | 15 | 104 | 39–223 | Burkitt, Walker and Painter (1972, 1974) |
| | Teenage boarding school pupils | 9 | 110 | 71–142 | Ibid. |
| | Vegetarians | 24 | 225 | 71–488 | Ibid. |
| | Hospital patients (fiber added to diet) | 6 | 175 | 128–248 | Ibid. |
| | Laboratory staff | 4 | 162 | 123–224 | Greenberg (1976) |
| | Medical students | 33 | 132 | ND | Cummings (personal communication) |
| | Medical staff (age 22–36 years) | 11 | 107 | 60–182 | Goy and others (1976) |
| United States | Cincinnati, Ohio | 5 | 115 | 76–148 | Connell and Smith (1974) |
| | Philadelphia, Penn. | | | | |
| | Black students | 10 | 148 | ND | Goldsmith and Burkitt (1975) |
| | White students | 10 | 192 | ND | Ibid. |
| | San Francisco, Calif. | | | | |
| | Medical staff | 5 | 91 | ND | Gray and Tainter (1941) |
| | Norwalk, Conn. | | | | |
| | Volunteers (age 23–47 years) | 6 | 103 | 49–160 | Fuchs, Dorfman and Floch (1976) |

ND. No data.

*Source:* John Cummings (British Medical Research Council's Dunn Nutrition Unit, University of Cambridge) compiled the information contained in the table.

a. Subjects were on *ad lib.* diets except where indicated

b. Full citations of sources in this and subsequent tables appear in the reference lists.

other words, a solids content of 2.8 percent. If paper is used for anal cleansing, the solids content will increase to around 5 percent. The solids content of night soil is therefore similar to that of primary sewage works sludge. Data from Japan, the island of Taiwan, and Thailand indicate a solids content for night soil in the range 2.0–4.2 percent, with mean figures of 2.7–3.7 percent (Pescod 1971).

*Chemical composition*

Excreta, especially feces, are of complex and variable composition. Typical figures of some constituents are given in table 1-2. Of particular interest to the sanitary engineer are the data on carbon and nitrogen content indicating that the C:N ratio in feces is in the region of 8, whereas in urine it is under 1. These figures have considerable bearing on the design of composting systems in which the C:N ratio must be around 20–30 for the process to proceed efficiently (Gotaas 1956).

Of equal importance to the public health engineer is the concentration of organic material, measured by the biochemical oxygen demand (BOD) or other similar index (such as chemical oxygen demand, or total organic carbon).[1] In a night soil system, the per capita $BOD_5$ contribution is equal to the $BOD_5$ in excreta plus whatever $BOD_5$ contribution is made by the paper or other material used for anal cleansing. In the United States, Laak (1974) has found that urine contains 8.6 grams of $BOD_5$ per liter and that feces contain 9.6 grams of $BOD_5$ per 100 grams. As fecal weights increase and moisture content rises, the $BOD_5$ contribution per unit weight of wet feces clearly will fall. In addition, it is possible that higher fecal weights will be associated with a higher fiber content that may not be readily biodegradable, causing the higher fecal weights to be accompanied by lower $BOD_5$ contributions per unit weight of dry feces.

Table 1-2. *Composition of human feces and urine*

| | *Approximate composition (percent of dry weight)* | |
|---|---|---|
| *Constituent* | *Feces* | *Urine* |
| Calcium (CaO) | 4.5 | 4.5–6.0 |
| Carbon | 44–55 | 11–17 |
| Nitrogen | 5.0–7.0 | 15–19 |
| Organic matter | 88–97 | 65–85 |
| Phosphorus ($P_2O_5$) | 3.0–5.4 | 2.5–5.0 |
| Potassium ($K_2O$) | 1.0–2.5 | 3.0–4.5 |

*Source:* Adapted from Gotaas (1956).

Possible $BOD_5$ contributions at different fecal weights are given in table 1-3. These are speculative calculations and require confirmation by field testing. Laak (1974) has found that the daily $BOD_5$ contribution of toilet paper in the United States is 3.5 grams per capita, and this figure may be lower in some developing countries where water or non-biodegradable material is used. Where heavy paper (cement bags or newspaper), corncobs, or leaves are used, however, the contribution of anal cleansing material may be as in the United States. Figures have been added in table 1-3 to account for the contribution of anal cleansing material to the $BOD_5$ in night soil.

If a total daily volume of excreta and anal cleansing material of 1.5 liters per adult is assumed, it is possible to calculate the $BOD_5$ strength of adult night soil (table 1-3). Although the weights of $BOD_5$ for children will be lower, the volumes will also be lower, so that the concentration will be similar to that for adults, and the final night soil strength may be as calculated. Pradt (1971) found a night soil $BOD_5$ content of 10,000 milligrams per liter in Japan, and Hindhaugh (1973) found 46,000 milligrams per liter of $BOD_5$ in night soil in Lagos, Nigeria. This last figure is extremely high and may reflect the practice in Lagos of disposing of garbage in the night soil buckets.[2] However, the daily volume of night soil produced in Lagos is about 1.5 liters per capita, the figure assumed in table 1-3.

In a sewerage system the per capita $BOD_5$ contribution is augmented by sullage, which contains organic wastes and thus will also exert an oxygen demand. Typical figures for sewage that includes sullage are presented in table 1-4. Further data on the $BOD_5$ in sullage can be found in the section "Characteristics of Sullage" in this chapter.

1. The BOD is the mass of oxygen required by microorganisms to oxidize the organic content of the waste. It is an indirect measurement of the concentration of biodegradable material present. $BOD_5$ denotes the oxygen demand exerted during the standard test, which is conducted at 20°C over 5 days. The chemical oxygen demand is the mass of oxygen consumed when the organic matter present is oxidized by strong oxidizing agents in acid solution. It includes some substances (such as cellulose) that are not available to microorganisms but excludes some (such as acetic acid) that are.

2. Garbage may be placed in the night soil buckets because of the lack of an adequate refuse disposal system. Huponu-Wusu and Daniel (1977) found that only 39 percent of 1,099 randomly sampled households in metropolitan Lagos are reached by the refuse collection service of the city council.

Table 1-3. *Possible standard biochemical oxygen demand ($BOD_5$) content of excreta and night soil*

| *Population* | *Assumed adult fecal weight (grams daily)*[a] | *Assumed adult urine weight (kilograms daily)* | *Estimated water in feces (percent)* | *$BOD_5$ content* | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | *In wet feces (milligrams per gram)*[b] | *Per adult in feces (grams daily)* | *Per adult in urine (grams daily)* | *Total per adult in excreta (grams daily)* | *Per adult in anal-cleansing material (grams daily)* | *Strength of night soil (milligrams per liter)*[c] |
| Europe and North America | 150 | 1.2 | 75 | 96[d] | 14.4 | 10.3 | 24.7 | 3.5[d] | 18,800 |
| Developing country | | | | | | | | | |
| Urban | 250 | 1.2 | 80 | 77 | 19.3 | 10.3 | 29.6 | 3.0[e] | 21,700 |
| Rural | 350 | 1.2 | 85 | 58 | 20.3 | 10.3 | 30.6 | 2.0[e] | 21,700 |

*Notes:* This table is speculative and should not be used if actual data are available.

a. Fecal weights are taken from the ranges given in table 1-1.

b. Calculated by assuming that the $BOD_5$ contribution is constant per unit weight of dry feces. This assumption is unlikely to be accurate because the proportion of fiber will increase as fecal weight increases, and fiber is not readily biodegradable.

c. Assuming that 1.5 liters are produced by each adult daily.

d. From Laak (1974).

e. Where water is used for anal cleansing, this figure will be 0.

Table 1-4. *$BOD_5$ contributions* per capita *in urban sewage*

| *Country or region* | *$BOD_5$ per capita daily in sewage (grams)* |
|---|---|
| Brazil (São Paulo) | 50 |
| France (rural) | 24–34 |
| India | 30–55 |
| Kenya | 23–40 |
| Nigeria | 54 |
| Southeast Asia | 43 |
| United Kingdom | 50–59 |
| United States | 45–78 |
| Zambia | 36 |

*Note:* These figures were calculated by measuring the $BOD_5$ of raw sewage and multiplying it by the estimated daily water use per capita. This gives a most approximate result because urban sewage may contain a substantial proportion of commercial and industrial wastes. Domestic water use and $BOD_5$ contributions are not readily derived from data on total urban sewage, and these figures are not directly comparable with those in table 1-3.

*Pathogens in excreta*

Part Two of this work contains detailed information about the organisms causing human excreta-related diseases; however, a brief summation here of the major disease agents examined in Part Two may be of assistance. Four groups of pathogens—viruses, bacteria, protozoa, and worms—cause these diseases. In addition, excreta disposal may favor the breeding of insects, particularly mosquitoes, flies, and cockroaches, which will always have nuisance value and may act as vectors of human disease agents that may themselves not be found in feces or urine.

VIRUSES IN EXCRETA. Numerous viruses may infect the intestinal tract and be passed in the feces, whereupon they may infect new human hosts by ingestion or inhalation. One gram of human feces may contain $10^9$ infectious virus particles, regardless of whether the individual is experiencing any discernible illness. Although they cannot multiply outside a suitable host cell, the excreted viruses[3] may survive for many weeks in the environment, especially if temperatures are cool ($< 15°C$). Concentrations of $10^5$ infectious particles per liter of raw sewage have been reported, and excreted viruses can be readily isolated from soil and natural waters at sites which have been exposed to fecal discharges (World Health Organization 1979). Five groups of pathogenic excreted viruses (listed in table 1-5) are particularly important—adenoviruses, enteroviruses (including poliovirus), hepatitis A virus, reoviruses and diarrhea-causing viruses (especially rotavirus). Other virus groups are also found in feces. Infections with all of these, especially in children, are often subclinical.

As regards the enteroviruses, most poliovirus infections do not give rise to any clinical illness. Sometimes, however, infection can lead to mild influenza-like illness, to "virus meningitis," or to paralytic poliomyelitis, which may lead to permanent disability or death. It is estimated that paralytic poliomyelitis occurs worldwide in only about 1 out of every 1,000 poliovirus infections, but most children become infected in developing countries, and consequently the number of paralysis cases can be high. Echovirus and coxsackievirus infections can cause a wide range of diseases and symptoms including simple fever, meningitis, respiratory illness, paralysis, myocarditis, and other conditions (see chapter 9).

Rotaviruses, and other viruses, are found in the feces of a large number of young children suffering from diarrhea and are another important group of excreted viruses. Their precise causative role and epidemiology remain uncertain, but they are responsible for a substantial proportion of diarrhea episodes among young children in many countries (see chapter 11).

Hepatitis A virus is the causative agent of infectious hepatitis. Infection may lead to jaundice but, especially in young children, is often symptomless (see chapter 10).

BACTERIA IN EXCRETA. The feces of a healthy person contain large numbers of commensal bacteria of many

3. The term "excreted virus" is used here for comparability with "excreted bacterium," "excreted helminth," and so on. "Excreted virus" is synonymous with "enteric virus," which must be distinguished from the genus *Enterovirus*, which includes polio-, echo-, and coxsackieviruses.

Table 1-5. *Viral pathogens excreted in feces*

| *Virus* | *Disease* | *Can symptomless infections occur?* | *Reservoir* | *Chapter containing detailed information* |
|---|---|---|---|---|
| Adenoviruses | Numerous conditions | Yes | Man | 9 |
| Enteroviruses | | | | |
| Polioviruses | Poliomyelitis, paralysis and other conditions | Yes | Man | 9 |
| Echoviruses | Numerous conditions | Yes | Man | 9 |
| Coxsackie viruses | Numerous conditions | Yes | Man | 9 |
| Hepatitis A virus | Infectious hepatitis | Yes | Man | 10 |
| Reoviruses | Numerous conditions | Yes | Man and animals | 9 |
| Rotaviruses, Norwalk agent and other viruses | Diarrhea | Yes | Probably man | 11 |

*Note:* See table 9-1 for more information.

species. The species of bacteria found in the normal stool, and the relative numbers of different species, will vary among communities. The bacteria most commonly found and an indication of the variations in their concentrations in feces are given in table 1-6. Because these bacteria are ubiquitous and numerous in the feces of healthy people, they have been used as indicators of fecal pollution.[4] The most widely used indicator has been the fecal coliform *Escherichia coli*, the main constituent of the "enterobacteria" group in table 1-6, but enterococci (or, more generally, fecal streptococci), another widespread commensal group, are also used as indicators. Anaerobic bacteria also, such as *Clostridium*, *Bacteroides*, and *Bifidobacterium*, have served as indicators, and their potential value as indicators is currently attracting increased attention (Evison and James 1977).

On occasion, some bacteria listed in table 1-6, or their particular strains, may give rise to disease, as may other groups of bacteria normally absent from the healthy intestine. These pathogenic, or potentially pathogenic, bacteria are listed in table 1-7. They most commonly enter a new host by ingestion (in water, on food, on fingers, in dirt), but some may also enter through the lungs (after inhalation of aerosol particles) or through the eye (after rubbing the eye with fecally contaminated fingers). At some time during the course of an infection, large numbers of the bacteria will be passed in the feces, thus allowing the spread of infection to new hosts.

Diarrhea is a major symptom of many bacterial intestinal infections. The bacteria may also invade the body from the gut and cause either generalized or localized infections. This invasion is characteristic of typhoid infections and other enteric fevers caused by salmonellae. During infections restricted to the gut, bacteria will be passed only in the feces. When invasion has occurred, bacteria may be passed in the urine as well and will also be found in the bloodstream at some stage.

A carrier state exists in all the infections listed in table 1-7. Thus, in communities where these infections are endemic, a proportion of perfectly healthy individuals will be excreting pathogenic bacteria. These carriers play a prominent role in transmitting the infection they carry because they are mobile, dispersing their feces widely. Cholera provides an example of the problem. A patient with severe cholera will be in bed for most of the time he or she is excreting *Vibrio cholerae*. Those who nurse the patient clearly are at risk, but the patient is not disseminating bacteria in the community. A patient with a mild case, or a carrier, by contrast may look relatively healthy and be mobile while excreting up to $10^6$ chloera vibrios per gram of feces. In some infections the carrier state may last for a duration similar to the illness itself, but in others it may persist for months or even a lifetime. Some carriers may show symptoms of illness and continue to excrete the bacteria, whereas others may be healthy throughout infection. A carrier becomes especially dangerous when engaged in food preparation or handling or in water supply.

Some of the pathogens listed in table 1-7 are excreted entirely (or almost entirely) by man, but others are excreted by a wide range of animals. This fact limits disease control through improvements in human excreta disposal alone, because any changes made will likely not affect transmission of pathogens from animal feces to humans. Three of the major infections listed in table 1-7 (typhoid, shigellosis, and cholera), however, are assumed to be exclusively human infections, whose spread is from one person to another.

In summary, all the viral and bacterial pathogens listed, respectively, in tables 1-5 and 1-7 are passed in the feces of man or animals; they are not free living.[5] Infection of a new host normally follows ingestion of the pathogens and because transmission is primarily through the swallowing of minute quantities of infected feces, the sanitary disposal of all feces (both human and animal) and perfect personal hygiene would largely eliminate these diseases. For many infections, this has unfortunately proved an unattainable goal in even the most affluent societies, and so a more modest target than eradication must be set: the reduction of transmission to a manageable level.

Bacteria of the genus *Leptospira* have been excluded from the discussion above because they cannot be included in the generalizations made. Although leptospirosis in the majority of human cases gives rise to a benign, self-limiting, febrile illness, it occasionally leads to severe, even fatal disease characterized by jaundice and hemorrhage (Weil's syndrome) whereupon death may result from kidney failure. *Leptospira* are excreted in the urine of animal carriers, and usually reach new animal and human hosts through skin abrasions or mucous membranes contaminated by infected urine. Man may be an intermittent carrier for a few weeks (rarely months) after an acute infection. Leptospirosis is considered here because of the risk to workers who handle excreta, which may contain leptospires either from animal carriers (for example,

4. The use of indicator organisms is discussed in more detail in chapters 4 and 13.

5. This may not be true for *Vibrio cholerae*; see chapter 17.

Table 1-6. *Bacterial microflora of human feces by national diet*

| *National diet* | *Country* | *Number of bacteria in feces (mean $\log_{10}$ per gram)* | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | *Entero-bacteria*[a,b] | *Enterococci*[b] | *Lactobacilli* | *Clostridia* | *Bacteroides* | *Bifido-bacteria* | *Eubacteria* |
| Largely carbo-hydrate | Guatemala | 8.7 | 7.9 | 9.0 | 9.3 | 10.3 | 9.4 | ND |
| | Hong Kong | 7.0 | 5.8 | 6.1 | 4.7 | 9.8 | 9.1 | 8.5 |
| | India | 7.9 | 7.3 | 7.6 | 5.7 | 9.2 | 9.6 | 9.5 |
| | Japan | 9.4 | 8.1 | 7.4 | 5.6 | 9.4 | 9.7 | 9.6 |
| | Nigeria | 8.3 | 8.0 | ND | 5.9 | 7.3 | 10.0 | ND |
| | Sudan | 6.7 | 7.7 | 6.4 | 4.9 | 7.8 | 8.5 | ND |
| | Uganda | 8.0 | 7.0 | 7.2 | 5.1 | 8.2 | 9.4 | 9.3 |
| Mixed Western | Denmark | 7.0 | 6.8 | 6.4 | 6.3 | 9.8 | 9.9 | 9.3 |
| | England | 7.9 | 5.8 | 6.5 | 5.7 | 9.8 | 9.9 | 9.3 |
| | Finland | 7.0 | 7.8 | 8.0 | 6.2 | 9.7 | 9.7 | 9.5 |
| | Scotland | 7.6 | 5.3 | 7.7 | 5.6 | 9.8 | 9.9 | 9.3 |
| | United States | 7.4 | 5.9 | 6.5 | 5.4 | 9.7 | 9.9 | 9.3 |

ND. No data.

*Sources:* England, India, Japan, Scotland, United States, Uganda (Drasar 1974); Denmark, Finland (International Agency for Research on Cancer 1977); Hong Kong (Crowther and others 1976); Nigeria, Sudan (Draser, personal communication); Guatemala (Mata, Carrillo and Villatoro 1969).

a. This group mainly contains *Escherichia coli.*

b. These two groups are the most commonly used fecal indicator bacteria.

Table 1-7. *Bacterial pathogens excreted in feces*

| *Bacterium* | *Disease* | *Can symptomless infection occur?* | *Reservoir* | *Chapter containing detailed information* |
|---|---|---|---|---|
| *Campylobacter fetus* ssp. *jejuni* | Diarrhea | Yes | Animals and man | 12 |
| Pathogenic *Escherichia coli*[a] | Diarrhea | Yes | Man[b] | 13 |
| *Salmonella* | | | | |
| *S. typhi* | Typhoid fever | Yes | Man | 15 |
| *S. paratyphi* | Paratyphoid fever | Yes | Man | 15 |
| Other salmonellae | Food poisoning and other salmonelloses | Yes | Animals and man | 15 |
| *Shigella* spp. | Bacillary dysentery | Yes | Man | 16 |
| *Vibrio* | | | | |
| *V. cholerae* | Cholera | Yes | Man | 17 |
| Other vibrios | Diarrhea | Yes | Man | 17 |
| *Yersinia enterocolitica* | Diarrhea and septicemia | Yes | Animals and man[c] | 18 |

a. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli.*

b. Although many animals are infected by pathogenic *E. coli*, each serotype is more or less specific to a particular animal host.

c. Of the 30 or more serotypes identified so far, a number seem to be associated with particular animal species. There is at present insufficient epidemiological and serological evidence to say whether distinct serotypes are specific to primates.

the sewer rat, *Rattus norvegicus*) attracted to such environments or, occasionally, from infected people.

PROTOZOA IN EXCRETA. Many species of protozoa can infect man and cause disease. Among them are several species that are harbored in the intestinal tract of man and other animals, where they may cause diarrhea or dysentery. Infective forms of these protozoa are often passed as cysts in the feces, and man is infected when he ingests them. Only three species of human intestinal protozoa are considered to be frequently pathogenic: *Giardia lamblia*, *Balantidum coli*, and *Entamoeba histolytica* (see table 1-8). An asymptomatic carrier state is common in all three and, in the case of *Entamoeba histolytica*, it is carriers who are primarily responsible for continued transmission.

HELMINTHS IN EXCRETA. Many species of parasitic worms, or helminths, have human hosts. Some can cause serious illnesses, but a number generate few symptoms. Only those helminths whose eggs or larval forms are passed in the excreta are of concern to this study. Only *Schistosoma haematobium* (the agent of urinary schistosomiasis) is voided in the urine; the others examined are all excreted in the feces. The helminths that begin a new cycle of transmission by escaping from a blister on the carrier's skin (guinea worm, *Dracunculus medinensis*), or by entering the body of a blood-feeding insect to be transmitted through its bite to a new host (*Onchocerca volvulus*, the agent of human onchocerciasis, or river blindness), are not considered.[6]

Helminths (except for *Strongyloides*) do not multiply within the human host, and this is of great importance in understanding their transmission, the ways they cause disease, and the effects of environmental changes on their control. Helminthic disease is not an all-or-nothing phenomenon. In infections due to viruses, bacteria and protozoa, where massive asexual reproduction occurs within the host, once infection occurs its severity cannot be related easily to the infecting dose of organisms. One either has measles, or a common cold, or not and it is not meaningful to say that someone has "a lot of measles". By contrast, with helminthic infections it is essential to think quantitatively. The

6. An exception, discussed in detail in chapter 36, is the bloodborne larva of the filarial worm causing elephantiasis, which may be transmitted by *Culex pipiens* mosquitoes, which breed in sewage, sullage, and other polluted waters. *Culex pipiens* is a complex of mosquito species and subspecies. The main tropical species, and the major vector of filariasis in those tropical areas where the infection is *Culex*-transmitted, is *Culex quinquefasciatus* (previously also known as *Culex pipiens fatigans*, *C.p. quinquefasciatus*, or *C. fatigans*). Other important species are *C.p. pipiens*, *C.p. molestus* (the vector of filariasis in Egypt), and *C.p. pallens*. More details on the complex are provided in chapter 36. "*Culex pipiens*" will be used throughout the text unless a particular member of the complex is being referred to. Because they are not specifically associated with excreta, other insectborne pathogens (such as trypanosomes and [illegible] their vectors, and the diseases they cause are excluded from the purview of this study.

Table 1-8. *Protozoal pathogens excreted in feces*

| *Protozoon* | *Disease* | *Can symptomless infections occur?* | *Reservoir* | *Chapter containing detailed information* |
|---|---|---|---|---|
| *Balantidium coli* | Diarrhea, dysentery and colonic ulceration | Yes | Man and animals (especially pigs and rats) | 19 |
| *Entamoeba histolytica* | Colonic ulceration, amebic dysentery, and liver abscess | Yes | Man | 20 |
| *Giardia lamblia* | Diarrhea and malabsorption | Yes | Man and animals | 21 |

question is not just whether or not someone has a hookworm infection but how many worms has he (in other words, how "heavy" or "intense" is the infection). Sometimes worm burdens can be determined by purging the patient immediately after an anthelminthic, but more usually the output of eggs in the excreta is determined and used as an index of the intensity of infection. Even though there is a good deal of variation from day to day, the relation is valid at community level and in any case the egg output is always a better measure of transmission and sometimes a better guide to pathology than the burden of adult worms.

Worm burdens and levels of egg output are not evenly or randomly distributed among their human hosts, and within any sex and age group of an infected community there will be a few people who are carrying a heavy worm burden and a much larger number with light intensities of infection. In general, the risk of illness and its severity increases with the worm burden. It is therefore common in helminth infections to find many of the community infected, occasional people (often with heavy infections) ill, and a few dying. It is relatively easy to see the public health importance of the heavy infections but far harder to assess disability in the lightly infected majority where consequences are likely to be nonspecific and effects cumulative with those from other infections.

The number of heavy infections is not simply proportional to the prevalence of infection. At high prevalences, increased transmission will tend mainly to push up the proportion of heavy infections while at low prevalences there may be few people heavily infected and the number may change little with transmission. Where immunity acquired by the host is unimportant, a reduction in transmission due to control of excreta may reduce the number of heavy infections and so reduce the burden of disease even if it affects the prevalence of infection rather little.

Because of this quantitative characteristic, the development of pathology in helminthic infections is usually the result of cumulative worm burdens, often carried over many years as a product of regular and repeated reinfection. This further contrasts with the asexually replicating organisms, which may cause an overwhelmingly heavy infection and a state of gross disease within a few days or weeks after a single infective dose enters the body.

The excreted helminths are listed in table 1-9. Often the developmental stages through which they pass before reinfecting man, their life cycles, are very complex (as is shown in the table). The helminths are classified in two main groups: the roundworms (nematodes) and those worms that are flat in cross-section. The flatworms again form two groups: the tapeworms (cestodes), which form chains of helminth "segments," and the flukes (trematodes), which have a single flat, unsegmented body. The roundworms may cause mechanical obstruction (*Ascaris*), rectal prolapse (*Trichuris*), itching around the anus (*Enterobius*), or anemia (hook-worms). They also divert food to themselves and produce abdominal pain in some cases (many cases, however, are symptomless). Adult tapeworms create health problems mainly by depriving their host of nutrients. Of the trematodes, some inhabit and damage the liver (*Clonorchis*) or lungs (*Paragonimus*). The schistosomes live outside the intestine in small blood vessels; their eggs that fail to escape from the host may damage several organs. The intestinal flukes may occur in large numbers, are mostly transmitted through food, and cause relatively mild symptoms.

Most of the roundworms infecting man, and also the schistosome flukes, have separate sexes, so that transmission depends upon infection with both male and female worms and upon the meeting, mating, and egg production of these worms within the human body. A number of individuals may be infected with a single

Table 1-9. *Helminthic pathogens excreted in feces*

| *Helminth* | *Common name* | *Disease* | *Transmission* | *Distribution* | *Chapter containing detailed information* |
|---|---|---|---|---|---|
| *Ancylostoma duodenale* | Hookworm | Hookworm | Man → soil → man | Mainly in warm wet climates | 22 |
| *Ascaris lumbricoides* | Round worm | Ascariasis | Man → soil → man | Worldwide | 23 |
| *Clonorchis sinensis* | Chinese liver fluke | Clonorchiasis | Man or animal → aquatic snail → fish → man | Southeast Asia | 24 |
| *Diphyllobothrium latum* | Fish tapeworm | Diphyllobothriasis | Man or animal → copepod → fish → man | Widely distributed foci, mainly temperate regions | 25 |
| *Enterobius vermicularis* | Pinworm | Enterobiasis | Man → man | Worldwide | 26 |
| *Fasciola hepatica* | Sheep liver fluke | Fascioliasis | Sheep → aquatic snail → aquatic vegetation → man | Worldwide in sheep- and cattle-raising areas | 27 |
| *Fasciolopsis buski* | Giant intestinal fluke | Fasciolopsiasis | Man or pig → aquatic snail → aquatic vegetation → man | Southeast Asia, mainly China | 28 |
| *Gastrodiscoides hominis* | n.a. | Gastrodiscoidiasis | Pig → aquatic snail → aquatic vegetation → man | India, Bangladesh, Vietnam, Philippines | 30 |
| *Heterophyes heterophyes* | n.a. | Heterophyiasis | Dog or cat → brackish-water snail → brackish-water fish → man | Middle East, southern Europe, Asia | 30 |

| | | | | | |
|---|---|---|---|---|---|
| *Hymenolepis nana* | Dwarf tapeworm | Hymenolepiasis | Man or rodent → man | Worldwide | 29 |
| *Metagonimus yokogawai* | n.a. | Metagonimiasis | Dog or cat → aquatic snail → freshwater fish → man | East Asia, Siberia (USSR) | 30 |
| *Necator americanus* | Hookworm | Hookworm | Man → soil → man | Mainly in warm wet climates | 22 |
| *Opisthorchis felineus* | Cat liver fluke | Opisthorchiasis | Cat or man → aquatic snail → fish → man | USSR, Thailand | 24 |
| *O. viverrini* | n.a. | | | | |
| *Paragonimus westermani* | Lung fluke | Paragonimiasis | Pig, man, dog, cat, or other animal → aquatic snail → crab or cray-fish → man | Southeast Asia, scattered foci in Africa and South America | 31 |
| *Schistosoma haematobium* | Schistosome | Schistosomiasis; bilharziasis | Man → aquatic snail → man | Africa, Middle East, India | 32 |
| *S. japonicum* | | | Animals and man → snail → man | Southeast Asia | 32 |
| *S. mansoni* | | | Man → aquatic snail → man | Africa, Middle East, Central and South America | 32 |
| *Strongyloides stercoralis* | Threadworm | Strongyloidiasis | Man → man | Mainly in warm wet climates | 33 |
| *Taenia saginata* | Beef tapeworm | Taeniasis | Man → cow → man | Worldwide | 34 |
| *T. solium* | Pork tapeworm | Taeniasis | Man → pig (or man) → man | Worldwide | 34 |
| *Trichuris trichiura* | Whipworm | Trichuriasis | Man → soil → man | Worldwide | 35 |

n.a. Not applicable.

sex or with unmated worms. These cases are of no epidemiological significance because they do not transmit infection.

*Magnitude of pathogen excretion*

We can dramatize the magnitude of the potential health hazard from excreta by considering a typical load of pathogens excreted by a poor tropical community in a single day. Estimated data on the more prominent diseases threatening public health and the large fecal volume, often containing significant concentrations of pathogenic organisms, produced in a hypothetical community are given in table 1-10. Excreta-related diseases account for some 10–25 percent of illnesses that reach the health care services, and cause a vast amount of misery that goes unreported. Given the dangers of poor sanitation, it is crucial that the engineering profession and the appropriate governmental agencies of the world take seriously the responsibility to collect, transport, treat, and reuse human waste substances in ways that do not endanger the public.

*A note on urinary pathogens*

In general, urine is a sterile and harmless substance. There are, however, occasions when host infections cause passage of pathogens in the urine. The three principal infections leading to the significant appearance of pathogens in the urine are urinary schistosomiasis, typhoid, and leptospirosis. Coliform and other bacteria may be numerous in the urine during cystitis and other urinary infections, but they constitute no public risk. In venereal infections the microbial agents will also reach the urine, but they are so vulnerable to conditions outside the body that excreta are unimportant vehicle of transmission.

People infected with urinary schistosomiasis (caused by *Schistosoma haematobium*) will pass eggs chiefly in their urine. The worms live for years (occasionally decades) and superinfection occurs, so that those affected may pass eggs—sometimes accompanied by blood—for much of their lifetimes. In heavy infections, 10 millilitres of urine may contain over a thousand eggs if the urine is collected near to midday, when eggs are most numerous. During the phase of typhoid and paratyphoid fevers when bacteria are disseminated in the blood, the organisms will usually be shed in the urine. In cases where *S. haematobium* is also present, however, prolonged urinary carriage of typhoid may occur over many years. An individual with leptospirosis will pass *Leptospira* intermittently in the urine for a period of about 4–6 weeks; chronic human carrier states are rare.

## Characteristics of Sullage

Sullage, also known as graywater, is domestic wastewater not containing excreta—the water discarded from baths, sinks, basins and the like that may be expected to contain considerably fewer pathogenic microorganisms than sewage. Interest and research in the handling of sullage has increased in recent years, both in developing and affluent countries. In affluent countries there is growing interest in the use of sewerless chemical toilets and separate sullage disposal as a way of overcoming environmental problems associated with the disposal of large volumes of heavily contaminated sewage from urban areas. There is also interest in chemical toilets and on-site sullage disposal for use in nature parks, where environmental considerations are paramount (Winneberger 1974).

There is also a growing realization in developing countries of the financial and other difficulties associated with providing waterborne sewerage systems, and consequent increased interest in dry or on-site techniques such as improved pit latrines, composting toilets or cartage systems (Kalbermatten and others 1982). Some of these sewerless technologies require the separate disposal of sullage when the volumes of domestic wastewater become too great simply to drain away in the yard. Furthermore, a worldwide awareness is dawning that it is extravagant to use up to half of a household's high quality drinking water just to flush excreta along sewers. The need to design a sullage disposal system accompanies the development of any toilet not flushed by water.

*Quantities*

Sullage volumes depend upon domestic water use. Where people use public taps, daily domestic water use may be as low as 10 liters per capita (White 1977). In affluent households with full plumbing, daily water use may be 200 or more liters per capita, and all water not used for flushing toilets may be classed as sullage. Bennett, Linstedt and Felton (1974), studying homes in the United States, found that the toilet was used 3.6 times daily per capita, that the average flush used 15 liters, and that toilet flushing accounted for 33 percent of domestic water use. Witt, Siegrist and Boyle (1974), also studying homes in the United States, found corresponding figures of 2.3 times daily per capita, 15 liters for flush, and 22 percent of water use allocated to

Table 1-10. *Possible output of selected pathogens in the feces and sewage of a tropical community of 50,000 in a developing country*

| *Pathogen* | *Prevalence of infection in country (percent)*[a] | *Average number of organisms per gram of feces*[b] | *Total excreted daily per infected person*[c] | *Total excreted daily by town* | *Concentration per liter in town sewage*[b] |
|---|---|---|---|---|---|
| Viruses | | | | | |
| *Enteroviruses*[d] | 5 | $10^6$ | $10^8$ | $2.5 \times 10^{11}$ | 5,000 |
| Bacteria | | | | | |
| Pathogenic *E. coli*[e] | ? | $10^8$ | $10^{10}$ | ? | ? |
| *Salmonella* spp. | 7 | $10^6$ | $10^8$ | $3.5 \times 10^{11}$ | 7,000 |
| *Shigella* spp. | 7 | $10^6$ | $10^8$ | $3.5 \times 10^{11}$ | 7,000 |
| *Vibrio cholerae* | 1 | $10^6$ | $10^8$ | $5 \times 10^{10}$ | 1,000 |
| Protozoa | | | | | |
| *Entamoeba histolytica* | 30 | $15 \times 10^4$ | $15 \times 10^6$ | $2.25 \times 10^{11}$ | 4,500 |
| Helminths | | | | | |
| *Ascaris lumbricoides* | 60 | $10^4$[f] | $10^6$ | $3 \times 10^{10}$ | 600 |
| Hookworms[g] | 40 | 800[f] | $8 \times 10^4$ | $1.6 \times 10^9$ | 32 |
| *Schistosoma mansoni* | 25 | 40[f] | $4 \times 10^3$ | $5 \times 10^7$ | 1 |
| *Taenia saginata* | 1 | $10^4$ | $10^6$ | $5 \times 10^8$ | 10 |
| *Trichuris trichiura* | 60 | $2 \times 10^3$[f] | $2 \times 10^5$ | $6 \times 10^9$ | 120 |

? Uncertain.

*Note:* This table is hypothetical, and the data are not taken from any actual, single town. For each pathogen, however, the figures are reasonable and congruous with those found in the literature. The concentrations derived for each pathogen in sewage are in line with higher figures in the literature, but it is unlikely that all these infections at such relatively high prevalences would occur in any one community.

a. The prevalences given in this column refer to infection and *not* to morbidity.

b. It must be recognized that the pathogens listed have different abilities to survive outside the host and that the concentrations of some of them will rapidly decline after the feces have been passed. The concentrations of pathogens per liter in the sewage of the town were calculated by assuming that 100 liters of sewage are produced daily per capita and that 90 percent of the pathogens do not enter the sewers or are inactivated in the first few minutes after the excretion.

c. To calculate this figure it is necessary to estimate a mean fecal weight for those people infected. This must necessarily be the roughest of estimates because of the age-specific fecal weights and the age distribution of infected people in the community. It was assumed that people over 15 years old excrete 150 grams daily and that people under 15 excrete, on average, 75 grams daily. It was also assumed that two-thirds of all infected people are under 15. This gives a mean fecal weight for infected individuals of 100 grams.

d. Includes polio-, echo-, and coxsackieviruses.

e. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli*.

f. The distribution of egg output from people infected by these helminths is extremely skewed; a few people excrete very high egg concentrations.

g. *Ancylostoma duodenale* and *Necator americanus*.

flushing. Reviewing data from several studies, Witt and colleagues found that water from toilet flushing was between 22 and 45 percent of the total domestic water usage. Laak (1974) reviewed data from Canada, Sweden, and the United States that show the following percentage allocations of water use in houses with full plumbing:

| | *Mean* | *Range* |
|---|---|---|
| Bathroom | 26 | 12–40 |
| Kitchen | 9 | 5–16 |
| Laundry | 18 | 4–22 |
| Toilet flushing | 47 | 41–65 |

We have been unable to obtain comparable figures from urban households, either with or without sewer connections, in developing countries. Data for rural households without sewers, however, are available, and examples of water use allocations in Lesotho, Papua New Guinea, and Uganda are given in table 1-11. These figures highlight the immense differences in water use practice, and thus in the kind of sullage produced, in areas varying in culture, environment, wealth, and other factors. The health implications of sullage disposal will depend on the technologies used, which in turn must consider such variables as the household volume of sullage, density of housing, local climate, soil type, and groundwater conditions.

### *Composition*

The results of surveys of five households in the United States are shown in table 1-12 (from Laak 1974). The sullage contributed 53 percent of the sewage flow, 52 per cent of the $BOD_5$, 43 percent of the chemical oxygen demand, about 15 percent of the nitrogen, and 45 percent of the phosphates. The data in table 1-12 further indicate that, if the ratio of chemical oxygen demand to $BOD_5$ is used as the criterion, toilet wastes are more resistant to biodegradation than sullage. Hypes (1974) points out the effect of sink-installed garbage disposal units on the quality of sullage. In his test, sullage had a $BOD_5$ of 328 milligrams per liter when without garbage solids and 480 milligrams per liter when with garbage. Another report found that in Taipei, sullage contributed 40 percent of $BOD_5$ in sewage (but in Taipei, scraps were fed to pigs rather than washed down the sewers; World Health Organization 1970).

Witt, Siegrist and Boyle (1974) examined the bacterial content of sullage in the United States. Their results, summarized in table 1-13, show that water used for bathing and showering became less contaminated with fecal bacteria than water used in washing clothes. Furthermore, 38 percent of the total fecal streptococcal isolates were enterococci (*Streptococcus faecalis*, *S. faecium*, and *S. durans*); the majority of the bath water enterococci were *S. faecalis* var. *liquefaciens* (in contrast, only a few enterococci isolated from the clothing waters were of this species, now widely regarded as being nonfecal in origin). *S. bovis*, a primarily nonhuman species, accounted for 22 percent of all streptococcal isolates. These findings suggest that under half of the streptococci isolated were from human feces, and that the bath water was even less

Table 1-11. *Allocation of water use in sewerless rural households in developing countries*

| | *Country* | | | |
|---|---|---|---|---|
| | | | *Uganda* | |
| *Water use* | *Lesotho* | *Papua New Guinea (Enga Province)* | *Lango* | *Kigezi* |
| Average total daily use per capita (liters) | 18 | 0.68 | 18 | 8 |
| Bathroom (personal hygiene) (percent) | 15 | 0 | 66 | 20 |
| Laundry (percent) | 22 | 0 | | |
| Drinking | | | | |
| Animals (percent) | 2 | 8 | 0 | 0 |
| Humans (percent) | 45 | 79[a] | 19 | 6[a] |
| Kitchen (cooking and utensil hygiene) (percent) | | 11 | 13 | 74 |
| Vegetable gardens (percent) | 6 | 0 | 0 | 0 |
| Other (percent) | 10 | 2 | 2 | 0 |

*Sources:* Lesotho (Feachem and others 1978); Papua New Guinea (Feachem 1977); Uganda (White, Bradley and White 1972).

a. These are very small volumes of drinking water. In Papua New Guinea they may be due to low salt intake and consequent low fluid demand and to water intake from food, especially sugar cane. In Kigezi, Uganda, the practice of eating gruels and other high liquid foods may account for the low drinking water consumption.

Table 1-12. *Pollution loads of wastewater sampled from various plumbing fixtures in the USA (milligrams per capita daily)*

| Wastewater source | Biochemical oxygen demand (BOD) | | Chemical oxygen demand | | $NO_3$–N | | $NH_3$–N | | $PO_4$ | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Mean | Percent | Mean | Percent | Mean | Percent | Mean | Percent | Mean | Percent |
| Bathroom sink | 1,860 | 4 | 3,250 | 2 | 2 | 3 | 9 | 0.3 | 386 | 3 |
| Bathtub | 6,180 | 13 | 9,080 | 8 | 12 | 16 | 43 | 1 | 30 | 0.3 |
| Kitchen sink | 9,200 | 19 | 18,800 | 16 | 8 | 10 | 74 | 2 | 173 | 2 |
| Laundry machine | 7,900 | 16 | 20,300 | 17 | 35 | 49 | 316 | 10 | 4,790 | 40 |
| Toilet | 23,540 | 48 | 67,780 | 57 | 16 | 22 | 2,782 | 87 | 6,473 | 55 |
| Total | 48,690 | 100 | 119,410 | 100 | 73 | 100 | 3,224 | 100[a] | 11,862 | 100[a] |

*Source:* Adapted from Laak (1974).
a. Total percentage rounded to 100.

contaminated relative to the clothing water than the total counts suggested. Hypes (1974) found that coliform counts in sullage were about $1.9 \times 10^7$ per 100 milliliters irrespective of garbage content. After 24 hours of storage, this count had increased to $5.4 \times 10^8$, indicating that sullage is a favorable medium for coliform growth.

Available information on the microbiological quality of sullage is very limited and neither of these two data sets (Hypes 1974 and Witt, Siegrist and Boyle 1974) may be representative. A more recent study in the USA reports lower bacterial counts in clothing wash water (215 total coliforms, 107 fecal coliforms and 77 fecal streptococci per 100 milliliters), and higher counts in bath water (1,810 total coliforms, 1,210 fecal coliforms and 326 fecal streptococci per 100 milliliters), than those given in table 1-13 (Small Scale Waste Management Project 1978).

Although data are lacking, it may be assumed that sullage from bathrooms and laundries will contain small numbers of any pathogenic viruses, bacteria, protozoa, or helminth eggs being excreted by the people who use them. The washing of babies and their soiled clothing may substantially raise the pathogen content of sullage. It is also possible that some bacteria find warm sullage a suitable medium for multiplication. Data on the microbiological quality of sullage from the tropics might verify this possibility, and its collection should be a priority of sanitation research.

Table 1-13. *Bacterial content of sullage in the USA (per 100 milliliters)*

| Sullage source | Total coliforms | | Fecal coliforms | | Fecal streptococci | |
|---|---|---|---|---|---|---|
| | Geometric mean | Range | Geometric mean | Range | Geometric mean | Range |
| Bath and shower water | 1,100 | 70–($8.2 \times 10^3$) | 220 | 1–($2.5 \times 10^3$) | 44 | 1–($7 \times 10^4$) |
| Clothing washwater | 18,000 | 85–($8.9 \times 10^5$) | 1,400 | 9–($1.6 \times 10^4$) | 210 | 1–($1.3 \times 10^6$) |
| Clothing rinsewater | 5,300 | 190–($1.5 \times 10^5$) | 320 | 35–($7.1 \times 10^3$) | 75 | 1–($2.3 \times 10^5$) |

*Source:* Adapted from Witt, Siegrist and Boyle (1974).

*Sullage disposal and health*

There are five kinds of sullage disposal: casual disposal by tipping wastewater receptacles in the yard; garden watering; on-site disposal by soakaway; drainage into open drains; and drainage into covered drains or sewers. Each of these has different health implications.

Tipping in the yard may create breeding sites for insects such as *Culex pipiens* as well as muddy and unsanitary conditions close to the dwellings. Because it does not offer concealment, a clean, dry yard is less likely to be used by children for defecation, and any worm eggs their feces might contain will be less likely to mature (nematode eggs require a moist environment to develop).[7] Sullage containing pathogens from babies' bath water or adults' ablution water may also infect children playing in the yard. In well-draining soils, where sullage production or housing density is low, tipping of sullage outside the home is unlikely to be a major health hazard. Where soils are less permeable and where water use or housing density is high, however, an adequate method of sullage disposal is essential. (It should be noted that high housing densities are generally associated with poverty and thus with low water use and sullage production.)

Sullage disposal by watering vegetable gardens near the house is likely to create few if any health hazards, provided that prolonged ponding of wastewater is prevented (to discourage mosquito breeding) and that children are discouraged from defecating in or near the gardens. Sullage disposal by soakaway provides a low risk of groundwater contamination; the risk of microbiological groundwater pollution is much lower with sullage than it is with sewage.[8] The same is true of high nitrate pollution (as indicated in table 1-12, sullage contains little nitrogen compared with sewage).

Drainage of wastewater into open drains, perhaps into storm drains, provides the most readily identifiable health risk, namely that of promoting the breeding of *C. pipiens* and other mosquitoes. In areas of year-round rainfall, storm drains will contain water continuously. If they are kept free of garbage and are well designed, the drains will flow freely and provide few sites for mosquito breeding, and the presence or absence of sullage will not affect community health. But in areas of seasonal rainfall, and where the drains are liable to blockage and ponding, the addition of sullage will create year-round standing water and thus year-round *Culex* breeding where only seasonal breeding may previously have occurred. It is not, therefore, the quality of the sullage that poses a health risk, since ponded stormwater will also be sufficiently polluted to allow *Culex* breeding, but the continuous addition of sullage to storm drains subject to ponding that converts wet season breeding into year-round breeding. In this case the rise in *Culex* populations may lead to increased filariasis transmission and thus to more and heavier infections and so more disease.

An example of this effect can be found in the recent resurgence of Bancroftian filariasis as a major public health problem in Egypt (Southgate 1979). Since approximately 1965 a complex of factors—including major changes in irrigation practice, a proliferation of poorly maintained water supplies, and inadequate excreta-disposal facilities contaminating surface water—has increased *C. pipiens* breeding in parts of the Nile Delta. Consequently, the prevalence, intensity, and geographic spread of Bancroftain filariasis have increased. It has also contributed to explosive epidemics of Rift Valley fever in Egypt during 1977 and 1978 (Hoogstraal, Meegan and Khalil 1979).

Urban areas can suffer similar health risks when large-scale sullage disposal is into open drains with a tendency to blockage. Too often sullage makes its way to streams by natural gullies, and no formally defined drainage system exists. The solution to these problems is either to use an alternative method of sullage disposal or to prevent drains from blocking by covering them or by vigorous efforts to keep them clear. The latter approach is the more realistic and labor intensive and can be implemented by the employment of municipal workers, by subcontracting the job to the private sector, or by organizing and motivating community effort on a neighborhood basis.

Finally, sullage may be disposed of into a sewerage system, as is sewage, except that smaller-bore pipes are used. This means of disposal raises no special health problems, and conventional treatment before discharge or reuse should be highly effective. The load of pathogenic microorganisms in sullage will be small, so that discharge or reuse can take place without tertiary treatment.

7. Some of the classic studies on nematode infections (for instance, Cort, Otto and Spindler 1930; Otto, Cort and Keller 1931; Otto and Spindler 1930; and Winfield 1937) suggest that, among households of similar socioeconomic status, the contamination of the yard by the feces of young children is associated with increased *Ascaris* prevalence and intensity in the family (see chapter 23).

8. See chapter 7.

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# 2

# Environmental Classification of Excreta-related Infections

VARIOUS DISEASES are related to excreta, and the engineer, administrator, and community development worker cannot consider each disease separately in selecting improved excreta disposal technologies. Rather, a conceptual framework that links various kinds of excreta-related infections to the design and implementation of particular disposal or reuse technologies is required. Yet a biological classification grouping the excreted viruses, bacteria, protozoa, and helminths may be less helpful to understanding the health aspects of alternative approaches to excreta disposal than a classification of infections based upon transmission routes and life cycles. Such a classification would be an "environmental" one. In fact, the resemblance between a biological and an environmental classification is much closer in the case of the excreta-related infections than in the case of the water-related infections (see Bradley 1977).

The purpose of an environmental classification is to group infections in such a way that the efficacy of different preventive measures is made clear. An environmental classification for the water-related infections has already been proposed (Bradley 1977; Feachem, McGarry and Mara 1977); the object here is to propose an environmental classification for the infections associated with excreta. The devising of such a classification encounters two major limitations. The first is that remarkably little is known precisely about the transmission of several of these infections and the numbers of microbes needed to pass on the infections to susceptible people. The second is that the bulk of the excreted viruses, bacteria, and protozoa differ quantitatively rather than qualitatively in their transmission characteristics, making it easy to end up with a large, relatively uninformative category containing the majority of infections. Understanding these infections depends on some basic facts of transmission—especially latency, persistence of pathogens in the environment, and the infective dose for humans—and these and other key concepts are discussed before the environmental classification is set forth.

## Understanding Excreta-Related Infections

Excreta may be related to human disease in two ways (figure 2-1). The agents of many important infections escape in the body's excreta eventually to reach others—the first means of relation—and these are "excreted infections." In some cases the reservoir of infections escape in the body's excreta eventually to Because such infections cannot be controlled through changes in human excreta disposal practices, this study does not examine them. (A number of infections for which both man and other animals serve as a reservoir, however, have been included.)

The second way in which excreta relate to human disease is through the insect breeding that waste disposal often encourages. Insects may be a nuisance in themselves (as are flies, cockroaches, mosquitoes), but they may also mechanically transmit excreted pathogens either on their bodies or in their intestinal tracts (as do cockroaches and flies), and sometimes

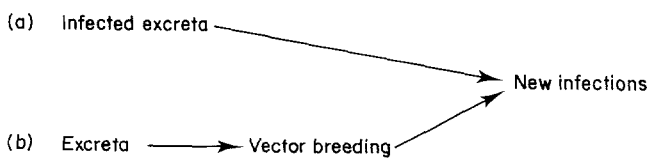


Figure 2-1. *The links between excreta and infection.* In (a), the excreta themselves contain the pathogens which may be transmitted by various routes to a new host. In (b), the excreta (or sewage) permits the breeding of certain flies and mosquitoes that may act as vectors of excreted and other pathogens

they may be vectors for pathogens that circulate in the blood (as are mosquitoes). The capacity of flies or cockroaches to serve as mechanical vehicles for excreted pathogens represents one of the many ways in which excreted disease agents are transmitted from anus to mouth. Careful disposal of human wastes, precautions in food storage and handling, and control measures directed against flies and cockroaches would minimize the threat to health of these pests. The blood-feeding nature of the mosquito, however, poses a more complex problem. The mosquito ingests agents of the diseases it transmits through biting already infected persons and perpetuates the cycle of infection to new hosts by the same means. The pathogens it carries are therefore nonexcreted, and the concepts discussed in this chapter have little relevance; the important factors are those which determine the breeding habits of those particular mosquito vectors that breed in sewage or sullage.[1]

The distinction between the state of being infected and the state of being ill must be kept in mind in considering the transmission of excreted infections. The most important segment of the population involved in transmitting an infection frequently shows few or no signs of disease; conversely, individuals in advanced states of disease may be of little or no importance in transmission. Schistosomiasis is a good example: as much as 80 percent of the total egg output in feces and urine reaching water from a human population may be produced by children 5 to 15 years old, many of whom will show minimal signs of disease. Conversely, middle-aged people in terminal stages of schistosomiasis may produce few or no viable schistosome eggs.

If an excreted infection is to spread, an infective dose of the disease agent has to pass from the excreta of a patient, carrier, or reservoir of the infection to the mouth or some other entryway of a susceptible person. Spread will depend upon the numbers of pathogens excreted, upon how these numbers change during the particular transmission route or life cycle, and upon the dose required to infect a new individual. Infective dose is in turn related to the susceptibility of the new host. Three key factors intervene to govern the probability that, for a given transmission route, the numbers of excreted pathogens (excreted load) from one host will form an infective dose for another: latency, persistence, and multiplication. These concepts will be discussed in turn; their relation is expressed in figure 2-2.

1. See category VI in the next main section of this chapter. The relation of insects to excreta and disease is examined in detail in chapters 36 and 37.

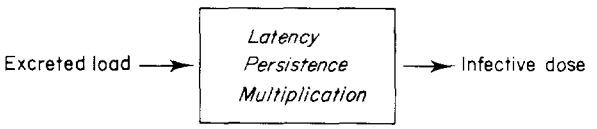


Figure 2-2. *Factors affecting the transmission of an infective dose*

### *Excreted load*

The concentration of pathogens passed by an infected person, or excreted load, varies widely. A person infected by a small number of nematode worms, for instance, may pass only a few eggs per gram of feces, whereas a cholera carrier may excrete $10^6$ vibrios per gram, and a patient with an acute attack of cholera may pass $10^{13}$ vibrios in a day. In areas where large numbers of pathogenic organisms are being passed in the feces, high pathogen concentrations in sewage are common (see table 1-10). Even in a developed, temperate country such as England, where water use is relatively high and salmonellosis relatively rare, raw sewage may contain $10^4$ salmonellae per liter. At these concentrations a removal efficiency of 99 percent in sewage works will still leave $10^2$ pathogenic organisms per liter of effluent. The health implications of these pathogens will depend upon the effluent disposal method, the pathogens' ability to survive or multiply, and the infective dose required.

### *Latency*

Latency is the interval between the excretion of a pathogen and its becoming infective to a new host. Some organisms—including all excreted viruses, bacteria, and protozoa—have no latent period and are immediately infectious in raw excreta. The requirements for the safe disposal of excreta containing these agents are different from those for helminthic infections which have prolonged latent periods. Latency can affect the choice of disposal systems: infections that have a considerable latent period are largely risk free in carted night soil, whereas the others constitute a major health hazard in fresh night soil. In the environmental classification that follows, therefore, the first two categories, in which no latency is observed, are separated from the remaining categories, in which a definite latent period occurs.

Among the helminthic infections (see table 1-9), only three have eggs or larvae that may be immediately infectious to man after being passed in the feces. These

are the pinworm (*Enterobius vermicularis*), a dwarf tapeworm (*Hymenolepis nana*), and occasionally a minute nematode (*Strongyloides stercoralis*). All the other excreted helminths require a distinct latent period, either because their eggs must develop into an infectious stage in the environment outside the body, or because these parasites have one or more intermediate hosts through which they must pass in order to complete their life cycles.

*Persistence*

Viability of the pathogen in the environment, or persistence, is a measure of how quickly it dies after leaving the human body. This single property is the most indicative of the fecal hazard: a highly persistent pathogen will create a risk throughout most treatment processes and during the reuse of excreta.

A pathogen with short persistence outside the body, however, must rapidly find a new, susceptible host. Transmission, therefore, cannot follow a long route through sewage works and the final effluent disposal site back to man, but will rather involve the family or other close group, within which infection is transferred from one person to another through lax personal cleanliness. More persistent organisms, in contrast, can readily generate new cases of disease much farther afield. As persistence increases, so then must concern for the ultimate means of excreta disposal. Similarly, pathogens that tend to persist in the general environment will require more elaborate processes to inactivate them in a sewage works. Methods of sequestering these pathogens, such as sedimentation into a sludge for special treatment, are often needed.

Measurement of pathogen persistence in a laboratory is easy. Laboratory results, however, need confirmation by field studies which are more difficult. Interpreting field results on persistence requires knowledge of how many pathogens are being shed in a community's excreta (relatively easy to determine) and the infective doses for man (extremely difficult).

*Multiplication*

Under favorable conditions certain pathogens will multiply in the environment. Originally low numbers can thus produce a potentially infective dose (see the next section). Bacteria may multiply on a favored substrate (for instance, *Salmonella* on food) and trematode worms multiply in their molluscan intermediate hosts. In the former case, light fecal contamination may increase bacterial numbers to the high minimal infective doses required in many excreted bacterial infections. This may determine the usual mode of infection, since multiplication in water is rare and limited compared with the massive increases possible in food. Excreted viruses and protozoa do not multiply outside their animal hosts.

Among the helminths transmitted by excreta, all the trematodes infecting man undergo multiplication in aquatic snails. This aquatic stage in their life cycles introduces a prolonged latent period of a month or more while the trematodes develop in the snail, followed by an output to the environment of up to several thousand larvae for each egg reaching the water. (Category v of the environmental classification below contains infections of this sort.)

*Infective dose*

In a predictable world the assessment of health risk could simply be calculated from the output of pathogens in the excreta of those infected, the median infective dose ($ID_{50}$) of particular organisms, and the efficiency of excreta treatment processes in inactivating pathogens. Because of the variable infective dose of most pathogens and the uneven distribution of infection in the environment, the real world is much less calculable than this. Although the minimal infective dose for some diseases may be a single organism, or very few, the doses required in most bacterial infections are much higher. Data on infective doses are very hard to acquire, since they involve administering a known dose of a pathogen to a human volunteer. Information is scanty and concerned with doses required to infect half those exposed ($ID_{50}$), rather than a small proportion, at a single exposure. The volunteers generally have been well-nourished adults usually from non-endemic areas. Results of this kind must therefore be applied with great caution to malnourished peasant children continually exposed to an infection. It has been found that changes in the manner of administering experimental doses, such as preceding a dose of cholera vibrios with an alkaline substance to reduce temporarily free gastric acid, may lower the $ID_{50}$ of such organisms by a factor of $10^3$ (Hornick and others 1971). And, although $ID_{50}$ may be the most reliable gauge of infectivity in human experimental studies, in natural transmission the infective dose for 5 percent or less of the population may be of greater epidemiological significance.

Uncertainty over the size of the minimal infective dose in nature makes it a difficult criterion to use in devising a classification; nevertheless, it is too important to be left out. The difficulties are greatest with the major excreted bacterial infections and with

protozoa. For excreted viruses there is evidence of low $ID_{50}$s in the laboratory, and in human populations (World Health Organization 1979). In helminthic infections a single egg or larva can infect if ingested, even though a high proportion of worms can fail to mature (especially in locations where immunity is present).

*Host response*

Host response is important in determining the effect once an individual has received a given dose of an infectious agent. Acquired immunity, and the relation of age to pathology, are particularly important in predicting the effects of sanitation. At one extreme would be infection with a short-lived parasite to which little immunity develops and for which the relation between infection and disease is not age dependent. A close, almost linear relationship between exposure and disease might be expected in this case, with appropriate improvements in sanitation yielding health benefits proportional to effect. *Ascaris* closely approximates this model.

At the other extreme would be infection with viruses or bacteria to which long-lasting immunity develops and for which the chance of overt, symptomatic disease in those infected rises with increasing age. An example of this case is infection with poliovirus (see table 1-5). Under poor sanitary conditions all persons are infected at a young age, older children and adults are immune, and disease is limited to a few of the youngest children, who may suffer chronic paralysis. If sanitation is improved, infection is deferred to later in life, when its pathological consequences are more serious. Thus, although poliovirus transmission may be reduced by improving sanitation, improvements will not necessarily curtail the disease, a result achieved in practice by immunization. This pattern may also apply to other excreted infections such as infectious hepatitis, and it has been proposed for typhoid. There are several other excreted infections, however, in which human immunity is of importance in regulating the amount of disease. Immunity tends to diminish the health significance of moderate sanitary improvements, and may in part explain the disappointing effects of some sanitary programs (table 2-1).[2]

In other words, the balance between exposure to infection and host response to it will determine the pattern of the excreta-related disease. If transmission, creating exposure to a particular infection, is limited, then most people will not have encountered the infection and will be susceptible. If a sudden increase in transmission of the disease occurs, it will affect all age groups in the form of an epidemic. Under these circumstances improvements in sanitation that strike at pathogen transmission will have a considerable

2. See also chapter 3 for a detailed discussion of the health benefits from improvements in sanitation.

Table 2-1. *Summary of selected literature on the effect on health of improved excreta disposal*

| *Country and type of study* | *Finding* | *Source* |
|---|---|---|
| *Brazil* | | |
| In a village of 1,041 inhabitants, a socioeconomic and schistosomiasis survey in 1961 was followed by introduction of schistosome control measures, including latrines, water supplies, laundry facilities, showers and health education. Fecal surveys were carried out in 1961, 1966, 1967 and 1968. Other villages without these interventions were surveyed in 1963 and 1969. | From 1961 to 1968, *Schistosoma mansoni* prevalence rates fell from 7 to 0 percent among 0–4 year olds, from 27 to 4 percent among 5–9 year olds and from 56 to 9 percent among 10–14 year olds. The prevalence of *S. mansoni* infection in domestic rodents and snails also fell considerably. The cost of the control measures was US$0.98 per month per protected person over 7 years. | Barbosa, Pinto and Souza (1971) |
| *Colombia* | | |
| 15 municipal primary schools in a poor suburb of Cali were visited and 8,444 schoolchildren were interviewed. The school's toilet facilities were inspected and the children were asked if they had had diarrhea, vomiting, colds or headlice over the past week. The observations of toilet facilities were used to compute a "hygienic score" for each school. | Diarrhea and vomiting were more common among children in schools with lower hygienic scores. The individual factors most associated with diarrhea prevalence were feces in the toilet bowl, and an absence of toilet paper, towels, soap or taps for hand washing. Hygienic scores were not related to colds or headlice, and classroom crowding was weakly related to vomiting, colds and headlice. | Koopman (1978) |

Table 2-1 (*continued*)

| *Country and type of study* | *Finding* | *Source* |
|---|---|---|
| *Costa Rica* | | |
| Diarrheal morbidity, intestinal bacteria, parasites, quality of water, meat and milk and the fly population were surveyed among 1,202 houses. Three types of excreta disposal facility were distinguished: none (12 percent of houses), pit latrine (76 percent of houses) and flush toilets with septic tanks (12 per cent of houses). | *Ascaris* prevalence decreased as the type of excreta disposal improved. *Trichuris* prevalence was the same among individuals with or without a latrine but was lower among individuals having a septic tank. *Shigella* organisms were not recovered where a septic tank was present. Diarrhea morbidity was *least* amongst those living in houses with no latrine. Excreta disposal facility was not associated with protozoal prevalence. | Moore, de la Cruz and Vargas-Mendez (1965) |
| An outbreak of 167 cases of infectious hepatitis was investigated between December 1963, and July 1964. The outbreak occurred during a severe drought. Person-to-person contact was considered the likely mode of spread. | Infectious hepatitis cases occurred in 1.6 percent of houses with a flush toilet, 2.7 percent of houses with an outdoor latrine and 2.6 percent of houses with no facility. | Villarejos and others (1966) |
| *Egypt* | | |
| Surveys were made in 1952 of helminthic and protozoal infections in two neighboring villages: A and B. Village A had been surveyed in 1950. Village A had improved water supply, a borehole latrine in 90 percent of houses, a refuse collection service and visiting nurses. Village B was untouched. | Protozoal prevalence rates and the mean number of protozoal infections per person were not reduced in Village A. *Ascaris* and hookworm prevalence rates and intensities were reduced. | Chandler (1953 and 1954) |
| Prisoners used bucket latrines and treated Nile water. Nearby villagers had no latrines and used untreated Nile water. Parasite infections of the villagers were compared with those of the prisoners after various periods of incarceration. | Schistosomiasis and hookworm prevalences in the local population were approximately 75 percent and 70–88 percent respectively. Among prisoners, the rates fell from 30 percent and 68 percent respectively to less than 20 percent in both cases after 5 years of incarceration and to about 10 percent after 12 years. Reinfection with *Ascaris* occurred regularly owing to contamination of sewage-irrigated vegetables. | Khalil (1931) |
| Various combinations of latrines and drug therapy were investigated in villages for 6 years from 1928. | Latrines had no impact on *Ascaris*, hookworm or schistosome infections. | Scott and Barlow (1938) |
| Various combinations of water supply, latrines, refuse disposal, fly control and therapy were investigated in 5 villages during 1948–51. | The installation of water supply and latrines did not alter the infant mortality or crude death rates and did not change the fly status in any of the villages. | Weir and others (1952) |
| *Guatemala* | | |
| Acute diarrheal rates among families having a latrine were compared with rates amongst those with no latrine. | In those families having a latrine, diarrheal rates were somewhat lower for those over 2 years old, but not for those under 2. | Gordon and others (1964) |
| Two lowland villages were studied during 1973–76. In-house water supply and sanitary education were implemented in one village; the other village provided a control. | The results of this study have not yet (mid-1981) been fully published. Preliminary reports indicate that malabsorption was somewhat lower in the intervention than the control village, that there were no differences in overall diarrhea incidence but that there was less diarrhea among 2–7 year olds in the intervention than the control village. | Schneider, Shiffman and Faigenblum (1978); Shiffman and others (1979) |

Table 2-1 (*continued*)

| *Country and type of study* | *Finding* | *Source* |
|---|---|---|
| *India* | | |
| The impact of a bored-hole pit latrine and health education program on the incidence of diarrhea in children in the village of Bharwara, near Lucknow, was investigated. | The authors state that the invervention was related to "a declining trend in diarrhoeal morbidity", but the data presented do not support this. | Kumar, Sehgal and Singh (1970) |
| A single stool examination on 13,267 hospital patients and their contacts was carried out at Karnal, Haryana State. A sanitary inspector was sent to the homes of the patients to collect information on hygiene and domestic facilities. | The prevalence of *Entamoeba histolytica* excretion among those living in homes with no latrines (38.3 percent) was a little higher than for those using latrines (31.6 percent) ($p<.01$). The authors point out that this difference cannot necessarily be attributed to the latrines. | Mathur and Kaur (1972) |
| *Iran* | | |
| Impact of mass treatment, sanitation and sanitation plus mass treatment on soil-transmitted helminths was studied in 15 villages in southwest Iran. Sanitation was one pit latrine per family and a communal water supply. | Mass treatment was highly effective in reducing both the prevalence and intensity of *Ancylostoma* and *Ascaris*. Sanitation, added to mass treatment, contributed nothing. Sanitation alone had an impact upon the intensity of both hookworm and roundworm and had a little impact on the prevalence of roundworm only. | Arfaa and others (1977) |
| Ascariasis was studied in a village of 850 people in southwest Iran before and after the construction of a water supply, a public bath-house, a laundry and 114 pit latrines (nearly one for every household). | The prevalence of infection fell from 67 percent to 57 percent over the study period (February 1963 to December 1965). Mean egg output fell from around 11 per milligram of feces to 4. The pit latrines cost US$0.5 per capita and were the major cause of the reduced ascariasis. | Sahba and Arfaa (1967) |
| *Japan* | | |
| A program of heat treating (with firewood) of night soil (up to 60°C) prior to agricultural application was implemented in a village in Shiga Prefecture. A control village was left untouched. | The prevalence of hookworm and *Ascaris* declined "strikingly" in the intervention village and there was a marked decrease in the count of *Ascaris* eggs found in the soil. These changes were not observed in the control village. | Katayama (1955) |
| Heat treating of night soil (with surplus night electricity) was implemented in a village near Osaka city. | Night soil treatment alone had only a slight effect on the prevalence of parasite infections. When mass chemotherapy was carried out, prevalences fell markedly (hookworm from 52 percent to 11 percent, *Ascaris* from 33 percent to 12 percent) and remained at this low level throughout the 5 month observation period. | Kawagoe and others (1958) |
| Night soil treatment with thiabendazole was implemented in a village of 5,000 people near Tokyo. Three areas were distinguished: Area A, night soil treatment + chemotherapy; Area B, night soil treatment only; Area C, chemotherapy only. Parasite prevalence was surveyed between July 1964 and March 1966. | The prevalence of ascariasis fell by 50 percent in Area A, by 30 percent in Area B and hardly at all in Area C. The rate of new infections with *Trichuris* was one-third, and that of hookworm was one-half, in Area A compared with Area C. | Kutsumi (1969) |
| *Mauritius* | | |
| Diarrheal rates in households with differing sanitation facilities were studied in 1960. | Compared with families with an indoor toilet, families with an outdoor toilet had 4 times the diarrhea incidence and families with no toilet had 10 times the diarrhea incidence. | van Zijl (1966) |
| *Panama* | | |
| A series of egg counts were made in two villages, one partially sanitated and the other entirely without latrines, before and after mass chemotherapy. | Reinfection after mass treatment was rapid, but reinfection with hookworm was delayed in those groups with more and better maintained latrines. | Cort, Schapiro and Stoll (1929) |

Table 2-1 (*continued*)

| *Country and type of study* | *Finding* | *Source* |
|---|---|---|
| *Panama (cont.)* | | |
| Children presenting at a clinic in Panama City were examined for excretion of enteropathogenic *Escherichia coli*, *Shigella* and *Salmonella*. These data were related to information about type of housing and sanitary facilities. | The three pathogens were absent from children coming from the best housing type, whereas for other housing types, about 8 percent of children had one or more of the pathogens in their feces. | Kourany and Vásquez (1969) |
| Surveys were conducted over 7 years into environmental conditions and helminthiases. | In villages without latrines, the prevalence and intensity of hookworm rose to, or above, original levels within 3 or 4 years after a mass drug campaign. In villages with latrines, prevalence and intensity also rose again following drug treatment but a degree of protection against reinfection was observed among women. | Sweet and others (1929) |
| *Philippines* | | |
| A region with endemic cholera was divided into 4 areas: Area A, control area having poor water and sanitation facilities; Area B, improved water supply; Area C, pour-flush pit latrines; Area D, improved water supply and communal latrines. | The apparent reductions in cholera incidence were 73 percent in Area B, 68 percent in Area C and 76 percent in Area D. | Azurin and Alvero (1974) |
| *Singapore* | | |
| 159 families living in modern flats and 169 families living in squatter housing were studied. The people in the flats had previously lived in the squatter housing but had been rehoused following a fire in 1961. Average family income of flat dwellers was S$165 per month whereas for squatters it was S$130 per month. Stool samples were collected from all children under 13 years old in the selected households. | *Ascaris*, hookworm and *Trichuris* prevalence rates were 9, 1 and 28 percent, respectively, among flat dwellers and 63, 2 and 58 percent among squatters. The high *Trichuris* prevalence among flat dwellers was attributed to the longevity of this worm. | Kleevens (1966) |
| *St. Lucia* | | |
| A longitudinal study of 229 children in three valleys. Weights and heights were recorded monthly; stools were examined for worm eggs every 6 months, and parents kept diarrhea diaries for their children. The children were 0–6 months old at the start of the study and were followed for 2 years. | Children in the valley with improved water and the valley with improved water and latrines had less ascariasis, trichuriasis and diarrhea, and grew better, than children in the valley with no improvements. | Henry (1981) |
| *Sudan* | | |
| Diarrhea incidence in households with differing sanitary facilities were studied in 1961. | In one particular month, families having a communal unsanitary privy experienced a higher diarrheal morbidity rate than similar families having no toilet. | van Zijl (1966) |
| *Union of Soviet Socialist Republics* | | |
| A village of 1,600 people was studied before and after the abolition of untreated night soil as a fertilizer and a campaign of "improving general hygiene." | Before the intervention, the prevalence of *Ascaris* eggs was 100 percent in soil samples and 71 percent in fruit samples. 41 percent of soil eggs and 19 percent of fruit eggs were viable. After the intervention, 35 percent of soil samples and 25 percent of fruit samples contained eggs. No eggs were viable. | Rosenberg (1960) |

Table 2-1 (*continued*)

| *Country and type of study* | *Finding* | *Source* |
|---|---|---|
| *United States* | | |
| 400 patients at a veterans' hospital in Georgia had stool examinations for intestinal protozoa and helminths, and completed questionnaires on their military service and living conditions. | The overall prevalence of infection with *Entamoeba histolytica* was 9.3 percent. Among those not infected, 22 percent had outside toilets, whereas among those infected, 55 percent had outside toilets ($p < .01$). Income was not significantly associated with *Ent. histolytica* infection. | Brooke, Donaldson and Brown (1954) |
| A survey of 357 people in 4 areas near Little Rock, Arkansas, was carried out in 1961. Stools were examined for intestinal protozoa. | The overall prevalence of infection with one or more protozoon (APR) was 33 percent. Among all individuals served with piped indoor water supply the APR was 31 percent, whereas among those using well water it was 35 percent (no significant difference). However, among 0–4 year old piped water users the APR was 13 percent, whereas among 0–4 year old well-water users it was 37 percent ($p < .05$). Many of the houses with piped water also had sewerage, whereas well-water houses had septic tanks or outside pit latrines. | Brooke and others (1963) |
| 2,657 people living in a rural area of West Tennessee were surveyed for intestinal parasites. 90 percent were black. Details of family size, cleanliness, housing, water supply and excreta disposal were also collected. | *Entamoeba histolytica* and *Ascaris* prevalence rates were 19 and 8 percent, respectively, among those with clean latrines, 36 and 11 percent among those with dirty latrines, and 29 and 15 percent among those with no latrines. Parasite prevalence was also found to be associated with family size, fecal contamination of the premises, cleanliness of house and person but not with water pollution. | Eyles, Jones and Smith (1953) |
| A survey of shigellosis among children under 10 years old in farm labor camps in California was conducted. | The prevalence rates of *Shigella* excretion were 1.6 percent in cabins with inside water, shower and toilet, 3.0 percent in cabins with inside water but shared shower and toilet facilities, and 5.8 percent in cabins with all services shared. | Hollister and others (1955) |
| White females (age 18–76 years) at a mental institution in California were studied during 1954–57. They were originally housed in an old building in which standards of sanitation were poor. They were then rehoused in a new, modern hospital building with excellent sanitary facilities. Stool examinations were made on 110 patients prior to rehousing and on 8 subsequent occasions. | The percentage of people infected with *Ent. histolytica* and *Giardia lamblia* rose steadily during the survey, indicating that transmission was continuing throughout the period. However, although the percentage of people infected with hookworm (73 percent) and *Trichuris* (83 percent) remained constant, as would be expected in the absence of mass chemotherapy, no new cases of hookworm and only 3 new cases of *Trichuris* were reported while the patients were in the new building. Thus, the move to the new building interrupted the transmission of the helminths but not the protozoa. | Jeffery (1960) |
| In 1952 a program of borehole latrines was implemented in Boston, Georgia. The prevalence of *Shigella* excretion, in Boston and control towns, was surveyed in children under 10 years old. | The latrine program was associated with a reduction in the detection of *Shigella* from rectal swabs from 4.7 percent to 2.8 percent. Rates in control towns did not fall over this period. | McCabe and Haines (1957) |

Table 2-1 (*continued*)

| *Country and type of study* | *Finding* | *Source* |
|---|---|---|
| *United States (cont.)* | | |
| Excretion of *Entamoeba histolytica* among 1,115 urban school children in North Carolina was studied. These data were related to excreta disposal, water supply and garbage disposal facilities in the homes of the children. | *Ent. histolytica* prevalence rates were 6 percent for those with an inside flush toilet, 12 percent for those with a shared flush toilet and 58 percent for those with a pit latrine. Infection with *Ent. histolytica* was also associated with type of water supply and garbage disposal facilities. | Mackie and others (1956) |
| Hookworm and *Ascaris* surveys were conducted in Virginia. | The introduction of pit privies in the mountainous areas of Virginia was effective in reducing the hookworm prevalence, but not *Ascaris*. | Otto and Spindler (1930) |
| Environmental studies were made of 329 families in the mountain region of Tennessee and 202 families living in the central basin, western plains and lowlands of the state. | *Ascaris* and *Trichuris* infections were confined largely to the mountain areas. Yard pollution, and with it heavy *Ascaris* infection, were present regardless of the presence or absence of latrines. | Otto, Cort and Keller (1931) |
| Studies were conducted in 11 mining camps in eastern Kentucky from 1954 to 1957. Reported diarrheal disease rates, *Shigella* isolations from rectal swabs of pre-school children and parasite prevalence were investigated. | *Shigella* and *Ascaris* prevalence rates were 1.1 and 7 percent, respectively, among those with water and flush toilet inside, 2.4 and 25 percent among those with water inside and latrine outside, and 5.9 and 42 percent among those with water and latrine outside. | Schliessmann and others (1958) |
| *Shigella* infection data from 28,000 rectal swabs were analysed according to the type of housing. Housing was divided into 4 categories (poor, fair, good, very good) according to water supply, excreta disposal, fly population and esthetic and structural quality. | The rates of new *Shigella* infections occurring during the study period were:<br>6.2 percent among those in "poor" houses,<br>2.2 percent among those in "fair" houses,<br>0.6 percent among those in "good" houses and<br>0.3 percent among those in "very good" houses. | Stewart and others (1955) |

*Note*: The limitations of the literature on health benefits from sanitation and the difficulties in assessing these benefits are discussed in chapter 3.

effect in reducing an epidemic's likelihood and its magnitude if one occurs.

By contrast, if transmission is vigorous, most people will be repeatedly exposed to an infection, having first acquired it in childhood. Subsequent exposures may be without effect if immunity is developed after the first attack, or immunity may develop cumulatively from a series of attacks. The infection will nevertheless always be present, and can be described as endemic. Under these conditions much of the transmission is ineffective because of human acquired immunity, and reduced transmission through improved sanitation will only delay the occurrence of infection somewhat, so that older children exhibit symptoms. Extensive sanitary improvements will either render the infection rare or, if the disease was originally highly transmitted, make it an adult disease. Diseases exemplifying this state of affairs are typhoid, which can be completely prevented in a community by adequate management of excreta and of water supplies, and poliomyelitis, which can be prevented only by immunization.

The consequences of a disease's juvenile prevalence—not only that children chiefly suffer, but also that children are the main sources of infection—presents a further challenge to sanitation. The acute need for better community excreta disposal must focus on young children, the group perhaps least inclined to use any facilities that are made available.

### *Nonhuman hosts*

Some excreted infections (for example, shigellosis) are confined strictly to humans, and the control of human excreta alone is required for their prevention. Many others (for example, salmonellosis) involve wild or domestic vertebrate animals as well as man. Such an infection is called a zoonosis.

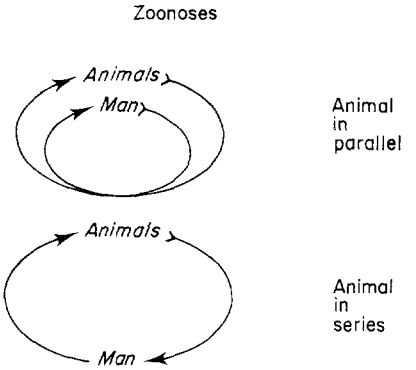


Figure 2-3. *Involvement of other vertebrates in transmission of human excreted infections.* Examples of zoonoses in parallel are salmonellosis and balantidiasis; examples of zoonoses in series are beef and pork tapeworm infections

There are two groups of zoonoses, and each has quite different implications for sanitation (figure 2-3). In the first group, animals act as hosts alternative to man: even if human excreta is under completely safe control, the excreta of other animals can continue to transmit the infection. In effect, the animal involved is "in parallel" with man, and it is necessary to control both human and animal excreta. In the second group, the animal is an essential step in the transmission of the disease from one human to another (figure 2-3, "in series"). In this case, control of either human excreta alone or the animal infection alone will suffice to end transmission. In the environmental classification below, this second group which contains the human tapeworms of the genus *Taenia*, is therefore separated from the other categories.

Some excreted helminthic infections have invertebrate intermediate hosts (see table 1-9); they will be controlled if excreta are prevented from reaching the intermediate hosts, or the intermediate hosts are controlled, or if people do not eat the intermediate host uncooked or do not have contact with the water in which the intermediate host lives (depending on the particular organism's life cycle).

## Categories of Excreta-related Infections

There are several ways in which the excreted infections can be grouped according to the epidemiological features discussed above, but a classification that considers the effects of excreta disposal and changes in disposal facilities and technologies has been chosen, and is given in table 2-2. Six categories of infection have been distinguished in the table, and the relevant environmental or epidemiological features broadly considered are latency, infective dose, persistence, multiplication, and transmission. Further data on specific excreted pathogens—arranged by category and epidemiological feature—are provided in table 2-3.[3] Control measures appropriate to each environmental category of pathogen are indicated in table 2-2, and data on immunity and pathogen concentrations in excreta, which vary with each organism, are contained in table 2-3.

There is a clear difference between the first five categories of excreted pathogens and the last, which contains excreta-breeding insect vectors of disease, in that the insects themselves are not pathogens and that a variety of sanitation methods and additional specific measures can be directed against these vectors. For these reasons category VI is not included in table 2-3.

The excreted infections are divided on the basis of the presence (categories III to V) or absence (categories I and II) of a latent period (health problems associated with fresh feces or night soil occur primarily in these first two categories). The distinction between categories I and II and categories III to V is fundamental and clear-cut, corresponding closely to the biology of the pathogens (in that all infections in categories III to V are helminthic).

The subdivisions of the infections having latency are also clear, with category III containing the soil-transmitted worms, IV the tapeworms, which depend on the access of cattle and pigs to human feces, and V the trematodes and other worms requiring aquatic intermediate hosts. The subdivision of categories I and II, however, is difficult and somewhat arbitrary because the various concepts discussed above can arrange the infections of these categories in different ways. If categories I and II are divided, for instance, on the basis of $ID_{50}$ with the grave limitations of the available data on infective dose kept in mind, the approximate ranking of pathogens (in order of increasing $ID_{50}$) shown in table 2-4 emerges. But if the infections are listed in the order of increasing persistence outside their animal host, the approximate ranking shown in table 2-5 is appropriate. Another important factor in predicting the effects of improved excreta disposal facilities is whether or not a significant nonhuman reservoir of infection (see figure 2-3) exists for a

3. Part Two of the book is devoted to detailed analyses of individual pathogens and diseases according to these and additional environmental factors. But it was thought that, for easier reference, Part Two should group the pathogens by kind and not by the categories described in this chapter. Part Two is divided into section I, the excreted viruses; section II, the excreted bacteria; section III, the excreted protozoa; section IV, the excreted helminths; and section V, the excreta-breeding insects and the diseases they transmit.

Table 2-2. *Environmental classification of excreted infections*

| *Category and epidemiological features*[a] | *Infection* | *Environmental transmission focus* | *Major control measure* |
|---|---|---|---|
| I. Non-latent; low infective dose | Amebiasis<br>Balantidiasis<br>Enterobiasis<br>Enteroviral infections[b]<br>Giardiasis<br>Hymenolepiasis<br>Infectious hepatitis<br>Rotavirus infection | Personal<br>Domestic | Domestic water supply<br>Health education<br>Improved housing<br>Provision of toilets |
| II. Non-latent; medium or high infective dose; moderately persistent; able to multiply | *Campylobacter* infection<br>Cholera<br>Pathogenic *Escherichia coli* infection[c]<br>Salmonellosis<br>Shigellosis<br>Typhoid<br>Yersiniosis | Personal<br>Domestic<br>Water<br>Crop | Domestic water supply<br>Health education<br>Improved housing<br>Provision of toilets<br>Treatment of excreta prior to discharge or reuse |
| III. Latent and persistent; no intermediate host | Ascariasis<br>Hookworm infection[d]<br>Strongyloidiasis<br>Trichuriasis | Yard<br>Field<br>Crop | Provisions of toilets<br>Treatment of excreta prior to land application |
| IV. Latent and persistent; cow or pig as intermediate host | Taeniasis | Yard<br>Field<br>Fodder | Provision of toilets<br>Treatment of excreta prior to land application<br>Cooking, meat inspection |
| V. Latent and persistent; aquatic intermediate host(s) | Clonorchiasis<br>Diphyllobothriasis<br>Fascioliasis<br>Fasciolopsiasis<br>Gastrodiscoidiasis<br>Heterophyiasis<br>Metagonimiasis<br>Opisthorchiasis<br>Paragonimiasis<br>Schistosomiasis | Water | Provision of toilets<br>Treatment of excreta prior to discharge<br>Control of animal reservoirs<br>Control of intermediate hosts<br>Cooking of water plants and fish<br>Reducing water contact |
| IV. Spread by excreta-related insects | Bancroftian filariasis (transmitted by *Culex pipiens*)<br>All the infections in I-V able to be transmitted mechanically by flies and cockroaches | Various fecally contaminated sites in which insects breed | Identification and elimination of suitable insect breeding sites |

a. See table 2-3 for data on additional epidemiological features by pathogen.
b. Includes polio-, echo-, and coxsackievirus infections.
c. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli* infections.
d. *Ancylostoma duodenale* and *Necator americanus*.

Table 2-3. *Basic epidemiological features of excreted pathogens by environmental category*

| *Pathogen* | *Excreted load*[a] | *Latency*[b] | *Persistence*[c] | *Multiplication outside human host* | *Median infective dose* ($ID_{50}$) | *Significant immunity?* | *Major nonhuman reservoir?* | *Intermediate host* |
|---|---|---|---|---|---|---|---|---|
| CATEGORY I | | | | | | | | |
| Enteroviruses[d] | $10^7$ | 0 | 3 months | No | L | Yes | No | None |
| Hepatitis A virus | $10^6$(?) | 0 | ? | No | L(?) | Yes | No | None |
| Rotavirus | $10^6$(?) | 0 | ? | No | L(?) | Yes | No(?) | None |
| *Balantidium coli* | ? | 0 | ? | No | L(?) | No(?) | Yes | None |
| *Entamoeba histolytica* | $10^5$ | 0 | 25 days | No | L | No(?) | No | None |
| *Giardia lamblia* | $10^5$ | 0 | 25 days | No | L | No(?) | Yes | None |
| *Enterobius vermicularis* | Not usually found in feces | 0 | 7 days | No | L | No | No | None |
| *Hymenolepis nana* | ? | 0 | 1 month | No | L | Yes(?) | No(?) | None |
| CATEGORY II | | | | | | | | |
| *Campylobacter fetus* ssp. *jejuni* | $10^7$ | 0 | 7 days | Yes[e] | H(?) | ? | Yes | None |
| Pathogenic *Escherichia coli*[f] | $10^8$ | 0 | 3 months | Yes | H | Yes(?) | No(?) | None |
| *Salmonella* | | | | | | | | |
| *S. typhi* | $10^8$ | 0 | 2 months | Yes[e] | H | Yes | No | None |
| Other salmonellae | $10^8$ | 0 | 3 months | Yes[e] | H | No | Yes | None |
| *Shigella* spp. | $10^7$ | 0 | 1 month | Yes[e] | M | No | No | None |
| *Vibrio cholerae* | $10^7$ | 0 | 1 month(?) | Yes | H | Yes(?) | No | None |
| *Yersinia enterocolitica* | $10^5$ | 0 | 3 months | Yes | H(?) | No | Yes | None |
| CATEGORY III | | | | | | | | |
| *Ascaris lumbricoides* | $10^4$ | 10 days | 1 year | No | L | No | No | None |
| Hookworms[g] | $10^2$ | 7 days | 3 months | No | L | No | No | None |
| *Strongyloides stercoralis* | 10 | 3 days | 3 weeks (free-living stage much longer) | Yes | L | Yes | No | None |
| *Trichuris trichiura* | $10^3$ | 20 days | 9 months | No | L | No | No | None |
| CATEGORY IV | | | | | | | | |
| *Taenia saginata* and *T. solium*[h] | $10^4$ | 2 months | 9 months | No | L | No | No | Cow (*T. saginata*) or pig (*T. solium*) |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| CATEGORY V | | | | | | | | |
| *Clonorchis sinensis*[i] | $10^2$ | 6 weeks | Life of fish | Yes[j] | L | No | Yes | Snail and fish |
| *Diphyllobothrium latum*[i] | $10^4$ | 2 months | Life of fish | No | L | No | Yes | Copepod and fish |
| *Fasciola hepatica*[h] | ? | 2 months | 4 months | Yes[j] | L | No | Yes | Snail and aquatic plant |
| *Fasciolopsis buski*[h] | $10^3$ | 2 months | ? | Yes[j] | L | No | Yes | Snail and aquatic plant |
| *Gastrodiscoides hominis*[h] | ? | 2 months(?) | ? | Yes[j] | L | No | Yes | Snail and aquatic plant |
| *Heterophyes heterophyes*[i] | ? | 6 weeks | Life of fish | Yes[j] | L | No | Yes | Snail and fish |
| *Metagonimus yokogawai*[i] | ? | 6 weeks(?) | Life of fish | Yes[j] | L | No | Yes | Snail and fish |
| *Paragonimus westermani*[i] | ? | 4 months | Life of crab | Yes[j] | L | No | Yes | Snail and crab or crayfish |
| *Schistosoma* | | | | | | | | |
| *S. haematobium*[h] | 4 per milliliter of urine | 5 weeks | 2 days | Yes[j] | L | Yes | No | Snail |
| *S. japonicum*[h] | 40 | 7 weeks | 2 days | Yes[j] | L | Yes | Yes | Snail |
| *S. mansoni*[h] | 40 | 4 weeks | 2 days | Yes[j] | L | ? | No | Snail |
| *Leptospira* spp.[k] | urine(?) | 0 | 7 days | No | L | Yes(?) | Yes | None |

L Low ($<10^2$); M medium ($\approx 10^4$); H high ($>10^6$).

? Uncertain.

a. Typical average number of organisms per gram of feces (except for *Schistosoma haematobium* and *Leptospira*, which occur in urine).

b. Typical minimum time from excretion to infectivity.

c. Estimated maximum life of infective stage at 20°–30°C.

d. Includes polio-, echo-, and coxsackieviruses.

e. Multiplication takes place predominantly on food.

f. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli*.

g. *Ancylostoma duodenale* and *Necator americanus*.

h. Latency is minimum time from excretion by man to potential reinfection of man. Persistence here refers to maximum survival time of final infective stage. Life cycle involves one intermediate host.

i. Latency and persistence as for *Taenia*. Life cycle involves two intermediate hosts.

j. Multiplication takes place in intermediate snail host.

k. For the reasons given in chapter 1, *Leptospira* spp. do not fit any of the categories defined in table 2-2.

Table 2-4. *Category I and II pathogens (from table 2-2) ranked by median infective dose* ($ID_{50}$)

| Pathogen | $ID_{50}$ |
|---|---|
| *Balantidium coli* (?) | L |
| *Entamoeba histolytica* | |
| *Enterobius vermicularis* | |
| Enteroviruses[a] | |
| *Giardia lamblia* | |
| Hepatitis A virus (?) | |
| *Hymenolepis nana* | |
| Rotavirus (?) | |
| *Shigella* | M |
| *Campylobacter fetus* ssp. *jejuni* (?) | H |
| Pathogenic *Escherichia coli*[b] | |
| *Salmonella* | |
| *S. typhi* | |
| Other salmonellae | |
| *Vibrio cholerae* | |
| *Yersinia enterocolitica* (?) | |

L Low ($<10^2$); M medium ($\approx 10^4$); H ($>10^6$).
? Uncertain.
a. Includes polio-, echo-, and coxsackieviruses.
b. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli*.

Table 2-5. *Category I and II pathogens (from table 2-2) ranked by persistence outside host*

| Pathogen | Persistence |
|---|---|
| *Balantidium coli* | L |
| *Campylobacter fetus* spp. *jejuni* (?) | |
| *Entamoeba histolytica* | |
| *Enterobius vermicularis* | |
| *Giardia lamblia* | |
| *Hymenolepis nana* | |
| *Salmonella typhi* | M |
| *Shigella* spp. | |
| *Vibrio cholerae* | |
| Enteroviruses[a] | H |
| Pathogenic *Escherichia coli*[b] | |
| Salmonellae | |
| *Yersinia enterocolitica* | |

L Low (<1 month); M medium ($\approx$ 1 month); H high (> 1 month).
? Uncertain.
a. Includes polio-, echo-, and coxsackieviruses.
b. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli*.

particular pathogen; four of the pathogens in categories I and II (*Campylobacter*, salmonellae *Balantidium coli* and *Giardia lamblia*) have significant animal reservoirs (table 2-3).

Socioeconomic considerations would divide the infections in categories I and II in yet another way. Infections that are commonly transmitted in affluent communities (in Europe, for instance) that enjoy high standards in sanitary facilities and hygiene might be expected to be reduced insignificantly by the introduction of limited sanitary improvements in poor communities of the developing countries. An approximate division on these grounds is shown in table 2-6. In some cases the reasons for this division are clear (the salmonellae, for instance, continue to be transmitted from animals to man in affluent communities through contaminated foodstuffs), whereas in other cases (such as the continued transmission of *Shigella sonnei* throughout Europe) they are obscure.

The most useful division of categories I and II has nevertheless proved to be one based on $ID_{50}$, even though knowledge of the $ID_{50}$ for infections affecting malnourished peasant children in the tropics is nonexistent. With $ID_{50}$ as the criterion, categories I and II break in a way that makes theoretical sense and also

Table 2-6. $ID_{50}$ *and persistence of category I and II pathogens (from table 2-2) commonly and rarely transmitted in affluent European communities*

| Pathogen | $ID_{50}$ | Persistence |
|---|---|---|
| *Commonly transmitted* | | |
| *Campylobacter fetus* ssp. *jejuni* | H(?) | L(?) |
| *Enterobius vermicularis* | L | L |
| Enteroviruses[a] | L | H |
| Pathogenic *Escherichia coli*[b] | H | H |
| *Giardia lamblia* | L | L |
| Hepatitis A virus | L(?) | ? |
| Rotavirus | L(?) | ? |
| Salmonellae | H | H |
| *Shigella sonnei* | M | M |
| *Yersinia enterocolitica* | H(?) | H |
| *Rarely transmitted* | | |
| *Balantidium coli* | L(?) | L |
| *Entamoeba histolytica* | L | L |
| *Hymenolepis nana* | L | L |
| *Salmonella typhi* | H | M |
| *Shigella* (other than *sonnei*) | M | M |
| *Vibrio cholerae* | H | M |

L Low: M medium; H high.
? Uncertain.
a. Includes polio-, echo-, and coxsackieviruses.
b. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli*.

correlates in some degree with the likely effects of improved excreta disposal facilities.

The transmission characteristics of the first five categories are illustrated in figure 2-4, in which the typical survival, latency, and multiplication features of the groups of infections are shown. These factors, in turn, affect the "length" of particular transmission cycles. Length has spatial as well as temporal implications, in that a long transmission cycle increases the opportunity of an infection's spreading over a wider area, thus changing the pattern of risk. These issues are developed in the next chapter, and are represented here in figure 2-5, in which the relative efficiency of sanitation improvements in controlling the various categories of infection is also indicated. Each category in table 2-2 implies some minimum sanitary requirements for control of the diseases within it and often control measures ancillary to excreta disposal facilities that further contribute to success. These requirements are elaborated in the discussion that follows.

*Category I*

These are the infections that have a low $ID_{50}$ ($<10^2$) and are infective immediately upon excretion. We argue that these infections may spread easily from person to person whenever personal and domestic hygiene are not ideal (see figure 2-5). It is therefore likely that changes in excreta disposal technology will have little effect on the incidence of these infections if such changes are unaccompained by sweeping changes in personal cleanliness, which, in turn, may require major improvements in water supply and housing, and major efforts in health education.

But what subsequently happens to excreta—how they are transported, treated, and reused—is of less importance for this group than the transmission of infection in the home. Although transmission can and does occur by more complex routes, most transmission in category I is direct, from person to person, and thus the provision of hygienic toilets alone will have negligible impact. A qualification of category I must follow this statement: categories I and II grade into each other and actually form a continuum (see a further explanation in the next section). In particular, the parasitic protozoa have some features of both groups. One extreme of the category I parasites is the pinworm, *Enterobius*, whose sticky eggs are laid on the anal skin by emerging females, so that transmission is by way of scratching fingers rather than by excretion of eggs in the feces. At the other extreme is *Giardia*, associated with well-documented, waterborne outbreaks of diarrhea, and therefore presumably subject to partial control by excreta management.

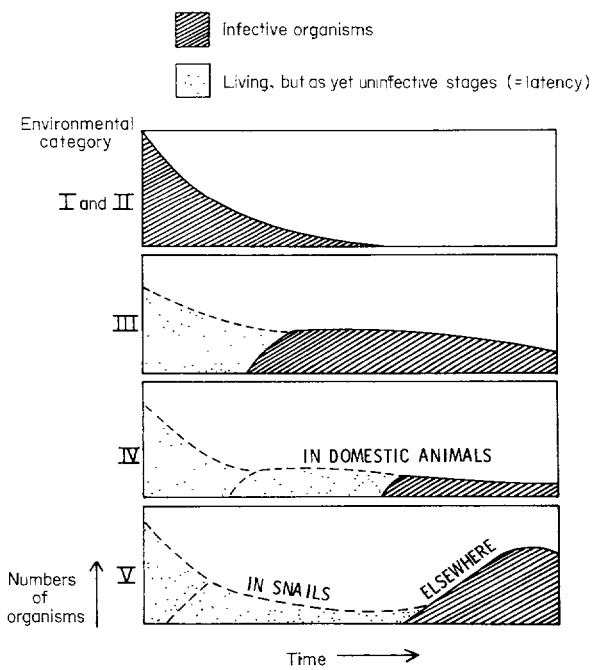


Figure 2-4. *Persistence outside the host of excreted pathogens (categories I–V from table 2-2) over time*

*Category II*

The infections in this category are all bacterial. They have medium or high $ID_{50}$s ($\geq 10^4$), and so are less likely to be transmitted by person-to-person contact than are category I infections. The bacteria are persistent and can multiply, so that even the small numbers remaining a few weeks after excretion can, if they find a suitable substrate (such as food), multiply to form an infective dose. Direct transmission routes are important, but so too are others with longer environmental cycles, such as the contamination of water sources or crops with fecal material (see figure 2-5). The control measures listed in table 2-2 for category I are important with the added provisions of sound excreta treatment and reuse practice. But, as in category I, changes in excreta disposal and treatment practices alone may have little effect on transmission. Control measures may most affect those infections that—as noted earlier—are not normally transmitted among affluent groups in Europe or elsewhere: cholera, typhoid, and shigellosis (other than *S. sonnei*). Any monitoring or evaluation program would do well to examine these, rather than infections with nontyphoid salmonellae or pathogenic *E. coli*.

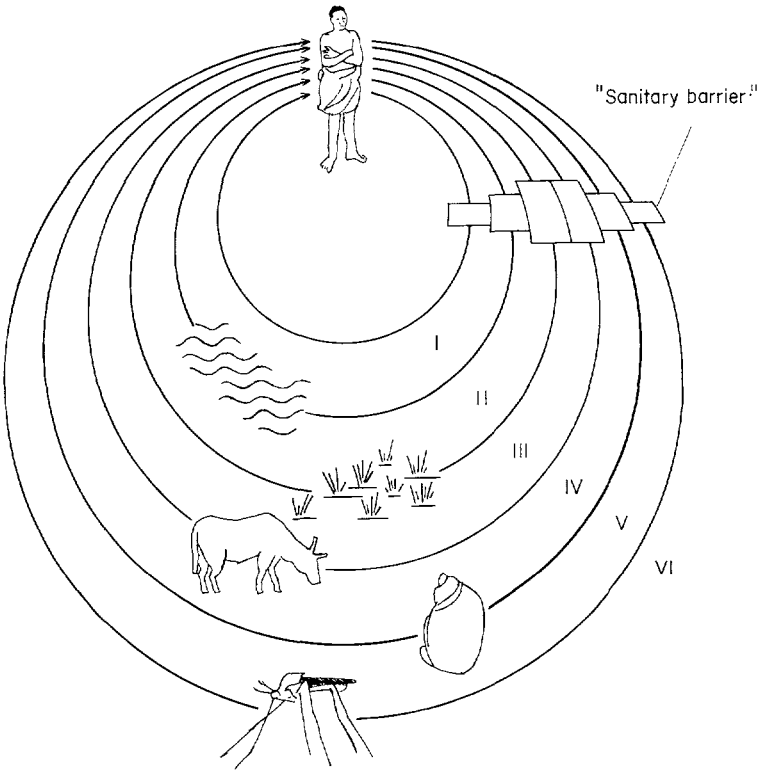


Figure 2-5. *Length and dispersion of transmission cycles of excreted infections (categories I–VI from Table 2-2).* The possible efficacy of improved excreta disposal is indicated by the "sanitary barrier"

The criteria used to differentiate categories I and II have been $ID_{50}$ and length of the environmental cycle, factors with predictive value for the efficacy of sanitation as a control measure. The reason that categories I and II do not form tidy groups is that the persistences of the pathogens involved vary. The extreme category I case—an environmentally fragile organism with a low $ID_{50}$—will clearly tend to be spread in a familial or similar tight pattern and will depend for its control more on personal cleanliness than on sanitation. (An extreme example, though not excreta-transmitted, can be found in the venereal diseases, which do not survive in the environment and depend on intimate contact for their spread.) However, an environmentally persistent organism with a low $ID_{50}$ will lead to infection difficult to reduce either by sanitation or personal and domestic cleanliness. Many excreted viruses exemplify this pattern and pose such major problems of control that induced immunity may be the best solution (this is certainly the case for poliomyelitis and probably also for infectious hepatitis and rotavirus diarrhea). For the infections of category II, the role of sanitation improvement is to interfere with the efficiency of the longer cycles and thus obtain a greater overall benefit than that possible for category I, in which these longer cycles have little significance.

### *Category III*

This category contains the soil-transmitted helminths, which are both latent and persistent (see figure 2-4). Their transmission has little or nothing to do with personal cleanliness because these helminth eggs are not immediately infective to man. Domestic cleanliness is relevant only as it concerns the preparation of vegetables grown in fields enriched by human excreta or the maintenance of latrines in conditions that do not allow helminth eggs to remain in the vicinity for the duration of their latency. If eggs are not deposited in soil, or other suitable media, transmission will not occur. Any kind of latrine that contains or removes excreta and does not permit contamination of the floor, yard, or fields, will therefore limit transmission. Because the persistence of helminth eggs is so long (see

table 2-3), it is not sufficient simply to prevent infected fresh feces from reaching the yard or fields: any fecal product that has not been adequately treated must not reach the soil. In societies that reuse their excreta on the land, treatment prior to application is therefore vital. Effective treatment for the removal of helminth eggs generally requires waste stabilization ponds or thermophilic digestion,[4] although prolonged storage will inactivate the eggs of many species.

*Category IV*

Category IV is for the beef and pork tapeworms. Any disposal system that prevents untreated human excreta from being eaten by cattle and pigs will control the transmission of these infections (see figure 2-5). Cattle are likely to be infected in fields treated with sewage sludge or effluent and may also eat feces deposited in the cowshed. Pigs are likely to become infected by eating human feces deposited around the dwelling or in the pigpen. The provision of toilets to which pigs and cattle cannot have access, and the treatment of all wastes prior to land application, are the necessary control methods. Measures to prevent birds, especially gulls, from feeding on trickling filters and sludge drying beds and subsequently depositing tapeworm eggs in their droppings on pastures are also required. In the absence of the measures described above, however, the thorough cooking of beef and pork is the most important control measure. Personal and domestic cleanliness, except the use and maintenance of safe toilets, are ineffective controls.

*Category V*

This category contains the water-based helminths, which need an aquatic host or hosts to complete their life cycles. Control is achieved by preventing untreated excreta or sewage from reaching water in which these aquatic hosts live (see figure 2-5). Any land application or dry composting system will therefore reduce transmission. There are two complications. First, in all cases except *Schistosoma mansoni* and *S. haematobium*, animals are an important reservoir of infection (see tables 1-9 and 2-3), and any measures restricted solely to human excreta can only have a partial effect. Second, in the case of *S. haematobium* it is the disposal of urine, far more difficult to control than the disposal of feces, that is important. Because multiplication of these helminths takes place in the intermediate hosts (except in the case of the fish tapeworm, *Diphyllobothrium*), one egg can give rise to many infective larvae. A thousandfold multiplication is not uncommon, and effective transmission can continue at low contamination levels. The requirements for adequate excreta disposal, in terms of the percentage of all feces reaching the toilet, may therefore be demanding.

*Category VI*

The excreta-related insect vectors of disease form three main groups. In the first of these, the cosmopolitan *Culex pipiens* complex of mosquito species preferentially breeds in highly contaminated water and is medically important as a vector of the worm (*Wuchereria bancrofti*) that causes Bancroftian filariasis. The other two groups, flies and cockroaches, proliferate where feces are exposed. Both have been shown to carry numerous excreted pathogens on their feet and in their intestinal tract, but their role in actually spreading disease from person to person is disputed (though their nuisance value is certain). Flies have been implicated, however, in the transmission of eye infections and in infecting and spreading skin lesions. The control measures implied for insects are those sanitary improvements of differing sophistication which prevent their access to excreta. In general, the simpler the facility, the more care is needed to maintain it insect-free. Cockroaches, flies, and *Culex pipiens* mosquitoes often have breeding places in addition to those associated with excreta disposal and will in many cases elude control by disposal improvements alone.

*Summary*

The correlation of the environmental features of the categories with the length and spread of transmission routes has been indicated in figure 2-5, and the discussion has emphasized the importance of complementary controls for most diseases. If excreta disposal alone is improved, however, likely control for each category is as follows:

| *Category* | *Control* |
|---|---|
| I | Negligible |
| II | Slight to moderate |
| III | Moderate to great |
| IV | Moderate to great |
| V | Moderate |
| VI | Slight to moderate |

The outstanding difference is between categories I and II, which depend strongly on personal and domestic cleanliness, and the other categories, which do not. The central changes necessary to control infections in

4. See the discussion of these processes in chapters 5 and 6.

categories III and IV are relatively simple—namely, the provision of toilets *which people of all ages*[5] *will use and keep clean*, and the treatment of fecal products prior to recycling on the land. The reason that reports on the effects of latrine programs often do not show a marked decrease in the prevalence of the infections in categories III and IV[6] is that, although latrines have been built, they have typically neither been kept clean nor been used by children or by adults when working in the fields.

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5. Of course babies and very young children are unable to use a toilet. Health education programs must include advice to mothers on how to dispose of their children's excreta in a suitably hygienic way.

6. See the next chapter and table 2-1.


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# 3

# The Risks of Excreta to Public Health

THE DISCUSSION has dwelled at length on the survival of pathogenic organisms in excreta, on which there is a good deal of information. This is the main health-related concern of the engineer when he is designing sanitary facilities. The planner and economist have a greater interest in epidemiological risk: if, in a given situation specific changes in excreta disposal are made, how much less disease will there be? This question can be rephrased in two ways, the first of which can be answered readily and the other only with the greatest difficulty. The easier question is: what are the disease problems associated with excreta, and thus, by implication, with inadequate excreta disposal facilities or inadequate personal or domestic cleanliness? The difficult question concerns the health benefits of improved sanitation: how much disease will be eradicated if a given sanitary improvement is undertaken? These questions are considered in general terms in this chapter.

## Illustrative Sketches

The effects of the diseases accompanying unsafe excreta disposal are dramatized in the sketches of two imaginary settings that follow. Formal case studies of sanitation and health are then outlined before a discussion of the benefits of sanitation improvements.

### *A Southeast Asian family*

In areas of Southeast Asia with high rainfall, a perennially hot climate, and a main cereal crop of irrigated rice, the diverse health hazards from excreta are illustrated by the following case history, a composite of several real sites and people. A family lives in a palm-roofed, wooden house surrounded by rice paddies and small irrigation channels, one of which, flowing near the house, acts as the domestic water supply. There are four children in the family: the mother has had six babies, one of whom died at the age of 15 months after a sudden attack of diarrhea; a school-age child died in a cholera epidemic that swept the region 4 years ago.

It is particularly difficult to control excreta in this damp environment—most feces are deposited close to the house, and the younger children urinate in the nearby canals. Several years ago a government campaign to provide pit latrines was mounted, and one was dug near the family's house. They used it for a while, until the pit flooded over in the monsoon season and a large quantity of fecal material was spread around the house. It was during the flooding that the cholera epidemic occurred; its sad consequences for the family, together with the unpleasant mess from the latrine, discouraged further use of the facility. The next government recommended that a concrete aquaprivy be built above the ground to avoid the floods, but the family could not afford this and returned to defecating close to the house during the day. Nocturnal excreta were collected in a bucket and deposited in a nearby fishpond.

How does this situation affect the family's health? All the children get diarrhea several times a year, the parents less regularly. The worst occasion was when two of the girls got it at the same time. The younger one, 15 months old, seemed to wither overnight, and she died the next day. Death was from rotavirus infection. (Why it is more often lethal in the tropics than in temperate countries is unclear: perhaps the poor sanitary facilities in this case gave the child an overwhelming dose of the virus; perhaps malnutrition, ubiquitous during the weaning period in communities such as this one, complicated the attack?) Most of the diarrheas are watery, sudden attacks, but last year the grandmother who shares the house with the family, was one of several people in the village who came down with a more painful diarrhea with bloody stools, from which she nearly died. Medicine from the dispensary four miles away helped initially, but she remained ill for

weeks. This attack was from bacillary dysentery (shigellosis), though to distinguish it from amebiasis would have required laboratory diagnosis.

All these illnesses were dramatic, but the family has several health problems of which they are barely aware. The eldest son has not grown properly: although he is 23, he looks as if he were in his early teens; his belly is always grossly swollen, and the dispensary attendant can feel his hard liver and spleen under the taut skin of his abdomen. His condition is caused by schistosomiasis, which is spread from one person to another by a tiny snail living in the damp grass beside the canals and in the water itself. Several members of the family are infected, but only this boy shows signs of the disease.

In this region of paddies and canals, fish—sometimes cooked, sometimes pickled raw in vinegar—is an acceptable and available food. A portion of the fish consumed is from ponds fertilized with human feces, and this practice has caused some of the family to be infected by the liver fluke *Clonorchis sinensis*. Another helminth that the family harbors in large numbers is *Fasciolopsis buski*, an intestinal fluke acquired from eating raw aquatic vegetables. Neither of these parasites causes catastrophic illness, but the diversion of nutrients to the parasites and their other insidious effects make life less satisfactory than it otherwise would be. The family also suffers from other intestinal worms occurring in even greater numbers and causing more illness (these are discussed in relation to another family, below).

One more infection attends the family's lack of safe excreta disposal. Within the pits of the latrines that have been flooded and abandoned, the liquid waste is colonized by mosquito larvae of the *Culex pipiens* group. When the adult mosquitos bite the members of the household, they transmit into the bloodstream the larvae of a parasitic worm (*Wuchereria bancrofti*) that lives in the tissues under the skin of the legs and elsewhere, particularly in the lymph nodes, where it blocks the lymphatic flow. Tissues near the blockage consequently become swollen from the accumulation of lymph, and some affected people develop massive elephantiasis. The father of the family is troubled by this in his right leg, which is so swollen that he cannot work in the fields as productively as he could before.

### *A North African family*

The next region visited is quite different in general appearance, but behind this exterior are certain similarities in the disease pattern. The village entered is a cluster of mud brick houses located in the subtropics. It is quite cold in the winter though summer temperatures are at least as high as in the Asian village just visited. The houses cluster on a mound rising from surrounding irrigated areas. The irrigation is from water brought from afar by great rivers, not from heavy rainfall, and the ground is baked hard where it has not recently been irrigated. Within the village the streets are narrow and unpaved, and large quantities of debris lie around.

The family selected consists of the parents, three children, and some elderly relatives. This family has also suffered the death of children from diarrheal disease. (Indeed, it would be difficult to find a tropical or subtropical area in which this is not a problem.)

As in Asia, schistosomiasis and elephantiasis are present. These are of somewhat different varieties, but create disability in similar ways. Although intestinal schistosomiasis occurs here, two of the younger children have a urinary form of the disease and pass blood in their urine every day. This looks more serious than it is (in fact, the blood loss is small); however, the children do suffer the pain and inconvenience of having to get up frequently to urinate at night. Not too long ago their uncle had to go to the hospital in the nearby city, where he was told that he had inoperable cancer of the bladder. He died a very painful death, and the surgeon told the family his death may have been a late consequence of the same infection causing blood in the urine of the children, but that only a few unfortunate people developed cancer from the infection.

The helminths associated with fish and aquatic plants that plagued the previous family are absent from this environment, but microscopic examination of the feces of the family show hookworm (*Ancylostoma*), roundworm (*Ascaris*) and whipworm (*Trichuris*) eggs in large numbers. The hookworm eggs are especially numerous—the infection has been picked up by walking barefoot on land that has been used for defecation and that has been kept moist enough by the nearby drains and canals for the worm larvae to develop in the soil. The mother has a particularly heavy infection. The worms inhabit the small intestine where they attach themselves to the intestinal wall; they are messy feeders, and a large amount of the blood they suck for their growth and production of their eggs passes straight through their bodies and is lost into the intestinal lumen. As a result the blood losses from this infection are high—indeed, the mother's loss is twice as heavy as that from menstruation and, because her diet is not overly rich in iron, she has become anemic and unable to work as hard as a fit person. The same is true of one of the children: his abdomen is swollen; he cannot run fast enough to keep up with the other children, and his condition gives considerable cause for

anxiety. Some other infection in addition to the hookworm might well claim his life.

All the family have roundworms. These parasites are quite large (over 100 millimeters long), and every now and then one of the younger children passes one in the stool. This elicits little more than comment, since there is no obvious illness except pain in the abdomen, a complaint difficult to ascribe to a specific cause. What is certain is that the worms are absorbing a good percentage of the nutrients the children need, and there is also the risk that the worms will get stuck in the narrowest part of the intestine and block it, thus necessitating a faraway surgeon's attention. The family are well aware of the problem, and have often visited the dispensary to get medicine. But in the absence of instruction and better methods of disposing of their excreta, the infection comes back every few months. The adults seem to have become somewhat immune to reinfection, and the children carry the brunt of the recurrence.

Most of those, especially the children, who have roundworms also have whipworm infections. These little worms, found mostly on the wall of the colon and rectal passage, have an uncertain effect on most of the family, generally adding to the burden of other parasites. Some while ago a neighbor's child picked up a very heavy infection with the whipworm. The rectal passage gradually prolapsed (that is, got partly pushed out of the anal orifice), which was both painful and unsightly, while an intractable diarrhea that had begun some months earlier persisted and made the child very anemic. A concurrent amebic infection led to some confusion over what was causing which symptoms, but the prolapse was certainly not a regular feature of amebiasis and the diarrhea could have been due to either cause.

What arrangements are made for excreta disposal here? Bore-hole latrines were made for each family's use but they filled up rather quickly and were then so unpleasant that no one wanted to use them. In any case, the latrines were in or near the houses and, because the families spend much of the day in the fields working on their rice and other crops most felt that it would be an unreasonable waste of their time to come all the way back to the house to defecate. It is also more convenient to relieve themselves in the field because their religion insists that they wash the anus after defecation, and there is no water readily available for this purpose within the compound. Because of the varying sites for defecation, roundworm and whipworm eggs are spread widely throughout the environment. The eggs are extremely resistant, even in the harsh climate of this part of the world, and find their way onto vegetables that are eaten raw. Eggs also occur in the mud and sand of the compound, where they readily contaminate the hands of crawling babies.

Another intestinal worm somewhat important in this milieu is the beef tapeworm (*Taenia saginata*), which is acquired from an infected cow by eating its beef undercooked (this readily occurs when a large piece of meat is roasted). The adult tapeworm matures in the intestines, adding segments to its length and competing for the family members' limited nutrients; its eggs, often contained in its swollen segments, are shed in large numbers when a tapeworm segment wriggles out of the anus. These worm segments are frequently ingested by browsing cattle; the worm undergoes further development within the muscles of the cow; the beef is inadequately cooked and eaten, and the cycle of infection is resumed. The family's religion prohibits the eating of pork and so they are spared its tapeworm (*Taenia solium*), whose larvae can develop in human muscles—an added and sometimes fatal hazard.

Except in the case of hookworm infection with its dramatic blood loss, all these helminth infections are so long lasting and ennervating that it is difficult to assess their specific damage; they are all infections that are often underrated because of their widespread occurrence and insidious, drawn-out course. The family also suffers from several acute infections, not only diarrheas but also typhoid and hepatitis. The incidence of typhoid in the village is particularly high because of inadequate excreta disposal. In addition, the presence of schistosomiasis in the inhabitants modifies typhoid and lengthens its course, and up to one in every twenty-five people may become a typhoid carrier in some of these villages, a rate which is an order of magnitude higher than seen elsewhere. Consequently, typhoid is extremely common, no less severe than elsewhere, and an appreciable cause of mortality. Hepatitis also occurs frequently: in the younger children it rarely produces serious symptoms, but in adults the patient may be bedridden for weeks or months, and sudden death is not unknown.

One feature that clearly emerges from the account of this family in North Africa is the extent to which it shares in the excreta-related health problems of the family in Southeast Asia. Indeed, as in few other disease patterns, there is a sameness to most of the serious, frequently transmitted, excreted infections that cannot be avoided: certain infections are peculiar to particular localities, but the pattern of diarrheal disease, enteric fever, numerous viral infections, and intestinal worms is repeated worldwide. Of the major excreted infections, only cholera and schistosomiasis have variable and patchy distributions.

Table 3-1. *Maximum prevalence of excreted pathogens (from table 2-2) by age in indigenous populations of endemic areas*

| Pathogen | Age group of highest prevalence of infection (years) | | | |
|---|---|---|---|---|
| | *Babies (0–2)* | *Children (3–12)* | *Teenagers (13–19)* | *Adults (20+)* |
| CATEGORY I | | | | |
| *Balantidium coli* | | | * | * |
| *Entamoeba histolytica* | | * | * | * |
| *Enterobius vermicularis* | * | * | | |
| Enteroviruses[a] | * | * | | |
| *Giardia lamblia* | | * | | |
| Hepatitis A virus | * | * | * | |
| *Hymenolepis nana* | * | * | | |
| Rotavirus | * | | | |
| CATEGORY II | | | | |
| *Campylobacter fetus* ssp. *jejuni* | * | * | | |
| Pathogenic *Escherichia coli*[b] | * | * | | |
| *Salmonella* | | | | |
| *S. typhi* | | * | * | * |
| Other salmonellae | * | * | * | * |
| *Shigella* spp. | * | * | | |
| *Vibrio cholerae* | | * | | |
| *Yersinia enterocolitica* | | * | * | * |
| CATEGORY III | | | | |
| *Ascaris lumbricoides* | | * | * | |
| Hookworms[c] | | * | * | * |
| *Strongyloides stercoralis* | | | * | * |
| *Trichuris trichiura* | | * | * | |
| CATEGORY IV | | | | |
| *Taenia saginata* and *T. solium* | | | * | * |
| CATEGORY V | | | | |
| *Clonorchis sinensis* | | | | * |
| *Diphyllobothrium latum* | | | | * |
| *Fasciola hepatica* | | * | * | * |
| *Fasciolopsis buski* | | * | * | |
| *Gastrodiscoides hominis* | | * | * | |
| *Heterophyes heterophyes* | | | | * |
| *Metagonimus yokagawai* | | | | * |
| *Paragonimus westermani* | | | | * |
| *Schistosoma* spp. | | * | * | * |

a. Includes polio-, echo-, and coxsackieviruses.
b. Includes enterotoxigenic, enteroinvasive, and enteropathogenic *E. coli*.
c. *Ancylostoma duodenale* and *Necator americanus*.

## Children and Excreta Disposal

Many of the excreted infections examined in this book have a markedly nonuniform distribution among different age groups. Although all the infections are found in people of all ages, many are concentrated in particular age groups. The age groups most afflicted by the main excreted infections in areas where these infections are endemic are shown in table 3-1. The data of the table clearly show that many of these illnesses are

primarily childhood infections, or that the infections afflict children as well as adults. This fact has the greatest relevance for disease control through improvements in excreta disposal.

In all societies children below the age of about 3 will defecate whenever and wherever they feel the need. A proportion of these children will be excreting substantial quantities of pathogens. In some societies these children's feces are regarded as relatively inoffensive, and the children are allowed to defecate anywhere in or near the house. In this case it is highly likely that these feces will play a significant role in transmitting infection to other children and adults. This applies not only to those infections without a latency period but also to infections such as ascariasis, in which the defecation habits of children may determine the degree of soil pollution in the yard and around the house and thus the prevalence and intensity of infection in the household. In other societies strenuous efforts are made to control and manage the stools of young children, either by making them wear diapers (nappies) or by cleaning up their stools whenever they are found. Either of these reactions should have an important controlling influence on the intrafamilial transmission of excreted infections.

Between these two extremes is a range of intermediate behavioral patterns by which adults react to the stools of young children. In most poor communities, the pattern is closer to the first reaction than to the second. The relevant response of government and other responsible agencies where these attitudes prevail should be health education programs to encourage in mothers the belief that the stools of young children are dangerous and should be hygienically disposed of. Although the problem is primarily in attitudes and behavior, the provision of some form of toilet for the disposal of a child's stool and, perhaps more important, a convenient water supply will greatly assist child hygiene.

Children over 3 years in age, in contrast, are capable of using a toilet if one of suitable design is available. Children of 3 to 12 years frequently do not use available toilets because they find their use inconvenient and it is not encouraged by adults; they are afraid of falling down the toilet's hole or of being attacked by the pigs that may live next to the latrine; they cannot use a toilet not designed to their scale; or they are prevented from doing so by adults who want to avoid cleaning up the toilet area after them.

As with very young children, it is of vital importance that the stools of children over age 3 be hygienically disposed of because some will be rich in pathogens. The solution lies in providing both toilets that children are happy to use and health education for mothers, so that they will compel their children to use them. Health education for school-aged children could also be effective here, and it is essential that all schools have well-maintained latrines as positive examples for the children.[1]

## Distribution of Sanitation Benefits

The transmission cycles typically followed by the infections in categories I through V (table 2-2 and figure 2-5) have been compared and discussed and the shorter or tighter cycles that categories I and II may follow over categories III to V have been indicated. The implication is that categories III to V are associated with a wider spread of their infections, a factor important in the selection of appropriate excreta disposal technology and, in particular, in assessing the willingness of an individual family to adopt an innovation. If, on the one hand, a household head believes, or can be persuaded to believe, that the adoption of a new technology will bring appreciable health benefits to his family, regardless of what is taking place in the neighborhood, then he will be more willing to innovate. If, on the other hand, it is clear that his action alone will have a negligible effect on his family's health, he is more likely to sit back and await clear evidence that a viable and effective improvement program is being carried out throughout his neighborhood.

In cases in which most pathogen transmission is intrafamilial—as in category I and, to a lesser extent, category II—it can be expected that improvement in excreta disposal *and cleanliness* in an individual family may lead to health benefits for that family. In fact, as we have already argued, cleanliness is probably more important than excreta disposal facilities *per se* in the reduction of category I (and to a lesser extent category II) infections, and therefore it is changes in hygienic behavior that may bring the greatest benefit to a single family in isolation from widespread changes in the community's sanitation.

There is one infection from categories III to V—ascariasis—which, although potentially having a long transmission cycle, is frequently transmitted within the family and diminishes with improvements in excreta disposal facilities without accompanying changes in personal cleanliness. Work in China and the US in the 1920's and 1930's showed that poor families, who used their latrines and prevented their children from defecating in the yard, had significantly lower

1. Indeed, the whole subject of health education, so difficult to discuss incisively, is crucial to the full realisation of the potential health benefits of improved excreta disposal facilities—see Chapter 8.

intensities of *Ascaris* infection than their neighbors (Otto, Cort and Keller 1931; Winfield 1937). Similar reductions on an individual family basis can be expected from the adoption of improvements in excreta disposal today. There are other specific circumstances in which a given infection may readily be reduced by the independent action of a single family. An example is hookworm in rural India, where in many villages much of the infection occurs when barefoot people visit the communal defecation grounds on the edge of the community. A family which installs a pit latrine and no longer visits the defecation ground may substantially reduce its exposure to hookworm infection. These cases demonstrate that, in planning and implementing an excreta disposal program, officials may find it useful to identify infections for which individual household action may be particularly effective. These infections might then be monitored and the family results used as part of a community propaganda exercise (for example: "the Sanchez family has adopted the new latrine and improved their domestic hygiene and they now have less roundworms than their neighbors").

## Health Benefits of Sanitation

Although major health problems are clearly associated with inadequate excreta disposal facilities, to relate the two causally—in particular, to say what the health benefits will be from a given proposed improvement of facilities—is difficult. The difficulties and the studies attempting to overcome them are reviewed in this section. Critical comments must not obscure the fact that without improved excreta disposal many of the diseases discussed will never be overcome, yet other complementary measures—and in some cases major social, economic, and political changes—will generally be required for success.

### *Methodological issues*

Studies of the health benefits of sanitation in the field have either compared disease levels in communities with varying sanitary facilities or monitored disease patterns before and after the improvement of sanitary facilities within a community. In both cases the difficulties in attributing benefits to the improved sanitation have arisen because other variables are often associated with the sanitation facilities. People who have better sanitation than their neighbors often also have higher incomes, better water supplies, and different habits of cleanliness. Similarly, if a single community is followed over time, improvements in the sanitary facilities are likely to be only one among several changes to benefit the community's health. To allocate all the health benefits to improved sanitation alone would therefore be unjustified. Conversely, a study that demonstrates no health improvement after appropriate changes in sanitary facilities cannot validly imply that such changes are useless. The facilities may have been unused for lack of health education or may have been improperly sited—it is often a mistake to generalize from a particular local result.

The economist ideally wishes to use data on health benefits to decide priorities in resource allocation, and the total health benefits are needed for such a decision. However, health as such is not measureable (except, possibly, in the form of statistics on the growth of infants), and it is diseases that are studied instead. Because sanitation affects a range of diseases not all measurable in a single study, a few indicator or index diseases are usually chosen to assess benefits. More often still, particular disease agents—such as *Shigella* bacteria or worm eggs—are assessed in the feces. The resulting measures of how infection rates change as sanitation is applied are several removes from health benefits *per se*, and the intermediate causal relationships are by no means linear. The relation between an infection and the development of disease depends on variables such as: the intensity of infection, nutritional status, other infections, age of the host, and health care available locally.

### *The literature*

Some of the relevant literature on the assessment of health benefits is listed in table 2-1. Almost none of the studies described there reaches the standards of epidemiological demonstration that make a study conclusive; melancholy criticism of the limitations of each paper is therefore avoided. Rather, the conclusions reported in the literature should be taken as an indication of trends.

An important component of any evaluation, but one that is much neglected, is time. The attainment of comparability between an area that has experienced sanitary interventions and one that has not requires that surveys be done soon after installation of the sanitary facilities. In the common case, observations are recorded only for up to a year and are begun months after construction. Such information has poor predictive value for the long term. If a special campaign has been mounted in relation to new facilities, the results may be transiently impressive but may fall off over time. Conversely, the community may take some years to adjust to and use the innovations, so that a

short-term study fails to demonstrate the real benefits changes bring. Even if these problems are avoided through long-term study or the observation of variations between communities with long-established differences in excreta disposal patterns, the difficulty of confounding variables arises: it is most unlikely that communities will stay comparable in all differences other than excreta disposal and its consequences over many years.

Considering these complexities, it is not surprising that studies on the benefits of excreta disposal assessed by health changes in the field are almost all of an insufficient standard to be convincing. Few indeed can be described as scientifically impeccable and productive of results inspiring confidence. This discussion of methodology might be considered niggling and academic if most of the published studies gave concordant results—but this is not the case, and some studies are frankly contradictory. Again, a detailed critique of each study listed in table 2-1 is not given because these defects in sampling, comparability of samples, and confounding variables recur with such consistency, whereas the actual use of facilities provided is scarcely ever assessed. A further methodological difficulty is that, in studies using recurrent medical treatment, observations are made during periods too short in duration to show long-term outcomes and to detect the large rise in noncompliance with therapy that tends to occur in time.

If all the studies in table 2-1 were summarized, however, they would collectively suggest that it is reasonable to hope for a halving of the incidence of category III, IV and V infections through improved excreta disposal facilities and concomitant supporting programs for facility maintenance and health education. If such programs are combined with safe water supply and appropriate behavioral changes, the risk from some other serious excreta-related diseases can become small, and such illnesses as typhoid and cholera (category II) can cease to be endemic. The impact of improved excreta disposal on category I infections is likely to be small in the absence of major improvements in domestic conditions, which may imply substantial socioeconomic change in the community at large.

## Limitations in Assessing Health Benefits

The planner seeks a clear, preferably monetary, statement of the health benefits of alternative sanitation improvements. The data are not adequate to provide one. It is quite feasible to list the present costs of treating sanitation-related diseases, but these estimates are small in relation to the estimates of the work and life lost to these diseases. The latter estimates themselves are subject to great uncertainty, and any figures put on such losses may be largely spurious.

Two examples may be given. Wagner and Lanoix (1969) attempted to estimate the costs of diarrheal disease and found that the largest component was from premature death in children under the age of 2 years. There are several means of placing an economic value to death at this age that give widely differing answers. More recently Latham, Latham and Basta (1977) estimated the cost of *Ascaris* infection to Kenya. The largest single component was the estimated reduction in food absorption and utilization by those infected, given as US$4.4 millions yearly, as compared with a total of US$0.7 million for all other costs such as present treatment, health care, and transport to health care facilities. Yet it is possible to pose reasons for the US$4.4 millions varying by $\pm 50$ percent.

It is also possible, however, to make informed assessments of the comparative benefits of different excreta disposal systems, and this is attempted below. No cost figures on different excreta disposal systems are given here—these may be found in the various other documents issuing from the World Bank's investigations of appropriate sanitation technologies.[2] It will be clear from our discussion of human behavior here and in chapter 8 that the greatest determinants of the efficacy of alternative facilities are, first, whether they are used by everyone all the time, and second, whether they are adequately maintained. Use will be dependent on the locality concerned: for instance, in urban situations, where alternative defecation sites are scarce, it will be easier to ensure widespread use of new facilities. There are also both private and public aspects to maintenance of all but basic on-site systems, and systems vary in their public maintenance needs (some withstand public neglect better than others).

### *Best inferences in an optimal case*

In the evaluation of the health benefits of excreta disposal, an optimal situation would be one in which everyone uses the facilities all the time and the town council responsible for their maintenance is meticulous in its duties. A corresponding worst case would be the total lack of sanitation facilities. In both cases, it is the disposal technologies rather than management systems that are the objects of comparison. The baseline

2. See, for example, Kalbermatten, Julius and Gunnerson (1982) and Feachem, Mara and Iwugo (1980).

situation will vary greatly in the absence of any sanitary provisions. Where population densities are high, as in many parts of rural Asia and in all the world's major cities, the base level of disease caused by excreted pathogens will be quite high. On a crude scale of ill health, this situation would be rated at 0. Where conditions include flush toilets, sewers and an efficient treatment plant—the best case—the resulting health benefits will rate a 10 as long as water supplies are adequate for optimal use of the sanitation system.

Although not adapted to the water use levels needed for the personal cleanliness required to minimize the infections of categories I and II (see table 2-2), pit latrines would, from the viewpoint of health rather than convenience, approximate the same rating as a waterborne sewerage system. Because a pit latrine has no effluent or product, it is in this regard safer than a sewerage system producing large volumes of a polluted effluent that is in general, even in the best treatment plants, not made completely pathogen free. A rating of 9 is given to pit latrines (but this rating does not apply wherever fecal material might soak through latrine walls to gain access ultimately to drinking water or wherever flooding or a high water table regularly recur).

Where composting, double-vault latrines (a rating of 8) are used and are dug out frequently, a residual hazard from long-lived helminth eggs persists and benefits are less. Reuse of the compost will further spread the eggs in the community. The "multrum" composting toilet is, again, safe if operated ideally, but in general its risks tend to be greater (hence a rating of 7) because the latrine's continuous process involves hazards from insufficiently composted pathogens.

An aquaprivy with a retention time longer than a month may yield an effluent with a low pathogen content, but this requires the regular addition of water to the tank at a rate that will not seriously reduce the retention time. Provided that an efficient sludge removal and treatment system is available, the resulting health benefits from the aquaprivy might approximate 9 on the scale. A septic tank with a retention time of only 1–3 days produces an effluent rich in pathogens and therefore is associated with greater risk (a rating of 8 is assigned). With a bucket-latrine system, major reductions in disease are unlikely, even in an ideal world, and a rating of 5 is considered appropriate. A well-managed vault and vacuum-truck cartage system would be a great improvement, but some risk of spillage and contact with fresh feces would still exist (hence a rating of 8).

The preceding ranks the health benefits of mainly on-site systems. If excreta are transported by cartage or water to a treatment plant, oxidation ponds for sewage, and batch thermophilic composting for night soils and sludges, will give a safe product. Alternative processes in treatment plants are inferior.

*Best inferences in actuality*

In the real world, of course, systems are not maintained impeccably, nor are facilities invariably used. Moreover, some systems clearly require less effort to maintain and use than others. Cartage in some Japanese towns using vacuum trucks is fully comparable to waterborne sewerage (Kalbermatten, Julius and Gunnerson 1982). In some cities in other countries, however, the great majority of trucks are typically out of operation. Health benefits are closely tied to operation and use, and some societies are better than others at operating particular systems. If change is contemplated, much greater effort than hitherto assumed may need to be allocated to the operation and use, rather than the installation, of new facilities.

Operation and maintenance require both user effort and municipal endeavor, and the necessary blend between these differs according to the chosen technology. A ranking of the various disposal technologies by ease of maintenance for the user and the municipality, water requirements, and the ideal (as discussed in the section above) and actual health benefits is given in table 3-2. It should be noted that the ideal benefits vary little among processes when the facilities are well maintained and used; only bucket latrines are intrinsically and substantially inferior. The proposed ranking of actual benefits reflects variables leading to neglect of the particular facilities, but this is a very provisional evaluation, and many other factors must be taken into account in selecting technology for a given site. The informed speculation in table 3-2 is intended to stimulate thought about the health-related aspects of technology choice, and to draw attention to the disparate advantages of the pit latrine and the bucket system.

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Table 3-2. *Ranking of excreta disposal technologies by ease of operation and maintenance, water needs, and health benefits*
(scale of 10)

| Technology | *Lack of effort required*[a] | | *Water needs* | *Health benefits*[b] | |
|---|---|---|---|---|---|
| | *By user* | *By municipality* | | *Ideal* | *Actual* |
| Flush toilet/ sewers/oxidation ponds | 10 | 4 | H | 10 | 9 |
| Vault/vacuum truck | 8 | 0 | L | 8 | 6 |
| Pit latrine | 8 | 5 | L | 9 | 6 |
| Septic tank | 6 | 5 | H | 8 | 7 |
| Aquaprivy | 5 | 5 | M | 9 | 6 |
| Bucket latrine | 3 | 1 | L | 5 | 1 |
| Batch composter (double vault) | 1 | 10 | L | 8 | 5 |
| Continuous composter (multrum) | 0 | 10 | L | 7 | 3 |

L Low; M medium; H high.
a. 0 = maximum effort; 10 = minimum effort.
b. 0 = no benefits; 10 = maximum benefits.

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# 4

# Detection, Survival, and Removal of Pathogens in the Environment

THE COMMENSAL and pathogenic organisms found in human feces—and the environmental characteristics of the latter's transmission and control—have been examined in earlier chapters. The focus of this chapter is on the suitability of various excreted bacteria as diagnostic organisms to indicate environmental fecal pollution and on the relation of these bacteria to other bacterial and nonbacterial pathogens. In addition to bacterial indicators, generic "pathogen indicators" are proposed for assessing the safety of the products of excreta treatments. The survival times of indicators and pathogens in different environments (the reader is referred to Part Two in this regard) and several issues affecting the choice of excreta treatment technologies are analyzed in the remainder of the chapter.

## Fecal Indicator Bacteria

Fecal indicator bacteria[1] are selected from among those commensal species that exclusively live in the intestinal tract of man and other warm-blooded animals without causing disease. Because they are always and naturally present in feces and are excreted in large numbers (up to $10^9$ or $10^{10}$ cells per gram of feces), their presence in water indicates beyond doubt that the water has been contaminated with fecal material and possibly with excreted pathogens. If a water is shown to contain fecal indicator bacteria, it is considered unsafe for human consumption. This is the rationale for the bacteriological testing of public water supplies that was developed in Europe and North America at the turn of the century when the major concern of water supply engineers was to reduce the incidence of epidemics of strictly waterborne disease. It is still an epidemiologically valid testing technique for disinfected water supplies throughout the world, but it has certain limitations when applied indiscriminately in the examination of wastes and wastewaters, particularly in hot climates. (These limitations are discussed in the section "Relation of Fecal Indicator Bacteria to Excreted Pathogens," below.)

The ideal fecal indicator bacterium should be:

- A normal member of the intestinal flora of healthy people
- Exclusively intestinal in habitat, and hence exclusively fecal in origin when found in the environment
- Absent from nonhuman animals (a requirement not met by any of the indicator bacteria currently used)
- Present whenever fecal pathogens are present, and present only when fecal pathogens might reasonably be expected to be present
- Present in higher numbers than fecal pathogens
- Unable to grow outside the intestine, with a die-off rate slightly less than that of fecal pathogens
- Resistant to natural antagonistic factors and to water and waste treatment processes to a degree equal to or greater than that of fecal pathogens
- Easy to detect and count
- Nonpathogenic.

No one bacterial species or group completely fulfills all these requirements, but a few come close to doing so. Three main groups of bacteria are used as fecal indicators in conventional water bacteriology: the fecal coliforms, the fecal streptococci and the anaerobic bacterium *Clostridium perfringens*. Recently, some other members of the anaerobic intestinal flora,

1. Fecal indicator bacteria are discussed in greater detail in chapter 13; see also specific chapters in Part Two for notes on the taxonomic nomenclature of particular pathogens.

notably *Bifidobacterium* spp., have been proposed as additional indicator bacteria. *Pseudomonas aeruginosa* has also been proposed, but its status as an intestinal organism is in doubt. An analysis of these bacteria and their uses as indicators follows.

### *Coliform bacteria*

There are two principal groups of coliform bacteria; the fecal coliforms (comprising mainly the bacterium *Escherichia coli*) and the total coliform group, that includes the fecal coliforms and comprises mainly species of the genera *Citrobacter*, *Enterobacter*, *Escherichia*, and *Klebsiella*. The former are exclusively fecal in origin, whereas the latter, although commonly found in feces also occur naturally in unpolluted soils and waters. Of the total coliform organisms found in fresh feces of warm-blooded animals, generally >90 percent are *E. coli*, the remainder being species of *Citrobacter*, *Enterobacter*, and *Klebsiella* (Dufour 1977).

Only the fecal coliforms (and especially *E. coli*) are definitive indicators of fecal pollution. In water bacteriology the total coliforms are regarded as "presumptive" indicators of pollution and should be absent from disinfected water supplies. In wastewater bacteriology, however, the total coliforms are of considerably less importance because many are nonfecal in origin and, especially in hot climates, they can multiply in the environment under suitable conditions, so that their presence or numbers may not necessarily relate to either the occurrence or degree of fecal pollution. In general, and despite the one report from India to the contrary (Raghavachari and Iyer 1939), only fecal coliforms (or better still, *E. coli*) should be used as indicators or tracers of fecal bacterial pathogens in wastes, wastewaters, and treatment and reuse processes.

Fecal and total coliforms are indistinguishable under the microscope: they are all Gram-negative rods measuring some 2–5 by 0.4 micrometers. In practice they are differentiated by the ability of fecal coliforms (mainly *E. coli* and thermotolerant *K. pneumoniae*) to ferment lactose with the production of acid and gas within 24 to 48 hours at a temeprature of 44°C. In addition, the most common fecal coliform, *E. coli*, can produce indole from the amino acid tryptophan at this temperature. In hot climates, however, some nonfecal coliforms can grow at 44°C and some can also produce indole at this temperature, thus mimicking the fecal coliforms (*E. coli* in particular). There are no satisfactory routine methods for differentiating between these organisms, and their simultaneous occurrence has prompted a search in recent years for alternative, more satisfactory indicator organisms for use in hot climates. A further disadvantage of fecal coliforms is that most standard enumeration procedures require an accurately controlled incubation period at 44° or 44.5°C, which is difficult to achieve in any small, or nonspecialized, laboratory.

### *Fecal streptococci*

The fecal streptococci (or Group D streptococci) are a group of bacteria that are morphologically similar (Gram-positive cocci, measuring approximately 1 micrometer in diameter and occurring in short chains) and are mostly found in the intestines of man and other warm-blooded animals. The group includes species mainly associated with animals (*Streptococcus bovis* and *S. equinus*), other species with a wider distribution (for example, *S. faecalis* and *S. faecium*, which occur both in man and other animals), as well as two biotypes (*S. faecalis* var. *liquefaciens* and an atypical *S. faecalis* that hydrolyzes starch) that appear to be ubiquitous, occurring in both polluted and unpolluted environments. These last two strains, essentially nonfecal (although included in the group of fecal streptococci), are indistinguishable from the true fecal streptococci under routine detection or counting procedures. Because *S. faecalis* var. *liquefaciens* has been reported as the predominant biotype present at low densities (below about 100 fecal streptococci per 100 milliliters; Geldreich 1970), the usefulness of the fecal streptococci group as an indicator is open to question, especially in clean water bacteriology. Yet fecal streptococci may still have a place in wastewater bacteriology, although not as indicators of the bacteriological quality of wastewater-irrigated crops, on which the two nonfecal biotypes may both be present as natural flora unrelated to the degree of fecal pollution. There is no information, however, on the distribution of these two biotypes in tropical environments.

Aside from the possible problem of nonfecal strains of Group D streptococci, fecal streptococci have major advantages as fecal indicators. They are enumerated by a single-step membrane-filter procedure at 37°C, a temperature readily attained in small field laboratories. They are less prone to regrowth, and generally survive somewhat longer, than fecal coliforms and may thus be better indicators of excreted bacterial pathogens (that have little regrowth tendency) and excreted virus (that survive for longer than fecal coliforms in cool waters). These points are discussed in greater detail in chapter 13.

*Fecal coliform to fecal streptococci ratio*

It has been found that human feces in the USA contain at least 4 times as many fecal coliforms as fecal streptococci, but that animal feces contain at least 1.4 times as many fecal streptococci as fecal coliforms (Geldreich 1966). It was therefore suggested that American surface waters that have fecal coliform-streptococci ratios of $>4$ are likely to have received predominantly human pollution, whereas those with ratios of $<0.7$ mainly have been contaminated by the feces of wild and domestic animals (Geldreich 1966).

This method, however, is of no value in practice. The fecal coliform-streptococci ratios in fresh feces vary widely in different animal species and geographical locations. It is not true that humans the world over excrete a ratio of $>4$, and animals $<0.7$ (Wheater, Mara and Oragui 1979). Once the feces have been excreted, the ratios will change because of the differential death rates of the various bacteria. The enterococci (*S. faecalis*, *S. faecium*, and *S. durans*) typically survive longer than fecal coliforms which in turn survive longer than *S. bovis* and *S. equinus* (McFeters and others 1974). It was therefore suggested that for human pollution, in which enterococci are the dominant fecal streptococcal species, fecal coliform-streptococci ratios in water samples returned to the laboratory will fall; whereas for animal pollution, in which *S. bovis* or *S. equinus* may be more numerous, the ratios in stored samples may rise (Feachem 1975). But it now appears that, whereas enterococci are the dominant fecal streptococcal species in humans in developed countries (and therefore human pollution is associated with falling ratios), enterococci can also be the dominant fecal streptococcal species in some animals (for instance, cats, ducks, hens, mice, pigs, rabbits, rats, and seagulls in Scotland; Wheater, Mara and Oragui 1979). Furthermore, *S. equinus* and *S. bovis* are common in the feces of people in some countries (for intance, India and Uganda; Drasar and Hill 1974). It may be concluded, therefore, that neither the ratio at the time of sampling, nor the change in ratio in a stored sample, conveys useful information about the origins of fecal pollution. The development of a routine test to distinguish human from nonhuman fecal contamination is the highest current priority for research in santirary microbiology. [See note on page 66.]

*Clostridium perfringens*

The bacterium *Clostridium perfringens* (formerly *C. welchii*) is anaerobic, spore-forming, Gram-positive, and measures approximately 4–6 micrometers in length by 1–2 micrometers in width. It is exclusively fecal in origin and is also pathogenic, causing gas gangrene and food poisoning (Chakrabarty, Narayan and Chandiramani 1977). Because it is a spore-forming organism, it can persist for long periods outside the intestine, and therefore can be used as an indicator of occasional or intermittent pollution, or of previous pollution of waters in which the presence of neither fecal coliforms nor fecal streptococci can be demonstrated (Bisson and Cabelli 1980; Cabelli 1977). *C. perfringens* is also more resistant than both fecal coliforms and fecal streptococci to antagonistic substances such as chlorine.

In wastewater bacteriology, however, its long persistence is a disadvantage because residual, dormant populations of the bacterium in waters may not reflect the true degree of pathogenic contamination. Type A *C. perfringens* from human feces may also grow in the soil (in contrast to other types of *C. perfringens* of animal origin, which seem to die-out in soil).

*Pseudomonas aeruginosa*

The organism is an opportunistic human pathogen that causes infection in wounds (especially burns) and also ear and urinary tract infections, meningitis, respiratory infections and other conditions (Cross 1979). It is often associated with sepsis in otherwise debilitated patients in hospital wards. *P. aeruginosa* is being increasingly implicated as a cause of ear infection and skin rash following exposure in inadequately disinfected swimming pools and whirlpool baths (Jacobson, Hoadley and Farmer 1976; McCausland and Cox 1975; Seyfried and Fraser 1978; Washburn and others 1976).

*P. aeruginosa* is a Gram-negative, aerobic, nonsporulating rod measuring approximately 0.5 by 2 micrometers. It occurs, normally at low concentrations of about 50 organisms per gram, in the feces of a small proportion (about 3 to 15 percent) of healthy people. It probably does not grow in the intestine of healthy people, and *P. aeruginosa* isolated in feces may be survivors of ingested bacteria. Studies in which *P. aeruginosa* was fed to volunteers demonstrated that large numbers ($\geq 10^6$) must be ingested to produce fecal carriage, which did not persist for more than 6 days (Buck and Cooke 1969).

*P. aeruginosa* is common in sink traps and flower water. It has been reported in fairly high concentrations ($>10^3$ per 100 milliliters) in urban stormwater runoff in Canada (Qureshi and Dutka 1979), and in higher concentration in sewage ($>10^5$ per 100

milliliters) and hospital sewage ($>10^6$ per 100 milliliters) in Scotland (Wheater and others 1980). It has been suggested (Cabelli, Kennedy and Levin 1976) that a consideration of *P. aeruginosa-E. coli* ratios in fecally contaminated waters can provide evidence on the possible origins of the pollution, with counts of >1,000 fecal coliforms and <1 *P. aeruginosa* per 100 milliliters being associated putatively with animal, rather than human, pollution. But *P. aeruginosa* occurs widely (albeit in highly variable numbers) in nature as a free-living organism (Green and others 1975; Parker 1971); it can therefore have little usefulness in studies of fecal contamination.

*Bifidobacterium and other anaerobic bacteria*

Bifidobacteria (previously known as anaerobic lactobacilli) are nonsporulating, anaerobic organisms that occur in the intestines of man and other animals; they are Gram-positive V- or Y-shaped cells, with each branch measuring about 0.8 by 3 to 4 micrometers. The most common species in man are *Bifidobacterium adolescentis* and *B. longum*. Bifidobacteria have recently been proposed as indicator organisms for use in tropical waters because the lactose-fermenting species are exclusively fecal in origin (Cabelli 1978; Levin 1977). They therefore overcome the principal disadvantage of fecal coliform counts on tropical samples—that such samples may contain a significant proportion of strains that can ferment lactose and produce indole at 44°C but do not derive from feces. An additional advantage of bifidobacteria is that, because they are strict anaerobes and grow poorly below 30°C, they have very low multiplication potential in extraintestinal environments. Work on bifidobacteria has only commenced relatively recently, and there is little information on their survival in extraintestinal environments other than in riverwater (Evison and Morgan 1978).

The bacterial flora of feces is predominantly composed of anaerobic bacteria (table 1-6). Bifidobacteria have been described, but feces contain large numbers of other nonsporulating anaerobes, such as *Bacteroides* spp. (commonly *B. fragilis*), the anaerobic Gram-positive cocci (*Peptoccus* spp. and *Peptostreptococcus* spp.) and *Eubacterium* spp. Current research is investigating the usefulness of these organisms as fecal indicators, but at present there are insufficient data on their extraintestinal ecology to know whether or not use of all or some of them as indicators will be practicable. Moreover, current techniques for their detection and enumeration are rather complex for routine use.

*Fecal concentrations, detection, and enumeration of bacterial indicators*

Approximate numbers of indicator bacteria commonly found in human feces are given in table 4-1. The cell counts in the table are average figures only and are mainly derived from American literature. Some communities, because of dietary differences (see table 1-6), may display considerably different numbers for one or more of the listed indicators (see also chapter 13).

Methods suitable for the detection and enumeration of coliform bacteria, fecal streptococci, and *C. perfringens* are described in the 15th edition of *Standard Methods for the Examination of Waters and Wastewaters* (American Public Health Association 1980) and in the 4th edition of *The Bacteriological Examination of Water Supplies* (Department of Health and Society Security 1969). A membrane-filter technique for enumerating *C. perfringens* is also described by Bisson and Cabelli (1979). *P. aeruginosa* populations can be counted by membrane filtration using the medium of Levin and Cabelli (1972) supplemented with 0.1 percent cetrimide (Wheater and others 1980; see also Brodsky and Ciebin 1978; Dutka and Kwan 1977; Hoadley 1977). The membrane filtration method and medium for *Bifidobacterium* spp. are described by Evison and Morgan (1978), but no satisfactory routine procedure for enumerating these bacteria has yet been developed. Reference may also be made to Mara (1974). [See note on page 66.]

## Relation of Fecal Indicator Bacteria to Excreted Pathogens

Fecal indicator bacteria were originally identified to assess the bacteriological quality of potable waters at a time when only the transmission of bacterial

Table 4-1. *Number of indicator bacteria commonly found in human feces*

| *Indicator* | *Cells per gram of feces (wet weight)* |
|---|---|
| *Bacteroides* spp. | $10^7$–$10^{11}$ |
| *Bifidobacterium* spp. | $10^7$–$10^{11}$ |
| *Clostridium perfringens* | $10^3$–$10^{10}$ |
| Coliforms | |
| Fecal | $10^6$–$10^9$ |
| Nonfecal | $10^7$–$10^9$ |
| Fecal streptococci | $10^5$–$10^8$ |

enteropathogens (such as salmonellae, shigellae, and cholera vibrios) was considered the major public health risk from drinking water. Historically (and to some extent, even now), attention has therefore focused on the relation of the fecal indicators to bacterial pathogens. Recent literature, continuing this emphasis, contains many reports on the persistence of fecal coliforms and salmonellae in the extraintestinal environment, but only a few reports on the comparative survival of the fecal indicators and nonbacterial fecal pathogens (such as viruses, protozoal cysts, and helminth eggs). This has partly been a result of the difficulty of routinely analyzing samples for these other pathogens (especially viruses), but an uncritical acceptance of the historical direction of research in the field has also contributed to the neglect. Thus, for example, there has been no report on the relation of the indicator bacterium *C. perfringens* and the eggs of the fecal helminth *Ascaris lumbricoides* (both persist for longer periods in the extraintestinal environment than do other organisms of their respective kind). Knowledge of such a relation would be of little value in assessing the safety of urban water supplies (in which *Ascaris* eggs are not a public health hazard) but it might be of assistance in assessing the quality of sewage sludges, composted feces, and some wastewater effluents.

This one example illustrates the longstanding preoccupation of sanitary bacteriologists with urban water supplies to the near exclusion of appropriate consideration of wastes and wastewaters, and of the comparative removal and persistence of fecal pathogens and indicator bacteria in treatment processes and reuse products. There are many data (mainly from North America and admittedly of variable quality) on the relation between the survival of bacterial pathogens and indicators in sewage treatment processes in temperate climates, but very little data from tropical countries. Predicting with confidence the likely density of salmonellae in a tropical sewage effluent, even when the number of fecal coliforms present is known, is extremely difficult; in contrast, reasonable estimates are possible with a temperate climate effluent. This neglect makes the establishment of a fecal coliform standard for most tropical sewage effluents a highly unscientific process. Because engineers design, for example, maturation pond systems on the basis of fecal coliform removal to the desired standard, this state of scientific uncertainty can lead to either overdesign (with a consequent unnecessary increase in cost) or underdesign (with a consequent increased risk, and perhaps actual damage, to public health).

When the hazards from nonbacterial excreted pathogens are considered, the bacterial fecal indicators are of limited usefulness. They are of some use in assessing the quality and resulting risks to health of irrigation waters, but even here the gaps in knowledge are considerable. Much of the existing information on the relation of fecal indicators and excreted pathogens comes from relatively wealthy communities (for example North America, South Africa, and Israel), and these data cannot be applied with much confidence to other communities in which climate, diet, disease patterns, agricultural practice and cultural attitudes to excreta reuse products are all different. This does not mean that information on, say, fecal coliform survival in Israel cannot be used to predict fecal coliform survival in, say, rural India but it does mean that the information may not be all that relevant to conditions in rural India, where the ability to make statements about fecal coliform survival may not help assess the degree of fecal pathogen contamination of crops irrigated with sewage effluent or fertilized with treated excreta. Caution must therefore be exercised in applying data on fecal indicator survival in environments other than those from which the information was obtained.

In summary, little is known about the relative concentrations of indicator bacteria and bacterial pathogens in effluents and fecal products in warm climates and practically no information exists on the relative concentrations of indicator bacteria and nonbacterial pathogens. In addition, it must be noted that the stability of the ratio between the concentration of an indicator bacteria and the concentration of a particular pathogen decreases as the size of the contributing population decreases. Thus, for systems serving small communities, or for individual systems such as aquaprivies or composting toilets, the ratios will vary enormously from place to place and from time to time, and no organism will act as a good indicator of another organism.

## Pathogen Indicators

Fecal indicator bacteria only demonstrate fecal contamination. This fact is useful in assessing the safety of drinking water supplies, but when the health aspects of sanitation systems, excreta and sewage treatments and reuse processes are considered, what is needed is not a fecal indicator bacterium (for feces are obviously present) but, rather, a pathogen indicator organism. A reliable measure of the total pathogen content of the end product of a treatment process is needed, so that the health risks associated with any reuse of the end

product, or with its discharge into the environment, can be gauged as accurately as possible. If these risks can be judged, responsible and informed decisions can be made—for instance, on whether the benefits from end-product reuse outweigh the possible health costs and whether further treatment is necessary to protect the health of those involved (either as producers or consumers) in the reuse process or, in the case of the end products being discharged into the environment, of the users of the environment.

It would be unrealistic to expect the same pathogen indicator organism to be useful in assessing the pathogen content of different kinds of fecal products—for example, composted feces and the effluent from waste stabilization ponds. In the former case the concern is primarily the viability of the persistent helminth eggs (notably *Ascaris lumbricoides* eggs) whereas in the latter case it is known that, if the total retention time in a stabilization pond system is more than 20 days, the effluent will be free of both helminth eggs and larvae but may contain excreted viruses and bacteria. Because of these variations, it is convenient to divide fecal products into two groups, effluents and noneffluents, and to examine which organisms are suitable pathogen indicators for each.

*Pathogen indicators for pond effluents*

The effluents from waste stabilization ponds and other sewage treatment processes are best considered separately because the vastly different retention times involved (weeks in ponds, hours or days in other processes) produce effluents with markedly different pathogen contents. If a pond system has a retention time of more than 20 days its effluent will be free from both pathogenic protozoa and helminth eggs and larvae, but it may still contain viral and bacterial pathogens. Because a routine analysis of pond effluents for pathogenic viruses and bacteria is not yet feasible (nor likely to become so in the immediate future), the choice of a suitable pathogen indicator is exceedingly difficult. Bacteriophages—and more specifically, coliphages—may provide a solution in the future but the laboratory techniques are not yet widely known. Fecal coliforms or fecal streptococci would seem an obvious choice, but there is little data on the usefulness of either as viral indicators, and the literature on their respective survival compared to pathogenic bacteria is only slightly less scant (especially for tropical pond effluents). There is no information available on the usefulness of bifidobacteria and the other non-sporulating anaerobes.

Although they are less than ideal for the purpose, fecal coliforms and fecal streptococci are perhaps the best pathogen indicators. It is difficult to determine what density of fecal coliforms or fecal streptococci—as an indication of the presence of endemic pathogens—should be permissible. The rather unhelpful answer is that the densities should be as low as possible, which in practice means at least below 1,000 per 100 milliliters of effluent (and preferably below 100 per 100 milliliters). Effluents reused for the irrigation of crops that may be consumed raw must have fecal coliform and fecal streptococci counts that are both below 100 per 100 milliliters. Viral and bacterial pathogens may or may not be absent at these indicator organism densities, but in general health risks will be so minimal that further treatment will not normally be economic.

*Pathogen indicators for effluents from other sewage treatments*

The effluents produced by sewage treatment processes other than waste stabilization ponds are likely to contain a full range of fecal pathogens—viruses, bacteria, protozoal cysts, and helminth eggs. There is no suitable fecal indicator organism in these circumstances; it is not possible to have a single organism indicate the presence of so diverse a group of pathogens. Fecal coliforms have been used, but only for historical reasons (they are totally inappropriate indicators for the helminth eggs for instance). This subsection can conclude with the generalization that if a sound economic argument can be put forward for the use of treatment processes other than ponds, then the effluent from such treatments should undergo tertiary treatment or be heavily disinfected or discharged well out to sea because, in the tropics, the health risks from the effluent may be similar to those from raw sewage. It should be noted that heavy disinfection is often ineffective (especially against viruses and helminth eggs) and has undesirable environmental consequences.[2]

*Pathogen indicators for noneffluents*

Noneffluents are taken here to include night soil, the contents of pit latrines and composting toilets and the sludges from aquaprivies, septic tanks and conventional sewage treatment works. It is reasonable to assume that, if ascariasis is endemic and there are no viable *Ascaris* eggs present in the wastes analyzed, then

2. Effluent disinfection is discussed further in chapters 6, 9, 13, and 23.

other pathogens are absent as well, since *Ascaris* eggs are so resistant. Thus, and in the current absence of any data on the comparative survival of the bacterium *C. perfringens*, the viable eggs of *Ascaris lumbricoides* would appear to be the best pathogen indicator currently available for noneffluents. This indicator has been accepted in China, where standards of 95 percent *Ascaris* egg mortality have been adopted for the agricultural reuse of excreta (McGarry and Stainforth 1978).

## Survival of Indicators and Pathogens

From the time of excretion, the concentration of all pathogens usually declines from the death or loss of infectivity of a proportion of the organisms. Viruses and protozoa will always decrease in numbers following excretion, but bacteria may multiply if they find themselves in a suitably nutrient-rich environment with a minimum of competition from other microorganisms. This can occur when salmonellae, for instance, contaminate certain foods, or when *E. coli* multiply in a chlorinated sewage effluent from which many other bacteria have been eliminated. Multiplication of bacterial pathogens is generally rare, however, and is unlikely to continue for very long. Intestinal helminths—except the trematodes, which have a multiplication phase in their molluscan intermediate hosts—will decrease in numbers following excretion. The multiplication possibilities for the excreted pathogens were summarized in table 2-3.

The ability of an excreted organism to survive is defined as its persistence (discussed in chapter 2). The natural death of organisms when exposed to a hostile environment is of the utmost importance because it reduces the infectivity of excreta independently of any treatment process. In fact, some treatment processes have little effect on excreted pathogens and simply allow the necessary time for natural die-off to occur. The effect of conventional sewage treatment on protozoal cysts is of this kind (see chapter 20). Certain treatment processes, however, create conditions that are particularly hostile to excreted pathogens and that promote their rapid death. The effects of activated sludge on fecal bacteria, or of thermophilic digestion on all organisms, are of this kind. The essential environmental factors in limiting pathogen persistence are time and temperature. The success of a given treatment process in reducing the pathogenicity of an effluent or sludge thus depends, in general, upon its retention time and its creation of an environment especially hostile to particular organisms. The sole environmental condition likely found in a night soil or sewage treatment system that is highly fatal to all pathogens in a reasonably short time (a few hours) is raised temperature (in the range 55–65°C). The only other low-cost process that causes 100 percent removal or destruction of most pathogens is the waste stabilization pond system with its long retention times, exposure to sunlight, and good sedimentation properties.

The elapse of time is a feature common to all treatment, disposal and reuse technologies; in many cases, it is the feature that most determines the pathogen removal achieved. The rate of loss of infectivity of an organism also depends very much on temperature; most organisms survive well at low temperatures ($\approx$5°C) and rapidly die at high temperatures (>40°C). Except in sludge or night soil digestion processes, temperatures approximate environmental temperatures—in most developing countries, generally in the range of 15–35°C and commonly of 20–30°C. It is therefore useful to know the persistence of pathogens at ambient temperatures in different environments so that the likely pathogen content of various fecal products can be predicted. In this section pathogen survival at ambient temperatures is reviewed with the following considered in turn: survival in feces, night soil and sludge; survival in water and sewage; survival in soil; and survival on crops. Under each heading that follows, the available knowledge is summarized as succinctly as possible. A greal deal of additional information is given in Part Two and some of the data are further tabulated in Feachem and others (1980).

The shape of the curve describing pathogen survival over time should determine the way in which survival is reported. Many bacterial populations decline exponentially, so that 90 or 99 percent of the bacteria are lost relatively quickly with a few organisms persisting for longer periods. Such a situation is best described by the probability of survival for a given time or by half life, the time required for half the population to die. For instance, 50 percent of fecal coliforms may die in 20 hours in water, whereas a few may persist for up to 50 days, and the results obtained will depend heavily on sampling procedures. Most of the literature gives data on the persistence of the small proportion of long-term survivors and only a few authors have reported the shape of the death curve or given the 50 to 90 percent destruction times. The discussion below will therefore mainly concern the overall persistence of a few organisms. This focus is epidemiologically appropriate for organisms that can replenish their numbers if they find themselves on food or other suitable substrates

(for example shigellae, salmonellae and pathogenic *E. coli*) or for organisms whose infective dose is believed to be low (the excreted viruses, for example). It is less appropriate for cases in which regrowth is unlikely and infective doses may be high (for example, *Vibrio cholerae*); in these cases it is the rapid decline of the bacteria to a level that no longer presents a major public health hazard that is important. In organisms having several developmental stages outside the human host (such as hookworms and schistosomes), each stage will have its own separate survival pattern. When a developmental stage is actively moving yet dependent on an unreplenished energy source (for example, the schistosome miracidium seeking its snail host) the length of life may be precisely defined.

*In feces, night soil, and sludge*

There is less literature on the survival of pathogens in these media than in the aqueous environments discussed in the following subsection. Some sources refer to survival of pathogens in sewage works' sludges, but survival in feces and night soil may be assumed to be broadly similar. Research on pathogen survival in these media may be summarized as shown in table 4-2.[3]

*In water and sewage*

Many studies on the survival of excreted organisms in water and sewage have been conducted.[4] The data are summarized in table 4-3. For all organisms survival is highly dependent on temperature, with greatly increased persistence at lower temperatures. Survival of bacteria is also highly dependent on the presence of other microorganisms in the water that might provide competition or predation. Bacteria often survive longer in clean water than in dirty water and the longest survival times are obtained by inoculating a single bacterial species into sterilized polluted water. There is some evidence that virus survival is enhanced in polluted waters, presumably a result of some protective effect that the viruses may receive when they are adsorbed onto solid particles in dirty water (see chapter 9). Coliforms, in particular *E. coli*, have attracted the most interest because of their established role as indicator bacteria. Substantial regrowth of coliforms is possible in organically polluted waters, but this growth phase will give way to a progressive die-off. Survival in excess of 50 days is most unlikely and, at 20–30°C, 20 days is a more likely maximum survival time. Mixed fecal streptococci have a similar (perhaps a little longer) survival but, if the streptococci are predominantly *S. bovis* or *S. equinus*, the survival times are substantially shorter (see chapter 13). *Salmonella* survival has also been widely reported. Survival of over 2 months has been recorded, but 1 month is a more common upper limit (see chapter 15). *Shigella* spp. and

Table 4-2. *Survival times of excreted pathogens in feces, night soil, and sludge at 20–30°C*

| *Pathogen* | *Survival time* (days) |
|---|---|
| Viruses | |
| Enteroviruses[a] | <100 but usually <20 |
| Bacteria | |
| Fecal coliforms | <90 but usually <50 |
| *Salmonella* spp. | <60 but usually <30 |
| *Shigella* spp. | <30 but usually <10 |
| *Vibrio cholerae* | <30 but usually <5 |
| Protozoa | |
| *Entamoeba histolytica* cysts | <30 but usually <15 |
| Helminths | |
| *Ascaris lumbricoides* eggs | Many months |

a. Includes polio-, echo-, and coxsackieviruses.

3. A compilation of original sources and findings on survival in feces, night soil, and sludge can be found in the appropriate sections of Part Two, chapters 9 through 35.

Table 4-3. *Survival times of excreted pathogens in fresh water and sewage at 20–30°C*

| *Pathogen* | *Survival time* (days) |
|---|---|
| Viruses[a] | |
| Enteroviruses[b] | <120 but usually <50 |
| Bacteria | |
| Fecal coliforms[a] | <60 but usually <30 |
| *Salmonella* spp.[a] | <60 but usually <30 |
| *Shigella* spp.[a] | <30 but usually <10 |
| *Vibrio cholerae*[c] | <30 but usually <10 |
| Protozoa | |
| *Entamoeba histolytica* cysts | <30 but usually <15 |
| Helminths | |
| *Ascaris lumbricoides* eggs | Many months |

a. In seawater, viral survival is less, and bacterial survival is very much less, than in fresh water.

b. Includes polio-, echo-, and coxsackieviruses.

c. *V. cholerae* survival in aqueous environments is a subject of current uncertainty—see chapter 17.

4. These studies are reviewed in the appropriate sections of Part Two, chapters 9 through 35.

*Vibrio cholerae* are less persistent, and survival of these bacteria for more than 20 days is seldom reported (see chapters 16 and 17).

The development of viral detection techniques in the 1950s led to the demonstration of the presence of excreted viruses in the environment. The enteroviruses (polio-, coxsackie-, and echoviruses) have been frequently isolated from water and wastewater (chapter 9) and the literature on this subject is growing rapidly at the present time. Viral survival may be longer than bacterial survival and it is greatly increased at lower temperatures. In the 20–30°C range, 2 months seems a typical survival time, whereas at around 10°C, 9 months is a more realistic figure.

Protozoal cysts are poor survivors in any environment. A likely maximum for *Entamoeba histolytica* in sewage or polluted water is about 20 days (see chapter 20). Helminth eggs vary from the very fragile to the very persistent. The most persistent of all are *Ascaris* eggs, which may survive for a year or more (see chapter 23).

### *In soil*

Survival times in soil are relevant in all situations where effluent, sludge, compost, or other fecal products are being applied to the land as fertilizers or soil conditioners.[5] Several factors, shown in table 4-4, affect the survival time of enteric bacteria in soil (Gerba, Wallis and Melnick 1975). Fecal coliforms can survive for many months under optimal conditions. In warm climates, especially when arid, survival is limited to 2–3 months at most. Fecal streptococcal survival is likely to be longer if human enterococcal species are dominant (see chapter 13). Survival of salmonellae may be up to 1 year if the soil is cool, moist and rich in organics (for example, if it is fertilized), but strain variation is considerable and 50 days would be a more typical maximum (see chapter 15). Data on *Shigella* or *Vibrio cholerae* survival in soil are limited (see chapters 16 and 17).

The information available on viruses suggests that virus particles adsorb to soil particles and become protected from environmental factors. Viral survival is greater at low temperatures: survivals of up to around 3 months have been reported in warm weather, increasing to around 5 months in European winter conditions (see chapter 9). Protozoal cysts in soil are most unlikely to survive for more than 10 days (see chapter 20). Helminth egg survival varies enormously,

5. A comprehensive review of the persistence of excreted pathogens in soil is contained in the appropriate sections of Part Two, chapters 9 through 35.

Table 4-4. *Factors affecting survival time of enteric bacteria in soil*

| *Soil factor* | *Effect on bacterial survival* |
|---|---|
| Antagonism from soil microflora | Increased survival time in sterile soil |
| Moisture content | Greater survival time in moist soils and during times of high rainfall |
| Moisture-holding capacity | Survival time is less in sandy soils than in soils with greater water-holding capacity |
| Organic matter | Increased survival and possible regrowth when sufficient amounts of organic matter are present |
| pH | Shorter survival time in acid soils (pH 3–5) than in alkaline soils |
| Sunlight | Shorter survival time at soil surface |
| Temperature | Longer survival at low temperatures; longer survival in winter than in summer |

*Source:* Adapted from Gerba, Wallis and Melnick (1975).

but *Ascaris* eggs can survive for several years (see chapter 23). The situation is summarized in table 4-5.

### *On crops*

Excreted viruses and bacteria cannot penetrate undamaged vegetable skins. However, there are many reports in the literature on the isolation of all kinds of excreted pathogens from the surface of vegetables that have been irrigated or fertilized with fecal products.[6] Root vegetables are more prone to contamination than others. Weather conditions have an important

Table 4-5. *Survival times of excreted pathogens in soil at 20–30°C*

| *Pathogen* | *Survival time* (days) |
|---|---|
| Viruses | |
| Enteroviruses[a] | < 100 but usually < 20 |
| Bacteria | |
| Fecal coliforms | < 70 but usually < 20 |
| *Salmonella* spp. | < 70 but usually < 20 |
| *Vibrio cholerae* | < 20 but usually < 10 |
| Protozoa | |
| *Entamoeba histolytica* cysts | < 20 but usually < 10 |
| Helminths | |
| *Ascaris lumbricoides* eggs | Many months |

a. Includes polio-, echo-, and coxsackieviruses.

influence on the survival of pathogens on plants; warmth, sunshine, and low air humidity greatly promote pathogen death. The survival characteristics of various excreted organisms on crops may be summarized as shown in table 4-6. As indicated in the table, pathogen survival times on vegetables are short compared to survivals in other environments. Protozoal cysts are rapidly killed. Viruses, bacteria, and worm eggs survive for longer, but little survival of any species is to be expected after 2 months.

## Pathogen Survival versus Removal in Waste Treatment

Pathogen survival, rather than pathogen removal, is purposely referred to in this book. This is because health hazards are posed by the pathogens that survive a treatment process, not by those that are removed by treatment. Figures such as 99 percent or 99.9 percent removal appear highly impressive but they represent 1 or 0.1 percent survival, respectively, and this degree of survival may be highly significant wherever incoming concentrations are great. If an influent to a sewage works contains, say, $10^5$ pathogenic bacteria per liter, then 99 percent removal will produce an effluent with $10^3$ pathogenic bacteria per liter. In areas where the effluent is to be reused, or where it is to be discharged to a stream that populations downstream use as a source of drinking water, such effluent quality may be inadequate.

Table 4-6. *Survival times of excreted pathogens on crops at 20–30°C*

| *Pathogen* | *Survival time* (days) |
|---|---|
| Viruses | |
| Enteroviruses[a] | <60 but usually <15 |
| Bacteria | |
| Fecal coliforms | <30 but usually <15 |
| *Salmonella* spp. | <30 but usually <15 |
| *Shigella* spp. | <10 but usually <5 |
| *Vibrio cholerae* | <5 but usually <2 |
| Protozoa | |
| *Entamoeba histolytica* cysts | <10 but usually <2 |
| Helminths | |
| *Ascaris lumbricoides* eggs | <60 but usually <30 |

a. Includes polio-, echo-, and coxsackieviruses.

6. These reports are reviewed in the appropriate sections of Part Two, chapters 9 through 35.

The emphasis in the literature on the exact proportions of pathogens removed by various treatment processes is thus misleading. For instance, most conventional treatment plants remove 90 to 99 percent of enteric bacteria.[7] This is a very poor removal; whether trickling filters remove a little less (say 95 percent) than activated sludge plants (say 99 percent), they are both technologies with poor pathogen removal characteristics (but they were never designed to have them—see the next section). A removal ability of less than 99 percent means always more than 1 percent survival, or always less than a log unit reduction of 2. In developing countries, where incoming wastes have high concentrations of pathogens (especially viruses, bacteria, and protozoal cysts—see table 1-10), a survival of more than 1 percent is usually inadequate.

In considering treatment technologies by their ability to remove pathogens, it is necessary not to dwell on trivial differences (for instance, 92.3 percent versus 97.8 percent removal), but to translate removal efficiencies into orders of magnitude. Conventional treatment works remove between 1 and 2 log units of enteric bacteria and should be contrasted with technologies, such as waste stabilization ponds, which remove 5 log units. In considering stabilization ponds or thermophilic digesters, which have high removal performances, it is also misleading to talk in terms of percentage removal (use of this convention disguises, for instance, the important difference between 99.99 and 99.999 percent removal).

The removal characteristics of treatment technologies should be related to the incoming concentrations of particular pathogens, to the intended reuse or disposal arrangements, and to the associated health risks. Different pathogens occur in varying concentrations and are affected in different ways by a given treatment technology. For instance, protozoal cysts will be found in raw sludge in relatively low numbers and will not survive sludge treatment. In contrast, *Ascaris* eggs may be found in sludge in high concentrations and will survive most sludge treatment processes.

## Objectives of Night Soil and Sewage Treatments

The primary objective in the treatment of night soil or sewage from communities in which excreted infections are endemic is the destruction of excreted

7. The processes that conventional sewage treatment comprises, and their ability to remove various excreted pathogens, are discussed in chapter 6 and reviewed at length in the chapters of Part Two.

pathogens. This is principally achieved by a combination of time and temperature, although other conditions of the extraintestinal environment are also important (for example, sunlight and oxygen availability). From the extensive literature review in part 2, it appears that no excreted pathogen—with the exception of spore-forming bacteria (for example *C. perfringens*) and possibly hepatitis A virus—can survive a temperature of more than 65°C for a few minutes. As the temperature falls survival increases; thus, at 10°C, for instance, *Ascaris* eggs may survive for several years, enteroviruses for 12 months, and shigellae for 2–3 months.

The degree to which night soil and sewage are treated is largely influenced by what is to be done with the sludge, compost or sewage effluent.[8] It is thus accepted engineering practice to discharge untreated sewage into the sea, provided that the outfall is designed to ensure that no pollution of beaches or shellfish-growing areas will occur; but if reuse of an effluent for the irrigation of edible crops is intended, the designer's goal should be the absence of excreted pathogens on the surface of crops, and he should accordingly design the treatment works for a very low degree of pathogen survival.

### *Excreta and night soil treatment*

The effectiveness of treatment methods for excreta and night soil depends greatly upon their time-temperature characteristics. The effective processes are those that retain the excreta for a long time (>1 year), or make it warm (>55°C), or effectively combine adequate retention time and high temperature.

Pit latrines (see the section of that title in the next chapter) have a useful life of a few years; when one becomes full, a second is dug, and the contents of the first are left undisturbed while the second is in use. Because of the time interval there are no health hazards associated with digging out the contents of previously filled and covered pit latrines. Provided the squatting plate is regularly cleaned, pit latrines pose no greater risks to health than do flush toilets (though insect breeding can be a serious problem—see chapters 36 and 37—and odor can be a nuisance).

Composting toilets (see the section of that title in the next chapter) are of two types: batch and continuous. If the composting period is over 1 year, only a few *Ascaris* eggs will be present in the product. With composting periods of under 1 year, varying numbers of other excreted pathogens will be present (see table 5-1). Composting toilets thus have definite health risks that, although slight, should be recognized by the designers and users of these systems. In strictly economic terms the value of the compost must be greater than the possible cost to health from its use.

The health hazards associated with the collection of night soil from bucket and vault latrines are described in the section "Cartage Systems" in the next chapter. If urine is collected as well as feces, the night soil is a fecal suspension similar to primary sewage sludge and may be treated by mesophilic or thermophilic digestion. It also may be treated in a pond system which can be designed to produce little effluent so that very long retention times are possible (>1 year) and, consequently, no survival of excreted pathogens. If the urine is not collected, or is allowed to drain away, the night soil (now principally feces) may be disposed of, treated, and reused in a number of ways (see also the section "Composting" in chapter 5). Night soil cartage and treatment systems will tend to have higher health risks than many other systems, although risks can be greatly reduced by the use of modern methods (such as those found in Japan). In high-density urban settings, where the only technological alternative may be a sewerage system, cartage systems will often be economically attractive despite their health hazards. In other settings, where a greater range of technologies is feasible, cartage may be less attractive.

### *Sewage treatment*

Those whose job is to select and design appropriate systems for the collection and treatment of sewage in developing countries must bear in mind that European and North American practices do not represent the zenith of scientific achievement, nor are they the product of a logical and rational design process. Rather, treatment practices in the developed countries are the product of history, a history that started about 100 years ago when little was known about the fundamental physics and chemistry of the subject and when practically no applicable microbiology had been discovered. Only since 1970 have the tools to do serious work in water and wastewater virology been developed, and only since 1975 have the roles of rotavirus, *Campylobacter*, and *E. coli*[9] in the etiology of diarrheas been demonstrated.

8. Treatment strategies for different reuse and disposal practices are discussed in chapter 7.

9. The epidemiology of infections with rotavirus, *Campylobacter*, and pathogenic *E. coli* are reviewed in chapters 11, 12, and 13, respectively.

The historical development of European and North American sewerage systems can be roughly summarized as follows:

- A growing awareness of squalor in the large cities and the consequent risks to health led to the construction of sewers that discharged raw wastes into rivers (in the mid-nineteenth century in London, for instance).
- This discharge of raw wastes yielded massive pollution and oxygen depletion in the rivers, which often became foul, open sewers.
- Various treatment technologies were developed to reduce the suspended load and the oxygen demand of the discharged wastes (for example, the UK Royal Commission on Sewage Disposal, 1899–1915, proposed effluent standards of $<30$ milligrams per liter for suspended solids and $<20$ milligrams per liter for biochemical oxygen demands, or BOD).
- In the 1950s and 1960s, a growing awareness of environmental problems, coupled with a now greatly increased population, led to tertiary treatment processes being introduced to protect receiving waters from further oxygen depletion, toxic substances, and eutrophication.
- At the same time it became clear that these sophisticated treatment technologies were not efficient at removing pathogenic microorganisms. Thus, in countries where environmental concern was acute (for example, the USA), or where effluents were commonly reused (for example, Israel), effluent chlorination was borrowed from the water treatment industry as a way of killing bacteria (and possibly viruses) in effluents. This technology, however, brought with it new and different environmental concerns.[10]

This highly simplified account illustrates the historical and conservative nature of the development of current waste treatment practices in industrialized countries. These practices are not especially clever, nor logical, nor completely effective—and it is not necessarily what would be done today if these same countries had the chance to start again.

Fluid retention times in conventional sewage works, oxidation ditches, and aerated lagoons treating domestic sewage are commonly less than 1, 3, and 6 days, respectively. Septic tanks typically have retentions of 1–3 days. These short retention times, in conjunction with temperatures that rarely exceed 35°C, allow high pathogen survivals, and the full range of excreted pathogens present in the raw sewage appears in the effluent. The sludges produced in conventional sewage works and oxidation ditches also contain the full range of excreted pathogens and require some form of treatment before disposal or reuse.

Conventional sewage works were originally developed in order to prevent gross organic pollution in European and North American rivers; they were never intended to achieve high removal of excreted pathogens. Their use in tropical countries in which excreted infections are endemic is only justifiable in special circumstances, for there is an alternative treatment process much superior in obtaining low survivals of excreted pathogens—the waste stabilization pond system.[11] Retention times commonly encountered in properly designed pond systems are $>25$ days, and this feature, in conjunction with such environmental factors as sunlight, high oxygen content and the presence of algal toxins, is responsible for the ability of pond systems to reduce greatly the survival of excreted pathogens. Indeed, protozoal cysts and helminth eggs and larvae can be completely eliminated from pond effluents. Pond systems have several more advantages over other treatment methods: they are the cheapest form of treatment, both to construct and operate, with minimal requirements for foreign exchange; their maintenance is very simple, requiring only unskilled labor; they are easily designed to achieve any required degree of treatment; and the algae produced in the ponds are a potentially valuable source of protein.[12]

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10. See chapter 6, the section "Effluent Chlorination."

11. Waste stabilization ponds are examined in more detail in the section of that title in chapter 6.

12. See the section "Reuse in Aquaculture" in chapter 7.

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## Note added in proof

Recent work in Yorkshire, England (Oragui 1982) has led to the development of bacteriological methods for distinguishing between human and animal fecal pollution of waters. *Streptococcus bovis*, which can be enumerated in water samples by the method of Oragui and Mara (1981), appears to be excreted exclusively by animals, whereas sorbitol-fermenting strains of *Bifidobacterium adolescentis* and *B. breve* are only excreted by man. Enumeration media for both sorbitol-fermenting and total bifidobacteria are described by Oragui (1982). These methods for distinguishing between human and animal pollution are currently being evaluated in Mexico, Nigeria and Zimbabwe.

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# 5

# Health Aspects of Excreta and Night Soil Systems

IN THIS AND THE NEXT CHAPTER, the health implications of the principal varieties of excreta collection and treatment systems are discussed. These are separated into night soil (or "dry") and sewage (or "wet") systems. (The health implications of reuse and effluent-discharge practices are considered in chapter 7.) Little attention is paid here to the technical details of the systems examined, except to those bearing on specific health problems. The reader wishing more information on technical aspects should consult the second volume of this series (Kalbermatten and others 1982), the related document published by the International Development Research Centre (Rybczynski, Polprasert, and McGarry 1978), and standard sanitary engineering texts. In this chapter, three excreta collection systems—the pit latrine and its various modifications, the composting latrine, and cartage systems—are described, and the discussion concludes with an examination of the health implications of dry treatment of night soil by trenching and composting. Excreta collection and treatment by wet systems are examined in chapter 6.

## Pit Latrines

Pit latrines are the simplest of all on-site disposal systems. Excreta fall into a hole in the ground, and a new pit is dug when the hole is about two-thirds full (see figure 5-1). A ventilated improved pit (VIP) latrine, and a modified pit latrine called a ROEC (Reed Odorless Earth Closet), are shown in figures 5-2 and 5-3, respectively. Pits are covered by sqatting slabs, seats, or pour-flush bowls.

### *Cleanliness*

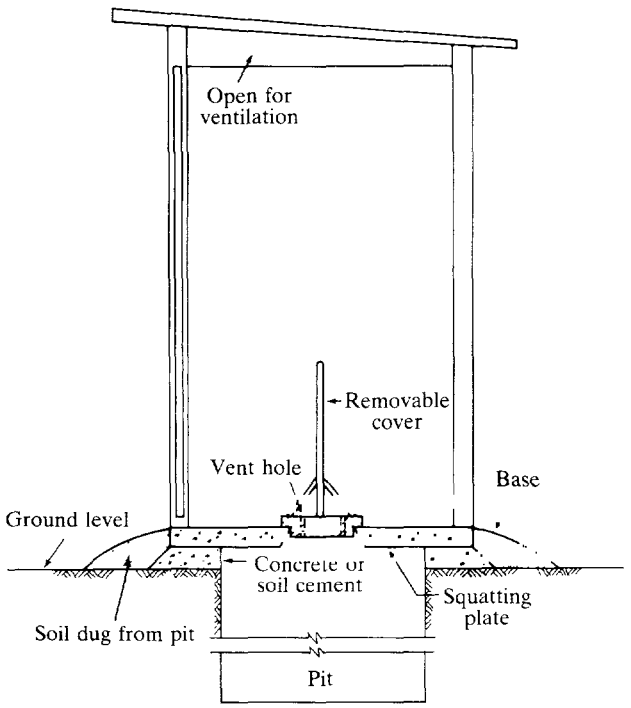
In all latrines cleanliness is of the utmost importance. Squatting slabs easily become fouled and pour-flush bowls may block up. Fouled and unhygienic pit latrines are found all over the world, often because they have been constructed in communities previously accustomed to defecation on the open ground who have also had inadequate community involvement or health education. Fouled pit latrines become a focus of disease transmission and may make health matters worse than before the sanitation intervention.

### *Odor*

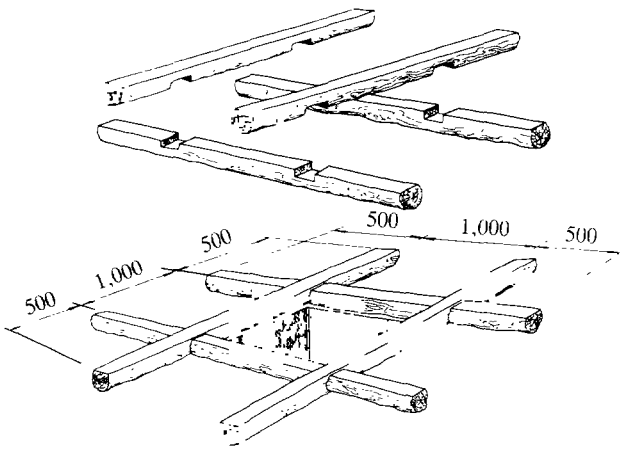
Pit latrines with squatting slabs often are malodorous. If they are, they may not be used and thus cannot yield any potential benefits in improved health. Odors can virtually be eliminated by fitting a vent pipe to the pit. This pipe should be at least 100 millimeters in diameter, painted black, and fitted on the sunny side of the latrine so that it can heat up, the heat creating an updraught. [See note on page 82.]

### *Insect breeding*

Pit latrines with squatting slabs will usually become breeding sites for flies. Flies that visit a pit latrine to breed or feed may carry pathogens when they leave and thus promote disease transmission. If the pits are wet, they may also become *Culex pipiens* breeding sites. Well constructed pits with pour-flush bowls will not allow such insect breeding. If squatting slabs are used, a vertical vent pipe—100–200 millimeters in diameter, covered by a fly screen, and combined with a dark interior to the superstructure—will greatly reduce both the amount of fly breeding and the escape of any flies that do breed. Flies breeding in the pit will be attracted by the light coming down the vent pipe and will attempt to escape by this route, only to be prevented by the fly screen. The effect of vent pipes on mosquito breeding in wet pits remains uncertain and the latest findings are reviewed in chapter 36.




**Side view**




**Alternative base using hewn logs**

Figure 5-1. *Conventional unimproved pit latrine* (dimensions in millimeters). In termite-infested areas, use treated wood or termite barrier. From Kalbermatten and others (1982); adapted from Wagner and Lanoix (1958)

*Pathogen survival in the pit*

Most pit latrines are filled in when two-thirds to three-quarters full and are either never dug up again or only dug up after many years. In either case pathogen survival is of no concern because all pathogenic organisms will be dead. In some areas, however, two alternating pit sites are used, a pit is dug out a year or two after closing, and the contents are used as fertilizer. This system resembles the double-vault composting toilet (see below) except that it operates on a longer cycle. If the pit has been left for a minimum of one year, there will be no viable pathogens (except, possibly, a few *Ascaris* eggs). The chances of viable *Ascaris* eggs being present are greater if the pit is wet and partly below the water table. The risk involved in reusing material that has been buried for at least 12 months is small, however, and the pit contents may immediately be used on the fields with confidence. [See note on page 82.]

*Groundwater pollution*

Pollution of this kind is a genuine hazard in areas where pit latrines are widely used and where the groundwater is high and is used as a water source. The subject is discussed in detail in chapter 7.[1]

## Composting Toilets

Developed countries have shown a growing interest in composting toilets because these sewerless facilities circumvent financial and ecological problems attendant on the waterborne disposal of human wastes.[2] Financial considerations and the lack of municipal effort required to maintain composting toilets make their use attractive in some developing countries, for which agricultural reuse of the composted product is an additional benefit. The precautions necessitated by the problem of pathogen survival in this product, however, must be noted.[3]

*Technical description*

There are two basic kinds of composting toilets, continuous and batch. Both require the addition of a carbon source, such as garbage, vegetable leaves, or sawdust. The continuous composting toilets are based on the Swedish "multrum" toilets, and an example of such a design is shown in figure 5-4. They have been under trial in Tanzania and Botswana since 1977 but have had no wide application in developing countries.

1. See the subsection "Effluent Discharge. To groundwater."

2. See chapter 1, "Characteristics of Sullage."

3. See chapter 3, "Limitations in Assessing Health Benefits" and table 3-2; see also chapter 4, "Objectives of Night Soil and Sewage Treatments. Excreta and night soil treatment."

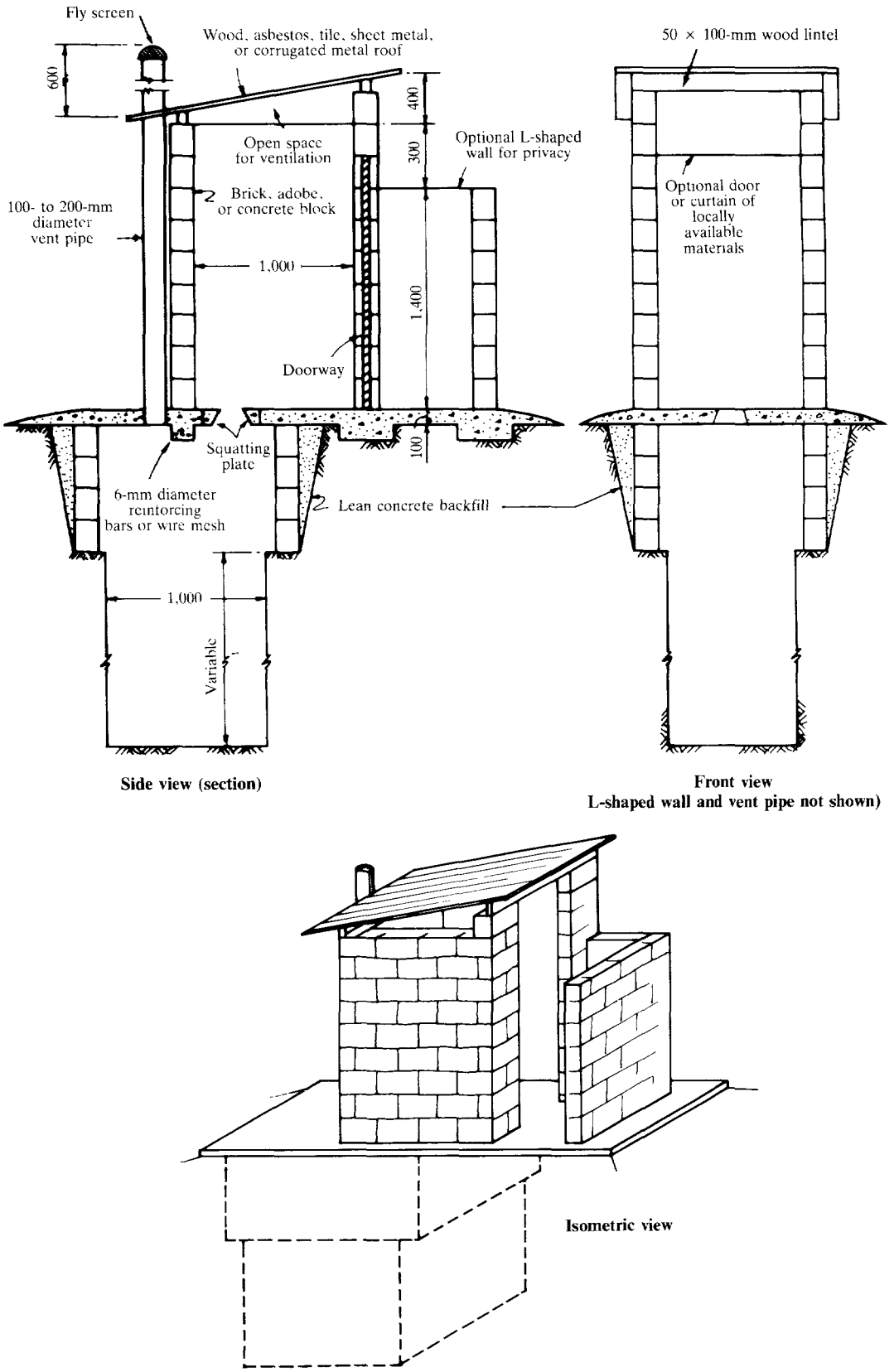



Figure 5-2. *Ventilated improved pit (VIP) latrine* (dimensions in millimeters). In the side view, a pedestal seat or bench may be substituted for the squatting plate. An opening for desludging may be provided next to the vent pipe. Dimensions of the bricks or concrete blocks may vary according to local practice. Wooden beams, flooring, and siding may be substituted for concrete block walls and substructure. From Kalbermatten and others (1982)

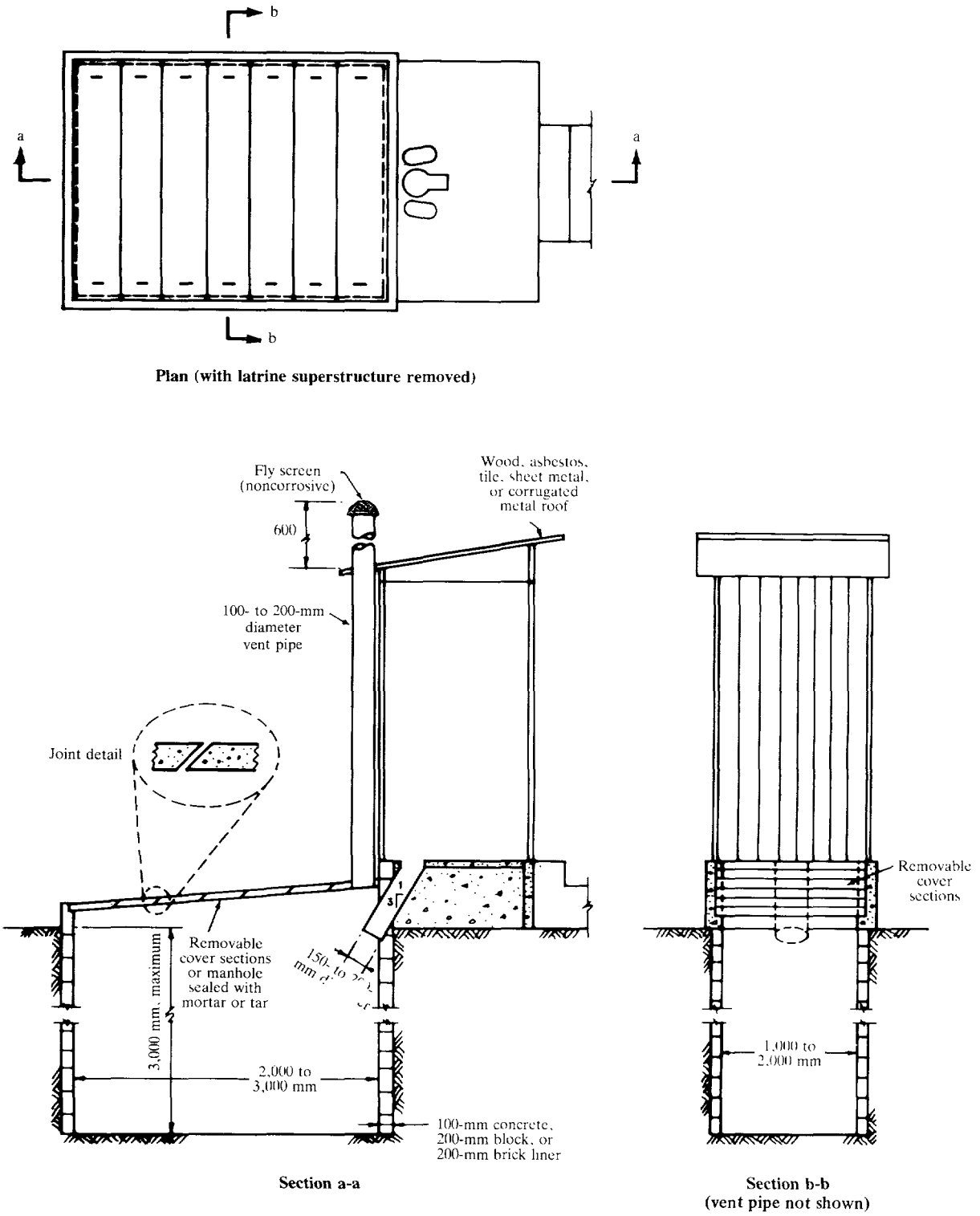



Figure 5-3. *Reed Odorless Earth Closet* (ROEC) (dimensions in millimeters). Pedestal seat with curved chute may be substituted for squatting plate. Construction materials and dimensions for the superstructure may vary according to local practice. From Kalbermatten and others (1982)

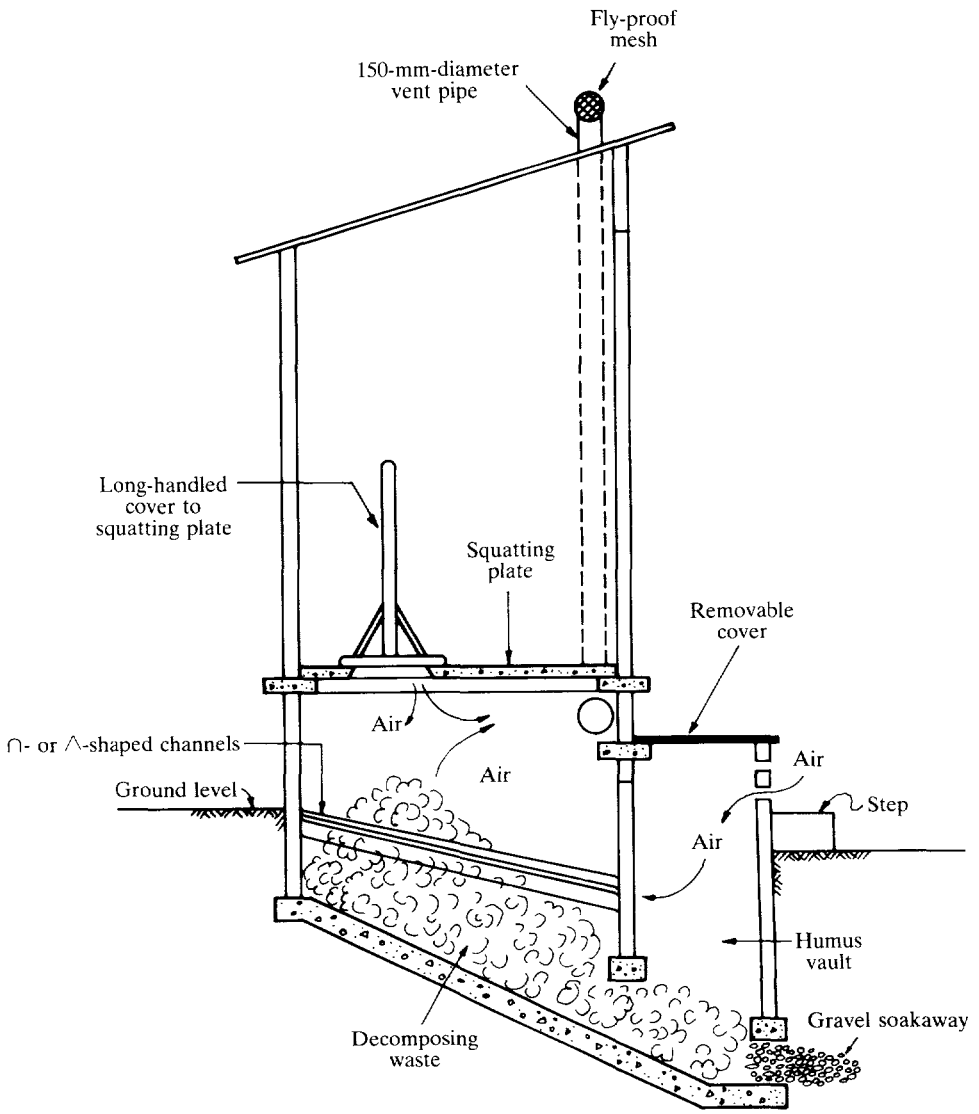


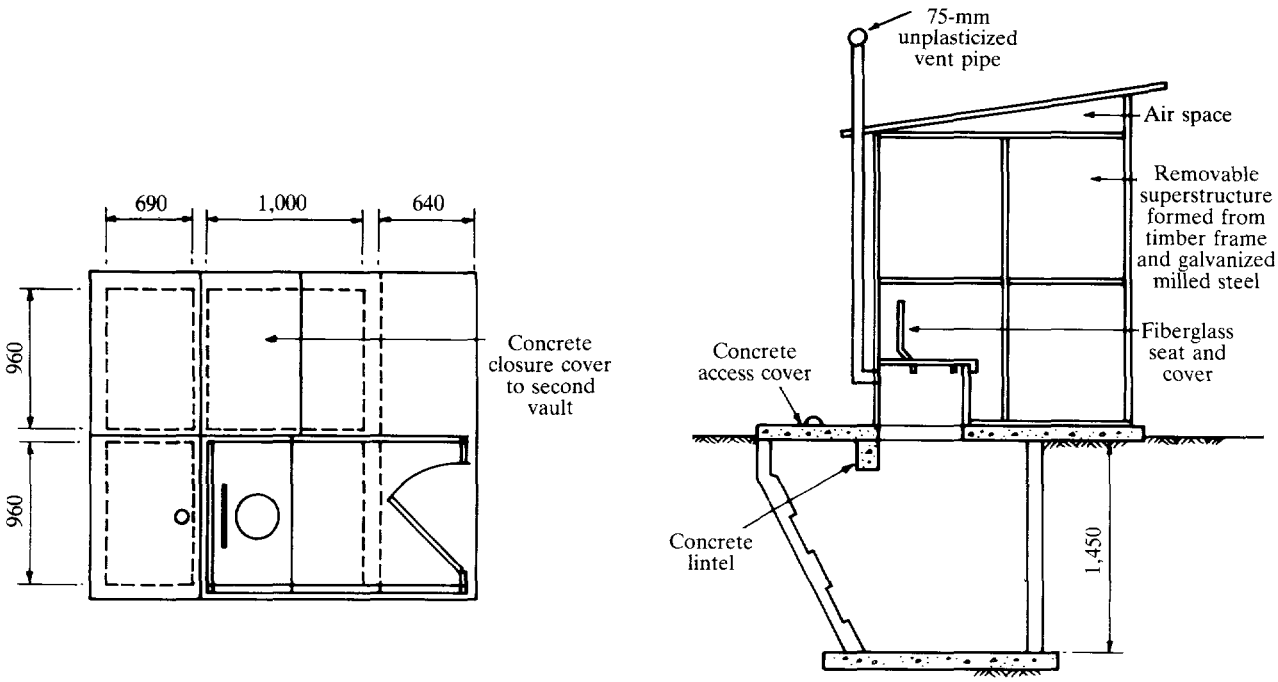

Figure 5-4. "Multrum" *continuous-composting toilet.* From Kalbermatten and others (1982); adapted from a drawing by U. Winblad

Only limited and inadequate microbiological data exist on continuous composters (Gurak 1978; reviewed by Feachem, Mara and Iwugo 1980). The batch composter is common in China and Vietnam, and the most usual design is the double vault (see figure 5-5). Again, no appreciable microbiological data on these toilets have been located, although such data may exist in China and Vietnam.

*Pathogen survival in product*

In both kinds of composting toilet, the composted product is used as an agricultural fertilizer and soil conditioner. It is important, therefore, that pathogen destruction should be as complete as possible. The two main factors affecting the survival of excreted pathogens are time and temperature. Temperature in the composting pit or vault depends on the air supply, the C:N ratio, and the moisture content. If the digestion is anaerobic, the temperature may remain ambient or it may rise at most to around 35°C. If it is aerobic, the temperature will rise to the 50–70°C range if the C:N ratio and moisture content are correctly regulated. These conditions may be difficult to achieve, especially in arid developing countries where little organic material (needed as a source of carbon) is available for adding to the wastes.

It is certain that double-vault composters will be anaerobic, and it is probable that multrums will be also. Anaerobicity and ambient temperature certainly



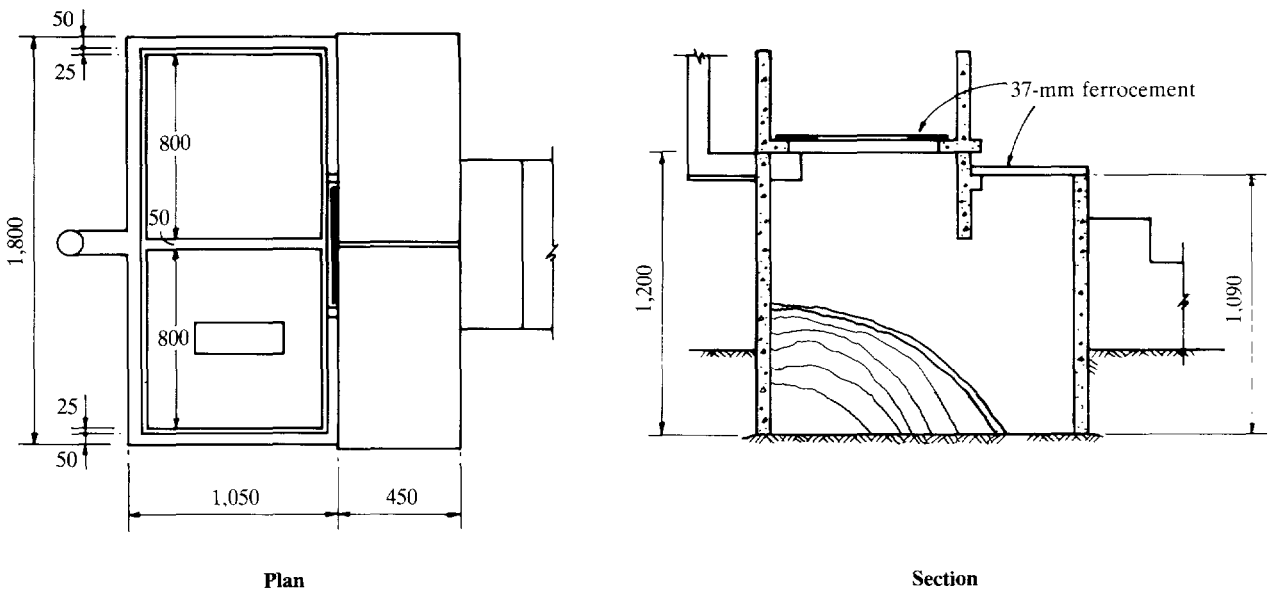




Figure 5-5. *Double-vault composting (DVC) toilet* (dimensions in millimeters). From Kalbermatten and others (1982); top, adapted from a drawing by R. A. Boydell

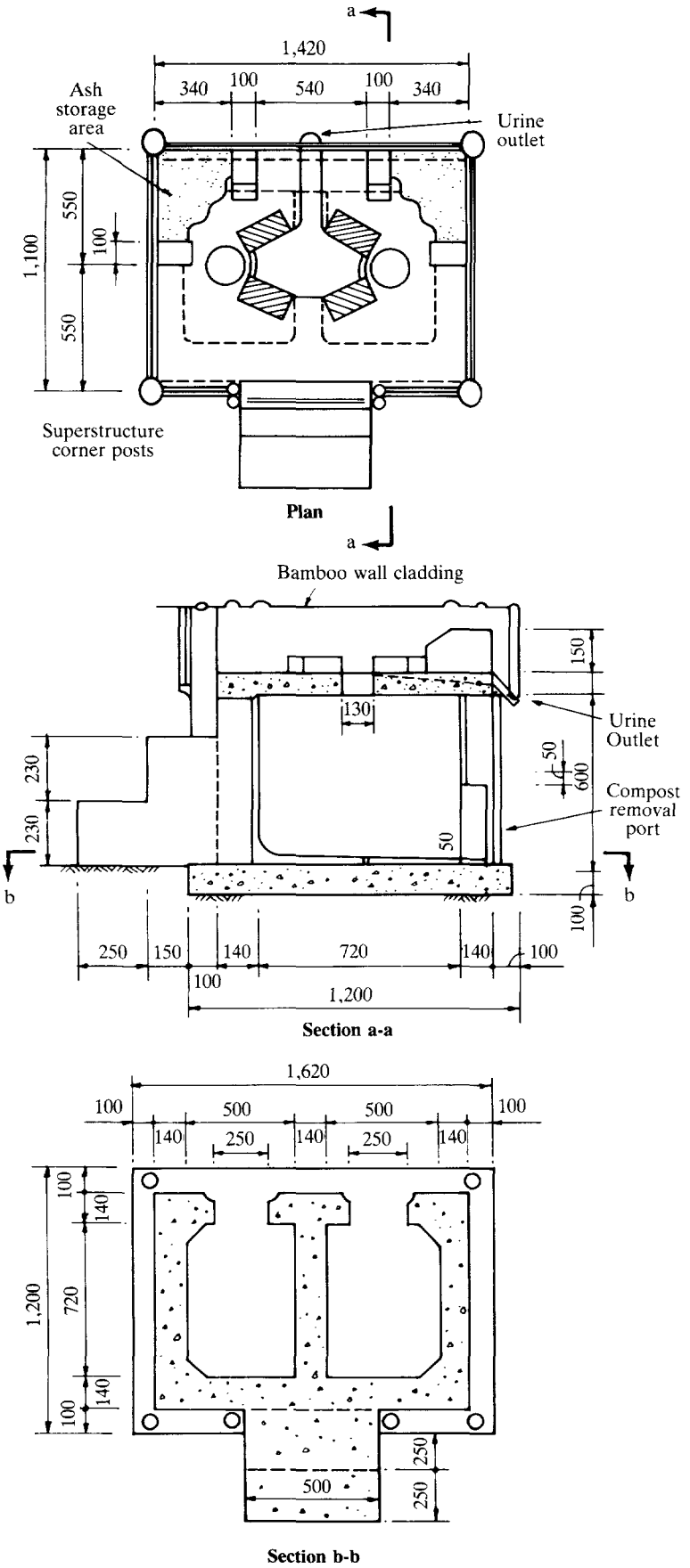



Figure 5-5 (*continued*) **Model used in Vietnam**

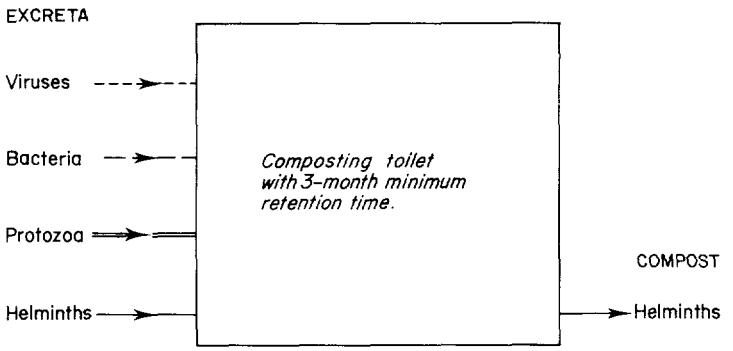



Figure 5-6. *Pathogen flow through a batch composting toilet (double-vault)*

are the correct, conservative assumptions to make where pathogen removal is the concern. Pathogen removal then depends on the retention time in the unit. There appears to be a wide variation in retention time used in both the multrum (continuous) and double-vault (batch) systems, and the pathogen removal efficiency of any given design can be estimated by consulting table 5-1. It is clear from the table that a minimum retention time of 3 months will yield a product free of all pathogens except the more persistent helminth eggs, as visualized in figure 5-6. Three possible pathogen control strategies can be adopted for compost:

- To use the compost as produced and accept the level of risk involved. This risk could be reduced to sufficiently low levels by using the compost only to prepare ground prior to planting or by not applying compost within 2 months of harvesting.
- To apply the compost only to industrial or fodder crops.
- To provide further treatment for the compost through heating it (probably impracticable) or through mixing it with an ovicide (also often impracticable).

The first of these strategies is probably the most realistic, and the quality of the product will become better as the retention time is increased beyond 3 months.

## Cartage Systems

Cartage systems include a variety of technologies by which night soil is periodically removed from containers in or near the house. One of the oldest—and, generally, least hygienic—systems is the bucket latrine. A squatting slab or seat is placed immediately above a bucket which is filled within a few days by the excreta of an average family (see figure 5-7). The bucket is positioned adjacent to an outside wall and is accessible from the street or back lane. A night soil collector ("scavenger" or "sweeper") will call regularly—preferably every day, but more typically once or twice a week—to empty the bucket.

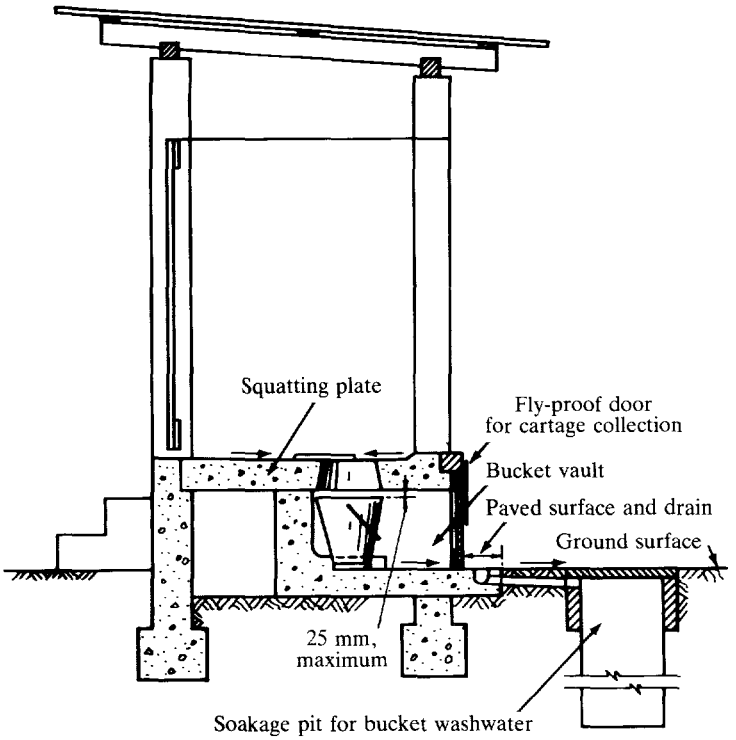
Table 5-1. *Probable pathogen content in final product of anaerobic composting toilets operating at ambient temperatures in warm climates*

| *Pathogen* | *Retention time* (months) | | | | | | |
|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 6 | 8 | 10 |
| Viruses | | | | | | | |
| Enteroviruses[a] | + | + | 0 | 0 | 0 | 0 | 0 |
| Bacteria | | | | | | | |
| Fecal coliforms | + | + | 0 | 0 | 0 | 0 | 0 |
| *Leptospira* spp. | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Salmonella* spp. | + | + | 0 | 0 | 0 | 0 | 0 |
| *Shigella* spp. | + | 0 | 0 | 0 | 0 | 0 | 0 |
| *Vibrio cholerae* | + | 0 | 0 | 0 | 0 | 0 | 0 |
| Protozoa | | | | | | | |
| *Balantidium coli* | + | 0 | 0 | 0 | 0 | 0 | 0 |
| *Entamoeba histolytica* | + | 0 | 0 | 0 | 0 | 0 | 0 |
| *Giardia lamblia* | + | 0 | 0 | 0 | 0 | 0 | 0 |
| Helminth eggs | | | | | | | |
| *Ascaris lumbricoides* | ++ | ++ | ++ | ++ | + | + | + |
| Hookworms[b] | + | + | 0 | 0 | 0 | 0 | 0 |
| *Schistosoma* spp. | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Taenia* spp. | ++ | ++ | ++ | ++ | + | + | + |
| *Trichuris trichiura* | ++ | ++ | + | + | + | + | 0 |

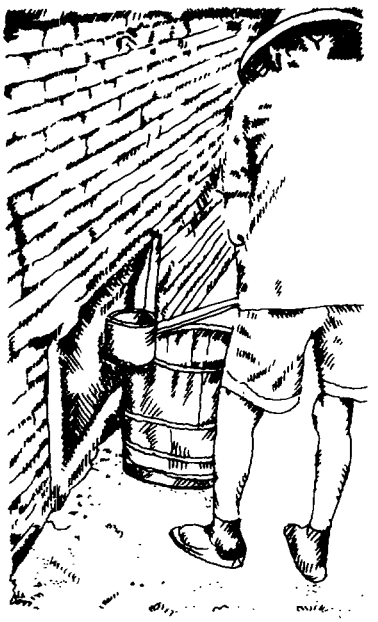
0 Complete elimination; + low concentration; ++ high concentration.

a. Includes polio-, echo-, and coxsackieviruses.

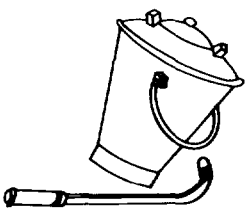
b. *Ancylostoma duodenale* and *Necator americanus.*



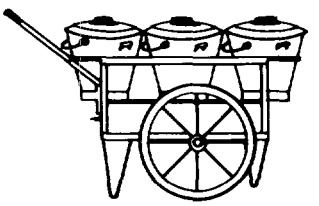

**Bucket latrine**



**Night-soil collection by dipper and bucket** (here a vault rather than a bucket is located in house)



Night-soil bucket and scraper



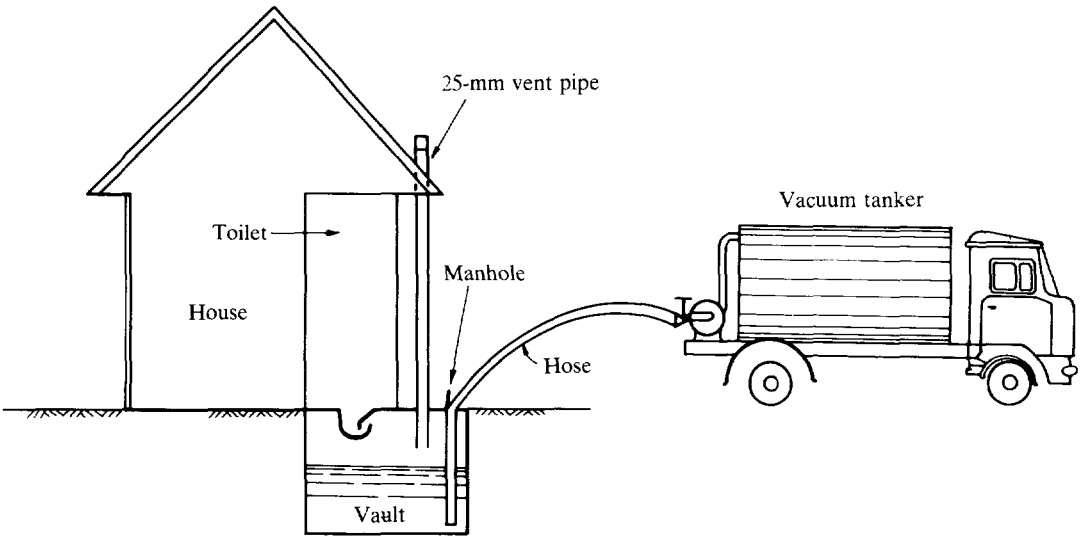
Cartage wheelbarrow for three or six buckets

Figure 5-7. *Bucket latrine and cartage.* Fly-proof doors and paved surfaces and drains are commonly missing in most existing bucket latrines. From Kalbermatten and others (1982); top left, adapted from Wagner and Lanoix (1958); top right, from a photograph courtesy of Michael G. McGarry; bottom, Department of Social Welfare, Ahmedabad, India

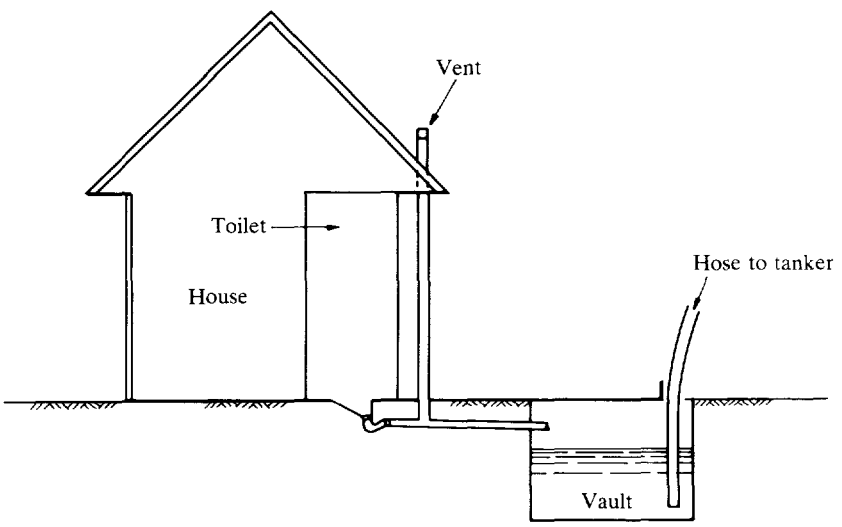
Many households in East Asia, and elsewhere, store their excreta (plus the small amounts of water used for pour flushing and anal cleansing) in sealed vaults under or beside the house (see figure 5-8) that are emptied by a vacuum truck about once every 2 weeks. This system has relatively high operating costs but may have relatively low initial costs. It is suitable for high-density urban areas where access by truck is possible and truck maintenance facilities exist. The health dimensions of a cartage system depend on the manner in which the night soil is deposited, collected, transported, treated, and reused. Each of these will be considered in turn.

### *Night soil deposition*

The two normal methods of deposit are into the bucket or vault. Both these depositories can be satisfactory if they are hygienically maintained. The bucket, a smaller vessel than the vault, is more likely to




**Vault below squatting plate**




**Offset vault**

Figure 5-8. *Alternative designs for vault toilets.* From Kalbermatten and others (1982)

overflow and to contaminate its surroundings. The bucket latrine is also almost certain to be malodorous, and this will discourage use. In contrast, the vault can be ventilated, making a hygienic and pleasant latrine.

The possibility of fly breeding depends on the frequency with which the depositories are emptied. Houseflies and blowflies require a minimum of 1 week to develop from egg to adult, and so a bucket emptied every 5 days will not permit fly breeding, provided it is well cleaned each time it is emptied. Vaults, however, are emptied less frequently, and fly breeding is a danger. Breeding can be reduced by installing a pour-flush water seal to prevent access of adult flies or by installing a vent pipe with a fly screen similar to the one

recommended above for pit latrines. A pour-flush water seal is probably the only reliable method of preventing fly breeding in vault latrines.

*Night soil collection*

Collection of night soil from vaults by vacuum trucks can be hygienic and risk free—provided that the outlet pipe from the vault is in good repair and that all fittings on the truck and suction hose are well maintained. A little spillage is probably inevitable, but it can be reduced to an acceptable minimum by good equipment and well-trained operating personnel.

By contrast, collection from bucket latrines is always messy. The worst method is to empty the buckets and immediately return them, which causes the latrine area to become progressively more fouled (with consequent risk of infection to the household, the sweeper, and passersby). Emptying the bucket, rinsing it out, and returning it is also undesirable and will probably result in the washwaters being deposited in the street. The best arrangement is to replace the bucket by another cleaned and disinfected one, with dirty buckets being returned to a central depot for cleaning and disinfection. Operation of this system is facilitated by use of a color code in which all buckets collected on Monday, for example, are red and the replacement buckets green. Such a bucket-replacement system is often not feasible on a large scale because of the difficulty of transporting large numbers of buckets. It can, however, work well in army camps, prisons, disaster relief camps, and other institutions of limited size.

It is clear that the risks from a cartage system depend greatly on the quality and regularity of the service provided. The system is sensitive to a few days' interruption in collections, whether from mechanical breakdown or absence of the sweeper.[4]

*Night soil transport*

The differences in health risks between the alternative bucket and vault-and-truck systems become obvious at the transport stage. The worst system is the one in which buckets are emptied by hand into open carts or into larger buckets, which are then carried by hand or on yokes. Under these arrangements there will always be spillage. People who come into contact with this fresh night soil risk infection from any of the nonlatent pathogens (categories I and II in table 2-2). This risk is not simply to the sweepers themselves, but also to anyone who lives on or walks, plays, or works in the streets or back lanes where the night soil has been spilled. The risk to children is obviously great because they commonly play in back lanes and alleys. The latent pathogens that develop on soil (category III—hookworms, *Ascaris*, and *Trichuris*) may well develop into their infective stages where they have been spilled in fresh night soil, and there is evidence that the cartage of night soil is partly responsible for the high levels of *Ascaris* egg contamination found in the soil of some cities. Vacuum trucks, by contrast, can transport night soil through the streets with minimal risk of spillage.

*Night soil treatment*

Night soil treatment is also discussed in conjunction with wet systems in the next chapter. Night soil can be digested and dewatered (as is sludge), it can be mixed with sewage and treated in conventional plants, or it can be sluiced into waste stabilization ponds (see chapter 6 for descriptions of these treatments). Night soil can also be treated by dry systems, such as trenching or, preferably, composting. Following adequate treatment, night soil can be used in agriculture, aquaculture or gas production (see chapter 7).

Where trenching is used, the health implications can be serious. A badly managed and inadequately controlled trenching ground will be a major health hazard to all who work on it or to those—children, for example—who may gain access. The families and close contacts of these people are also at risk. The proper management of a trenching ground is largely common sense: trenches should be at least 0.6 meters deep and should be filled with night soil to a depth of not more than 0.3 meters; they should then be rapidly covered with tamped earth, to make a small mound of earth over the trench, after which they are left for at least 2 years. Yet, however well managed the surface of a trenching ground is, the risk of groundwater pollution may always be present. This risk is minimized by careful location of the trenching ground following a hydrogeological survey. Given these limitations, in many situations the most appropriate and attractive method of night soil treatment is by mixing it with refuse and composting (see below).

*Night soil reuse*

Reuse is described in detail in chapter 7. The reuse of untreated night soil in agriculture is a widespread

4. See chapter 8, the sections "Influence of Social Structure and Organization" and "Social and Organizational Aspects of Excreta Cartage Systems."

practice, but one that is to be strongly condemned for its health hazards. There is much evidence that the use of untreated night soil on crops contributes to the transmission of infection to those working in the fields and, to a lesser (but still significant) degree, to those handling or consuming the crops. Treatment or storage of night soil should therefore always be provided prior to its reuse.

## Composting

Again it must be stressed that temperature and time are the two most important factors in the achievement of low pathogen survival in waste treatment processes. In the treatment of night soil or sludge for reuse, an almost pathogen-free product is required. This is only achieved by processes incorporating long retention times (such as ponds or protracted digestion and drying; see the next chapter), heat (such as thermophilic digestion; see the next chapter), or thermophilic composting (discussed here). The attraction of thermophilic composting is that it can yield a safe product for reuse in a relatively short time (<2 months) and that it does not require an external source of energy for heat. In addition, composting technologies are available that are relatively low cost and labor intensive. The compost produced is a useful soil conditioner and source of plant nutrients that may increasingly be in demand among poor farmers as the cost of industrially produced fertilizers rises (Food and Agriculture Organization 1975).

Composting has been thoroughly reviewed by Gotaas (1956), and more recent accounts are provided by Haug (1979); Polprasert, Wangsuphachart, and Muttamara (1980); and Shuval, Gunnerson, and Julius (1981). A wide range of fecal composting technologies are available. They all incorporate the mixing of night soil or sludge with a carbon source (such as refuse or sawdust) to achieve a C:N ratio of approximately 20–30. Moisture content (20–60 percent) must also be regulated for optimal performance, with wetting or turning (for drying) at appropriate intervals.

The most important feature of composting, from the health viewpoint, is the temperature achieved—and this depends on the oxygen content of the pile, C:N ratio, moisture content, particle size, and pH. If the process is anaerobic, temperatures will remain at (or only a little above) ambient temperature, and mesophilic microorganisms will predominate. Foul-smelling gases are usually produced, and the process of degradation proceeds slowly. If the process is aerobic, substantial heat is generated by the proliferation of thermophilic microorganisms, and degradation is more rapid and usually free of odor.

A newly erected compost pile will contain entrapped oxygen and, if the other factors mentioned above are correctly regulated, thermophilic aerobic processes will be established and the temperature at the center of the pile will rapidly rise to 55°C or above. As the available oxygen is used up, however, the process will become progressively more anaerobic and temperatures will fall. There are three methods commonly used to sustain the supply of oxygen and therefore maintain thermophilic temperatures: the pile is regularly turned, or ventilation tubes are arranged in the pile, or forced aeration is provided by blowers or suckers. In the last two cases, the pile is usually lagged to prevent heat loss. Temperatures can rise to 80°C in these well-managed, thermophilic, aerated composting systems, and it is possible to ensure that all parts of the pile spend several hours at temperatures above 60°C—of the utmost importance in curtailing pathogen survival.

### *Pathogen survival*

Pathogen survival in compost systems depends upon the time-temperature characteristics of various parts of the pile. The death curves derived for some pathogens, discussed further in Part Two, are plotted in figure 5-9. Time-temperature points above the curve for each pathogen represent certain, total destruction. It is clear that enteroviruses and *Ascaris* eggs are the most hardy, but the time-temperature combinations given in the note to figure 5-9 will ensure their destruction. If all parts of a compost pile can be brought to a time-temperature state within the "safety zone" in figure 5-9, complete pathogen destruction should be guaranteed (see figure 5-10). There are two possible exceptions. First, spore-forming bacteria—such as *Clostridium perfringens*, discussed in chapter 4—are more resistant but present little risk. Second, hepatitis A virus appears to resist rapid heating, and its ability to survive temperatures around 60°C for several hours is unknown.

Much of the literature on pathogen survival in compost, which has previously been reviewed by others (for intance, Kawata, Cramer, and Burge 1977; Krige 1964; Nell and Wiechers 1978; Reeves 1959; Shuval, Gunnerson and Julius 1981; Wiley 1962; Wiley and Westerberg 1969; WHO International Reference Centre for Wastes Disposal 1978) is reported in Part Two. This literature indicates that a well-designed system under good management produces a pathogen-free, or almost pathogen-free, compost if all sections of the pile reach the required temperature for the required

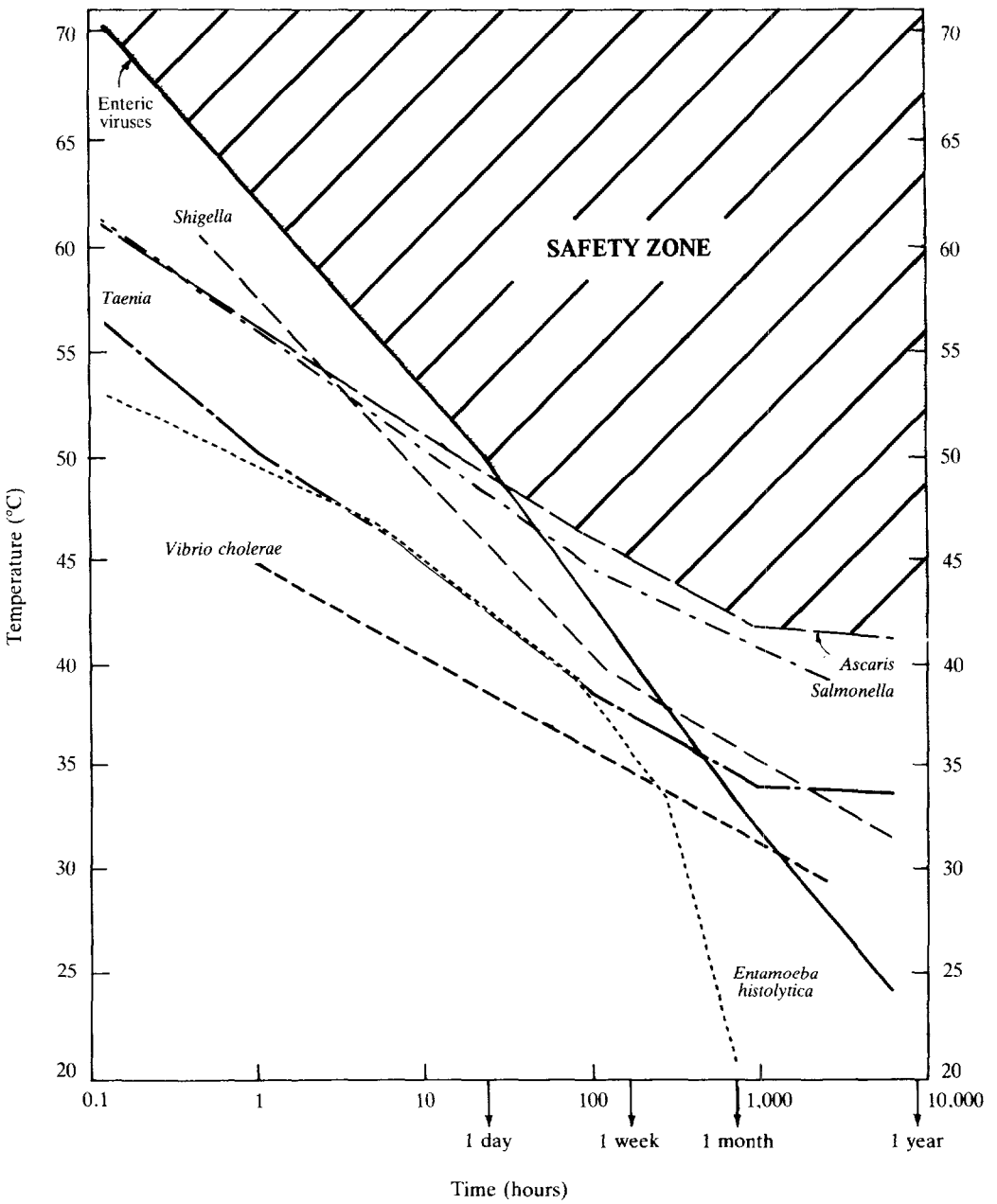



Figure 5-9. *Influence of time and temperature on selected pathogens in night soil and sludge.* The lines represent conservative upper boundaries for pathogen death—that is, estimates of the time-temperature combinations required for pathogen inactivation. A treatment process with time-temperature effects falling within the "safety zone" should be lethal to all excreted pathogens (with the possible exception of hepatitis A virus at short retention times). Indicated time temperature requirements are at least: 1 hour at $\geqslant$62°C, 1 day at $\geqslant$50°C, and 1 week at $\geqslant$46°C. For more detail on the time-temperature combinations lethal to these and other pathogens, see the graphs in chapters 9, 15–17, 20, 22, 23, 32, and 34 of Part Two (from which this composite was made)

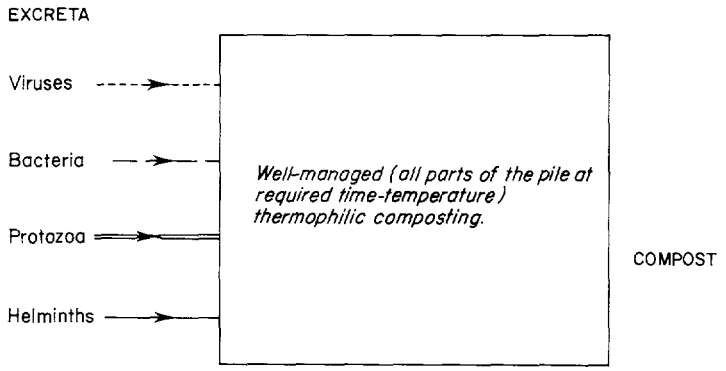



Figure 5-10. *Pathogen flow through a well-managed thermophilic composting process*

time. The organism most likely to survive this treatment is *Ascaris*, and *Ascaris* eggs may therefore be used as the indicator of successful composting.

*Fly breeding*

One of the major problems in managing composting operations is fly control. All raw materials used for composting attract flies and are good media for fly breeding. Flies can lay eggs in the material at the place of collection or during the handling of the material at the compost site. Different species predominate under different conditions, but good control measures should affect them all. Fly larvae cannot survive temperatures above 50°C, and so, as for other pathogens, the achievement of high temperatures in all parts of the pile is the essential requirement for control. Fly larvae may, however, migrate along temperature gradients to seek the cooler parts of the pile (such as the edges or the areas near ventilation shafts). These larvae may be destroyed by effective and well-controlled turning or by lagging unturned piles. The use of insecticides in compost piles is not desirable unless it has been demonstrated that these chemicals will not affect the composting process or the acceptability of the product to farmers.

Fly breeding may pose a general problem in all composting systems. The level of fly breeding provides some gauge of how successfully the pile is managed and whether it is being thoroughly heated, with minimum fly breeding an explicit goal for the management of all composting plants. It is possible to monitor the level of fly breeding by positioning flytraps at appropriate sites around the plant and recording the daily catch. This provides a continuous and immediate check of management and temperature control that is most useful to the staff in charge. Fly breeding will, of course, fluctuate markedly with the seasons, irrespective of the condition of the compost pile.

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## Note added in proof

Since this chapter was written, there have been a number of developments in the design of ventilated improved pit latrines, especially with regard to ventilation mechanisms (wind shear across the top of the vent pipe is now known to be more important than absorption of solar radiation) and the use of twin pit VIP latrines (which are permanent structures requiring each pit to be emptied in alternate years). There have also been significant developments in superstructure design, notably the spiral shape used in Zimbabwe which obviates the need for a door, while still ensuring privacy and good fly control. In rural Zimbabwe spiral latrines have been built almost entirely out of local materials and at a financial cost to the householder of only US$10. These and other developments are described in a series of working papers and technical notes prepared by the Technology Advisory Group established by the World Bank as executing agency for the United Nations Development Programme Interregional Project INT/81/047 "Development and Implementation of Low-cost Sanitation Investment Projects", they may be obtained by writing to The Project Manager, UNDP INT/81/047, Transportation and Water Department, World Bank, 1818 H St NW, Washington DC 20433, USA.

# 6

# Health Aspects of Sewage Systems

IN THIS CHAPTER the "wet" systems, which collect and treat excreta diluted by water, are considered. Not only conventional sewerage and sewage treatment systems are included, but also on-site sewage disposal methods such as septic tanks and aquaprivies. The reader wishing more technical information should refer to Kalbermatten and others (1982); Rybczynski, Polprasert and McGarry (1978); Mara (1976); Metcalf and Eddy, Inc. (1979); Okun and Ponghis (1975); and Tebbutt (1983).[1]

## Aquaprivies and Septic Tanks

Aquaprivies and septic tanks are similar systems and are thus examined together. They both incorporate a sealed settling chamber in which solids accumulate and out of which an effluent flows.

### *Technical description*

Septic tanks typically are located in the gardens of individual houses having water connections and full plumbing; they receive all wastewater from a house and have liquid retention times in the order of 1–3 days, after which the effluent normally goes to a soakaway. Aquaprivies are located directly under the toilet; they usually receive only excreta and small volumes of flushing water and have liquid retention times as high as 60 days, after which effluents flow to soakaways or into small-bore sewerage systems. In some designs aquaprivies also receive sullage, in which case retention times may decrease to a few days (depending on the volume of sullage produced). Designs for septic tanks and aquaprivies are shown in figures 6-1 and 6-2.

1. See also Part Two for a detailed review of the pathogen removal capabilities of the treatment systems examined in this chapter.

### *Pathogen survival*

Two fundamental processes affecting pathogen removal in waste treatment operate in septic tanks and aquaprivies. First, solids settle to the sludge layer at the bottom of the chamber; with them settle any bacteria or viruses adsorbed onto the solids and any helminth eggs or protozoal cysts sufficiently dense to settle. The settling action of the tanks is their chief function and their efficiency depends on retention time and design (particularly with regard to baffles or compartments designed to prevent hydraulic short-circuiting and to create quiescent conditions). Those pathogens which do not settle will remain in the liquid layers and eventually pass out of the tanks in the effluent. The degree to which their concentration decreases depends on retention time and on their reaction to the rich, anaerobic liquor in which they are held.

Generalizations about pathogen removal in aquaprivies and septic tanks are difficult to make because designs and retention times vary enormously. Moreover, as the sludge layer of a septic tank builds up, retention times decrease and the pathogen content of the effluent increases. It is common to find operating aquaprivies and septic tanks that are long overdue for desludging; in these cases any good design features and pathogen removal abilities initially present will largely have been negated by the failure to desludge at the correct, regular intervals.

Because the quality of aquaprivy effluent depends greatly on retention time, the system is sensitive to variations in hydraulic loading. If the loading rate is too low and the water level is allowed to fall below the drop pipe, the result will be the release of offensive odor and, probably, large-scale mosquito breeding. Attempts to guarantee an adequate water level by running sullage into the tanks, however, will shorten retention times and raise the pathogen content of the effluent.

There are few available data on the quality of effluent from aquaprivy installations. The literature on septic

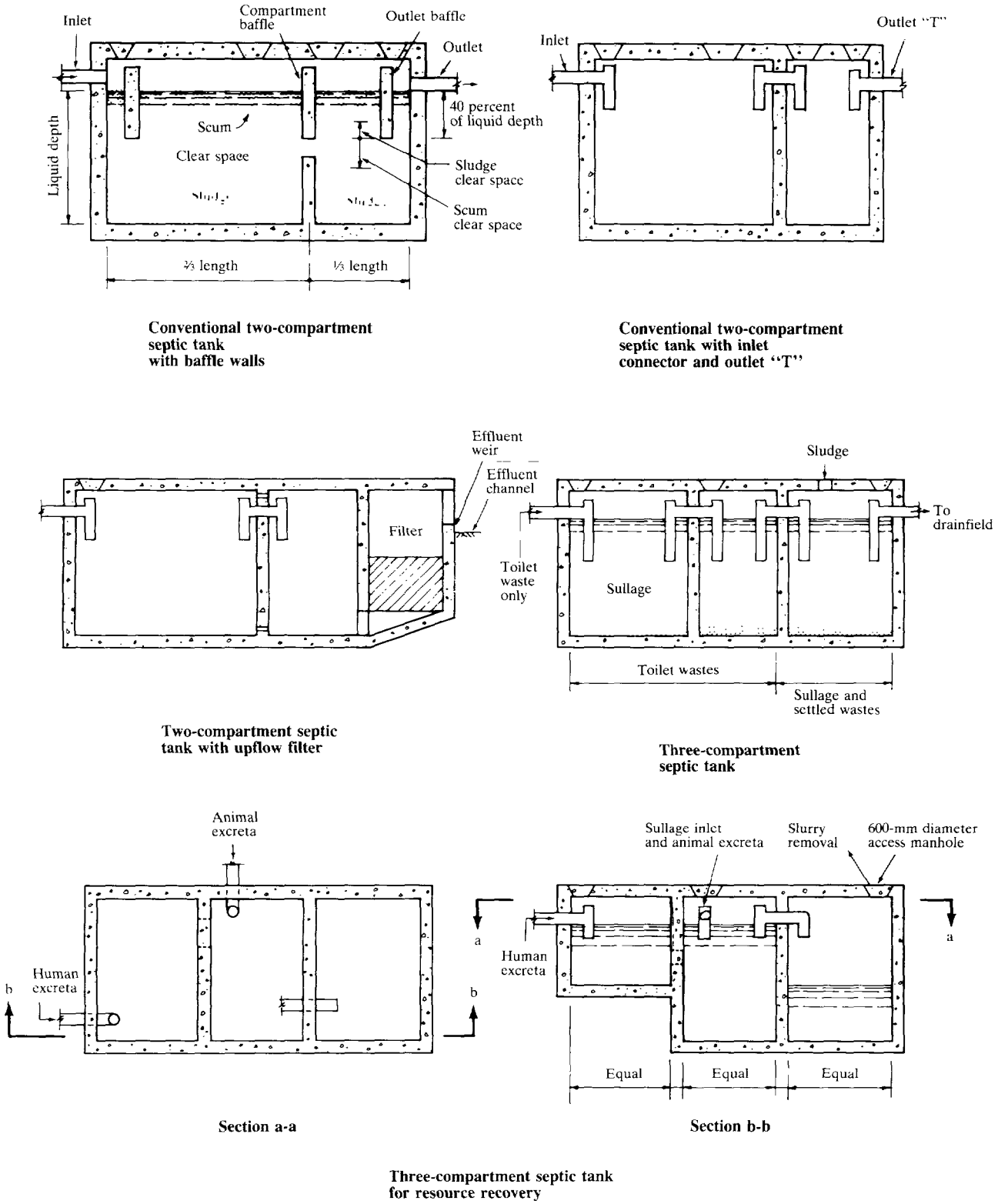
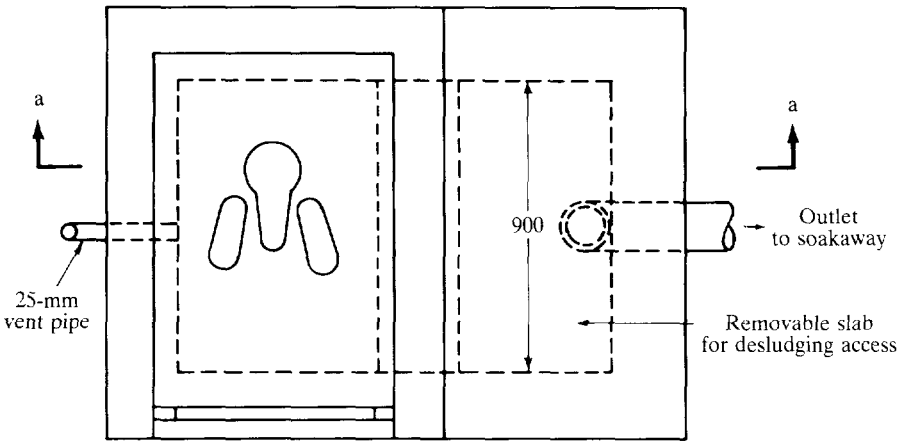
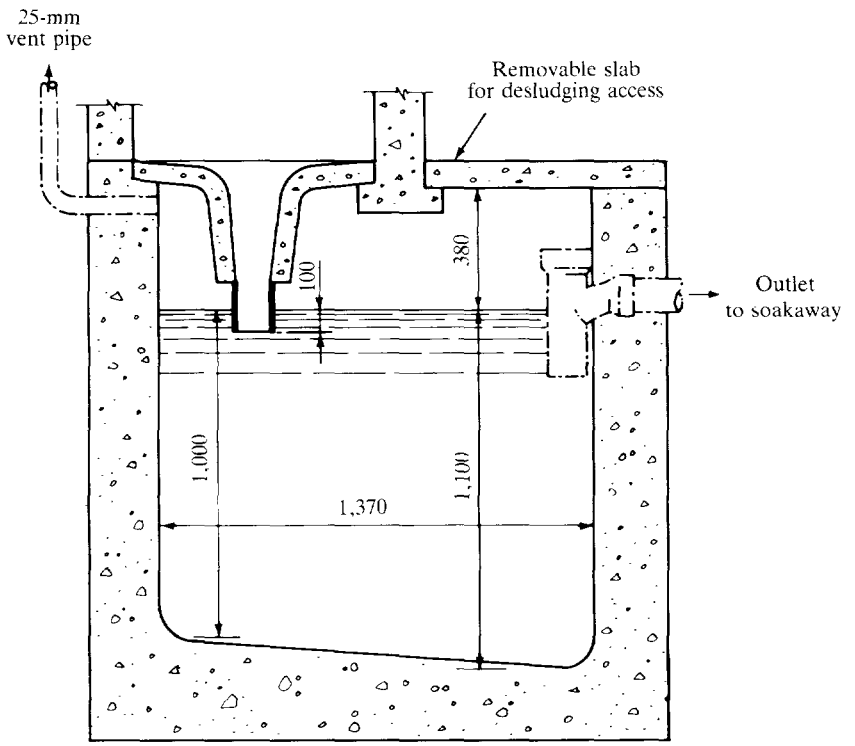



Figure 6-1. *Septic tank designs.* From Kalbermatten and others (1982)




**Plan**




**Section a-a**

Figure 6-2. *Conventional aquaprivy* (dimensions in millimeters). From Kalbermatten and others (1982); adapted from Wagner and Lanoix (1958)

tanks, reviewed in detail in Part Two, will therefore be summarized here. In a septic tank having a normal retention time (1–3 days), the effluent produced will be rich in all pathogens contained in the influent. This flow is illustrated in figure 6-3. Removal of various types of pathogens from the effluent is as follows:

| | *Reduction ($log_{10}$ unit)* |
|---|---|
| Viruses | 0–2 |
| Bacteria | 0–2 |
| Protozoa | 0–2 |
| Helminths | 0–2 |

Badly maintained and inadequately desludged tanks will have especially poor pathogen removal characteristics.

A proportion of all pathogens will settle, and fresh sludge will therefore contain significant numbers of pathogenic bacteria, viruses, protozoal cysts, and helminth eggs (figure 6-3). Whenever a septic tank is desludged, it is inevitable that some portion of the sludge will be fresh and, consequently, hazardous. Septic tank sludge should therefore be handled with great care and disposed of by burial, composting, or digestion (either aerobic or anaerobic) in the same way as any sewage sludge (and with the same effect on pathogens—see the previous chapter and the following section). A well-designed aquaprivy, with a longer retention time (>20 days) than a septic tank, may produce an effluent with only low concentrations of enteric bacteria, protozoa, or helminth eggs, and many of the viruses may settle when adsorbed onto solids. It is probable that an aquaprivy incorporating baffles and with a retention time this long will produce an effluent of substantially better quality than a normal septic tank (or, indeed, than a conventional sewage works). It must be assumed at present, however, that aquaprivy and septic tank effluents are highly pathogenic (figure 6-3). If they flow to sewers, they require treatment (probably in ponds) prior to any reuse. If they flow to soakaways, a groundwater pollution hazard may exist.[2]

## Conventional Sewage Treatment

A variety of unit processes combine to form conventional sewage treatment; commonly used combinations are shown in figure 6-4. These component processes will be discussed in turn, followed by a discussion of the effects of a complete treatment works.

### *Pretreatment and primary sedimentation*

Pretreatment by screening or comminution will have no effect on the pathogen content of sewage.

An almost universal first stage in conventional sewage treatment is the settling of suspended particles

2. See chapter 7, the section "Effluent Discharge. To groundwater."

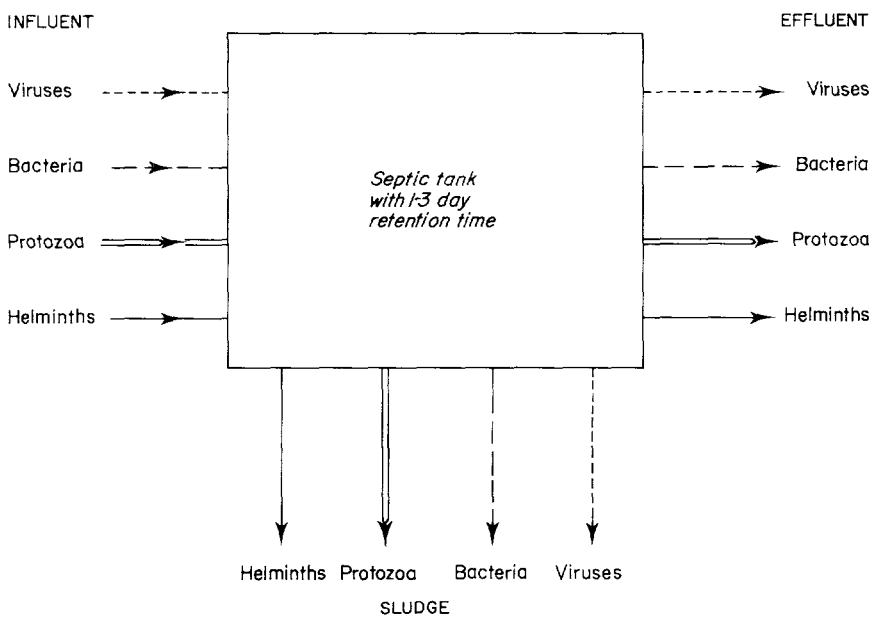



Figure 6-3. *Pathogen flow through a septic tank.*

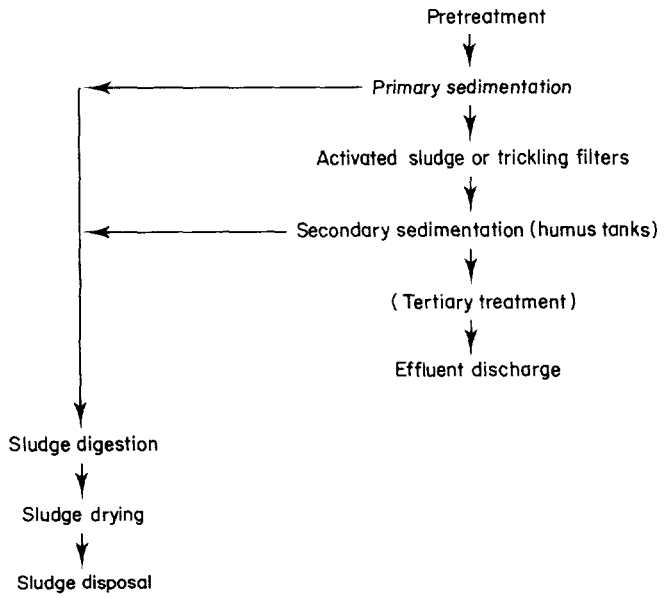



Figure 6-4. *Components of conventional sewage treatment*

in primary sedimentation tanks. A retention time in the tank of 2–6 hours is normal. A proportion of pathogens in the sewage will settle to the sludge layer either by direct sedimentation or by being adsorbed onto solids that are in the process of settling.

Many studies have found little or no virus removal by primary sedimentation, and in actual treatment works a removal rate of 50 percent seems to be a maximum. Bacterial removal by primary sedimentation may achieve 50–90 percent in 3–6 hours.

Shuval (1978) has collected data on the size and shape of eggs and cysts and has used these to compute the theoretical settling velocities shown in table 6-1. Actual settling velocities will be lower than these figures because in actual sedimentation tanks many factors hamper ideal settlement. The calculations indicate that only schistosomes, and maybe *Trichuris*, would have a reasonable degree of removal.

Studies on laboratory and full-scale primary sedimentation tanks have been done, but laboratory models always give higher removal efficiencies than actual plants because of more idealized and carefully controlled conditions.[3] *Entamoeba histolytica* cysts are reduced by 50 percent or less. Between 35 percent and 98 percent of helminth eggs settle, with 50–70 percent being the typical figure. Removal of various pathogens from the effluent is as follows:

| | *Reduction ($log_{10}$ unit)* |
|---|---|
| Viruses | 0–1 |
| Bacteria | 0–1 |
| Protozoa | 0–1 |
| Helminths | 0–2 |

Similar performance may be expected from secondary settling tanks, except that these are often designed with higher overflow rates.

Flocculation of sewage (with ferric chloride, lime, or alum) will greatly improve the settlement of cysts and eggs and perhaps of other pathogens as well.

### *Trickling filters*

Trickling filters alone do not appear to be efficient in removing viruses from sewage. Reductions reported

3. See Part Two, where the findings of such studies are reviewed.

Table 6-1. *Theoretical settling velocities of protozoal cysts and helminth eggs*

| | *Characteristics of Cysts and Eggs* | | | |
|---|---|---|---|---|
| *Pathogen* | *Size* (micrometers) | *Density* (grams per cubic centimeter) | *Assumed shape* | *Settling velocity* (meters per hour) |
| Protozoa | | | | |
| *Entamoeba hartmanni* | 5 | 1.1 | Spherical | 0.007 |
| *Entamoeba histolytica* | 20 | 1.1 | Spherical | 0.11 |
| Helminths | | | | |
| *Ascaris lumbricoides* | 55 × 40 | 1.11 | Spherical | 0.65 |
| Hookworms[a] | 60 × 40 | 1.055 | Spherical | 0.39 |
| *Schistosoma* spp. | 150 × 50 | 1.18 | Cylindrical[b] | 12.55 |
| *Taenia saginata* | 30 | 1.1 | Spherical | 0.26 |
| *Trichuris trichiura* | 50 × 22 | 1.15 | Cylindrical | 1.53 |

*Source:* Adapted from Shuval (1978).

a. *Ancylostoma duodenale* and *Necator americanus*.

b. *S. japonicum* eggs are spherical.

in the literature vary from 15 to 75 percent, with most results indicating 30–40 percent removal.[4]

Reductions in indicator bacteria in trickling-filter effluent vary between 25 and 99 percent. Typical reductions appear to be 80–95 percent. *Salmonella* reductions in the range of 71–99 percent are reported when removal by secondary sedimentation is included. The lower the loading rate on the filter, the higher the bacterial removal.

Many protozoal cysts and helminth eggs will pass through trickling filters. *Entamoeba histolytica* removal of 83–99 percent has been reported. Egg removal appears to be in the range of 20–90 percent, with higher reductions when the effect of secondary sedimentation is included.

Removal of various pathogens by trickling filters is as follows:

| | *Reduction ($log_{10}$ unit)* |
|---|---|
| Viruses | 0–1 |
| Bacteria | 0–2 |
| Protozoa | 0–2 |
| Helminths | 0–1 |

Several studies of trickling filters have examined effluent after it has passed through a secondary sedimentation or humus tank. This tank may be expected to act as a primary sedimentation tank. Reductions in helminth eggs of 94–100 percent have been reported in combinations of trickling filters and humus tanks.

### *Activated sludge*

Both laboratory data and field experience indicate that activated sludge systems are more effective in removing viruses than trickling filters.[5] Virus removals in activated sludge treatment works have been reported as up to 90 percent, although better results (up to 99 percent) are achieved in laboratory or pilot-scale models. In poorly maintained activated sludge plants, the finding of low virus removal rates is not unusual. Reductions of excreted bacteria are similar or a little better. Indicator bacteria removal rates are reported at up to 99 percent, but increases may occur. Pathogenic bacteria removal rates are commonly reported as between 60 and 99 percent at normal aeration times (6–12 hours), but may be as high as 99.9 percent following extended aeration for $\geqslant 24$ hours. The activated sludge process has little effect on protozoal cysts and helminth eggs, but substantial proportions of eggs will be removed in the secondary settling tanks. Complete activated sludge treatment plants have been reported to remove 80–100 percent of helminth eggs.

Considering the activated sludge process in isolation, pathogen removal efficiencies may be summarized as follows:

| | *Reduction ($log_{10}$ unit)* |
|---|---|
| Viruses | 0–1 |
| Bacteria | 0–2 |
| Protozoa | 0–1 |
| Helminths | 0–1 |

### *Sludge digestion*

It is clear from the discussion above that sludge from primary and secondary sedimentation tanks will contain a heavy load of excreted viruses, bacteria, protozoa, and helminth eggs. The fate of these pathogens depends on which of the many systems of sludge treatment is adopted. Anaerobic sludge digestion usually operates at one of three time-temperature combinations: 13 days at 50°C, 28 days at 32°C, or 120 days unheated. The first stage is often followed by a second-stage settling or thickening process, in which the sludge stands for a time similar to that of the first stage to allow the supernatant liquor to be drawn off.

If the digestion process is a batch process, thus ensuring that all the sludge has been at temperature $x$ for time $y$, the following pathogen removal performances at the specified time-temperature combinations may be expected:

| *Combination* | *Pathogens removed* |
|---|---|
| 13 days at 50°C | All |
| 28 days at 32°C | Viruses and protozoa: some bacteria and many helminth eggs remain |
| 120 days unheated (in warm climate) | Protozoa: persistent helminth eggs (especially *Ascaris* and *Taenia*) and a few bacteria and viruses remain |

But if the digesters are worked as a continuous process, with sludge being added and removed daily or more frequently, it is not possible to guarantee retention times, and pathogen survival will be appreciably higher than indicated above.

The expected pathogen removal characteristics of sludge treatments, as well as the effect of subsequent sludge thickening, are summarized in figures 6-5 and 6-6.[6] Protozoa will survive none of the digestion and

4. See Part Two for reports of pathogen removal by trickling filters.

5. Literature on the efficiency of activated sludge plants in removing excreted organisms is reviewed in Part Two.

6. See Part Two for a review of the literature on pathogen survival in sludge digestion.

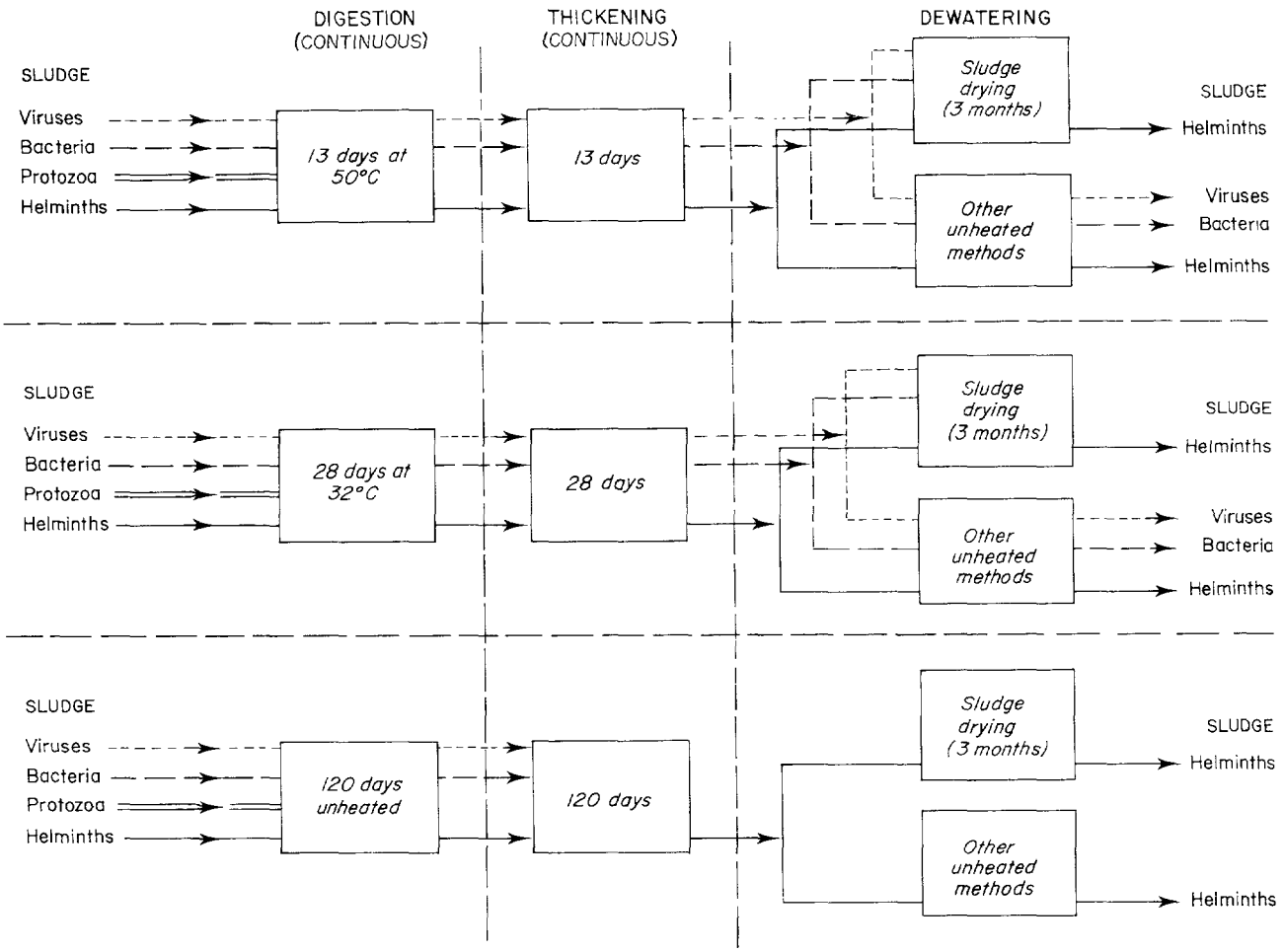



Figure 6-5. *Pathogen flow through various continuous sludge treatment processes*

thickening processes considered. Protozoal cysts are a feature of the effluents from conventional treatment plants and will not be found in treated sludges. With continuous operation, thermophilic digestion will leave small numbers of helminth eggs and excreted viruses and bacteria, whereas 120 days of unheated digestion in warm climates will leave only helminth eggs. The sole digestion process producing a thoroughly pathogen-free sludge is batch thermophilic digestion. Helminth eggs will always, and excreted viruses and bacteria will sometimes, be found in the sludges from all other digestion processes considered (Berg and Berman 1980).

### *Sludge dewatering*

Figures 6-5 and 6-6 also illustrate the effect of sludge dewatering on digested sludges. Drying sludge in open beds for 2–3 months will remove the great majority, possibly all, of excreted viruses and bacteria at warm temperatures (>20°C). Protozoal cysts will be destroyed. Only persistent helminth eggs will survive in significant numbers, especially those of *Ascaris*, *Trichuris*, and *Taenia*.[7] Other unheated dewatering processes—such as vacuum filtration, pressure filtration, and centrifugation—will have little effect on pathogen content.

### *Other sludge treatment processes*

Sludge may be composted with refuse, sawdust, woodchips, bark, straw or other material added to provide carbon, lower moisture content and improve texture. Thermophilic composting can achieve excellent pathogen removal and is discussed in the previous

7. The fate of various excreted pathogens during sludge drying is reviewed fully in Part Two.

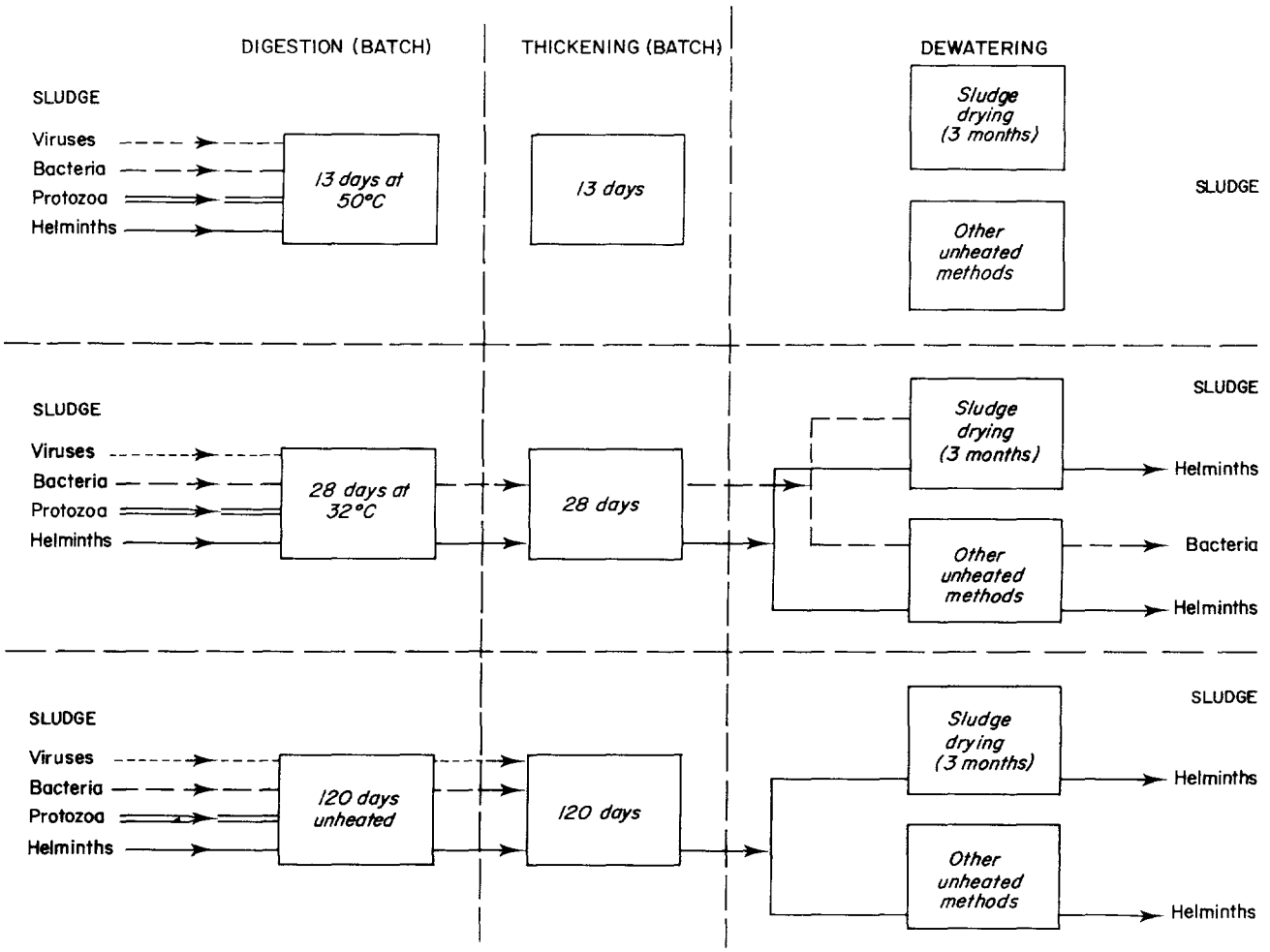



Figure 6-6. *Pathogen flow through various batch sludge treatment processes*

chapter (the section "Composting"). Several other sludge treatment processes are in use or under experimentation, but most of them are too technically complex and expensive to be appropriate for sludge treatment in developing countries. Those processes—such as wet oxidation (heating under pressure), pasteurization, incineration and pyrolysis—that involve temperatures of 80°C or above—produce a pathogen-free product (Osborn and Hattingh 1978). Sludge irradiation has attracted research interest and its effects on enteroviruses and fecal indicator bacteria are reviewed in chapters 9 and 13, respectively.

### *Complete treatment works*

The effect on pathogens of the unit processes having been examined, the effect of their combinations in conventional sewage treatment can now be discussed.[8] First considered is a treatment plant featuring trickling filters and primary and secondary sedimentation.

The effluent from such a plant will contain significant concentrations of excreted viruses, bacteria, protozoa, and helminth eggs and is unsuitable for direct reuse in agriculture (see figure 6-7). It may often be unsuitable for discharge to freshwater where such bodies of water are used without treatment for domestic water supplies by downstream populations. The minimum retention time for liquids in the total plant may be around 5 hours, and this explains why the effluent—even if it is of adequate chemical quality (for instance, the effluent might conform to the established

8. The effect of conventional treatment plants on various pathogens is reviewed fully in Part Two.

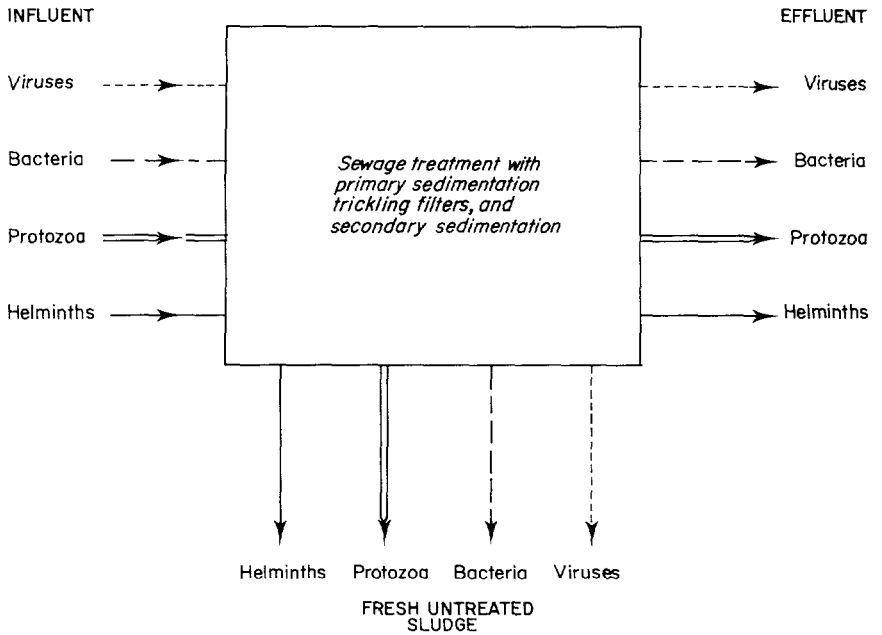



Figure 6-7. *Pathogen flow through a conventional sewage treatment plant featuring trickling filters*

physicochemical standard of <30 milligrams per liter of suspended solids and <20 milligrams per liter of standard biochemical oxygen demand, ($BOD_5$)—will be of poor microbiological quality. Effluent quality may be improved by using double filtration or recirculation, but the final effluent will still be highly pathogenic. The only way to produce an effluent of reasonably good quality from a health viewpoint is through certain tertiary treatment processes; even effluent chlorination may not be effective (see a discussion of both, below).

Effluents from activated sludge plants will be of marginally better quality than those produced by trickling filters but will still be heavily contaminated regardless of their chemical quality (see figure 6-8). The

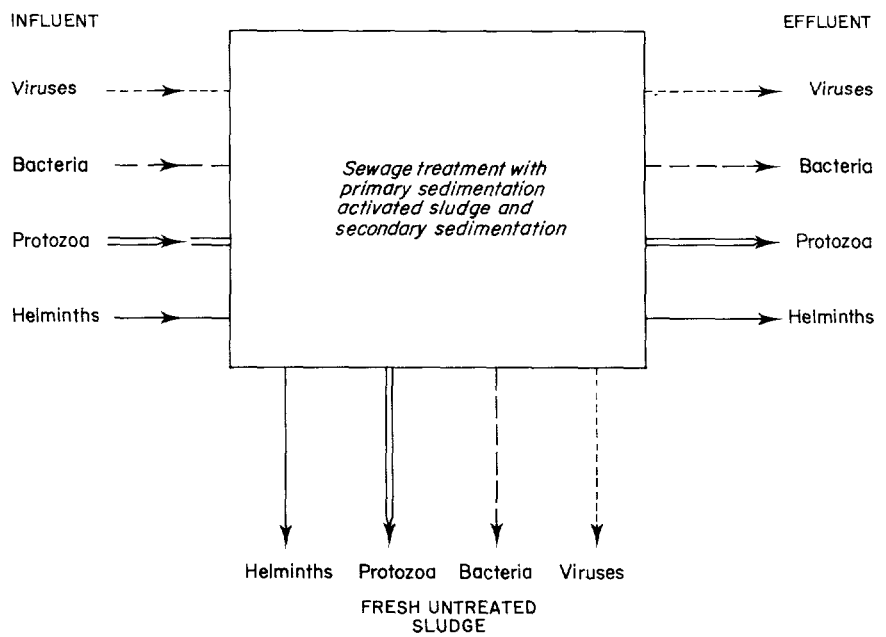



Figure 6-8. *Pathogen flow through a conventional sewage treatment plant featuring activated sludge*

minimum liquid retention time in the plant may be only 12 hours, and the final effluent will contain significant numbers of any pathogen found in the raw sewage. Tertiary treatment is indicated prior to reuse or prior to discharge into a river that downstream populations are using for water supplies.

The microbiological quality of the sludge depends on what treatment it receives. Fresh sludges from primary and secondary sedimentation tanks will contain pathogens of all kinds. Digestion at 50°C for 13 days will kill all pathogens, and digestion at 32°C for 28 days will remove protozoa and enteroviruses, provided that a batch process is used in both instances. Digestion for 120 days without heat in warm climates will remove all pathogens except helminth eggs, also only if a batch process is used. Continuous addition and removal of sludge will allow pathogens to pass through all processes. Sludge drying for at least 3 months in a warm climate is highly effective against all pathogens except helminth eggs. Other unheated dewatering techniques have little effect on the pathogenic properties of sludge.

The illustration of this somewhat complex situation in figures 6-5 and 6-6 shows that only a batch digester operated at 50°C will produce a pathogen-free sludge. Continuous digestion (as in practice) at 50°C may produce a sludge with excreted viruses and bacteria and helminth eggs if sludge drying beds are not used. All other alternatives will produce a sludge containing helminth eggs and some (such as mesophilic digestion followed by vacuum filtration) will produce a sludge with excreted viruses and bacteria as well.

The importance of temperature and time is clearly illustrated in figures 6-5 and 6-6. From a health viewpoint, the object of a sewage treatment works should be to retain all solids and liquids for the maximum time or to heat them to the maximum temperature feasible, or both. Batch processes are far more reliable in achieving this than continuous processes, and thought must be given to the design and economics of batch digesters in circumstances where sludge is to be reused in agriculture.

## Aerated Lagoons

Aerated lagoons resemble small waste stabilization ponds with floating mechanical aerators, but they are more correctly considered as a simple modification of the activated sludge process. Reference to the section on activated sludge earlier in the chapter and to the section on stabilization ponds below, along with the description here, will clarify the specifics of this system.

### *Technical description*

In aerated lagoons screened rather than settled sewage is aerated, and there is no sludge return (see figures 6-9). Retention times for domestic sewage are typically 2–6 days and lagoon depths are 2–4 meters. The effluent from the lagoon contains 200–500 milligrams per liter of suspended solids (activated sludge flocs) and therefore requires further treatment either in an ordinary secondary sedimentation tank (retention time: 2 hours, minimum) or in a settling pond (retention time: 5–10 days). The latter is more

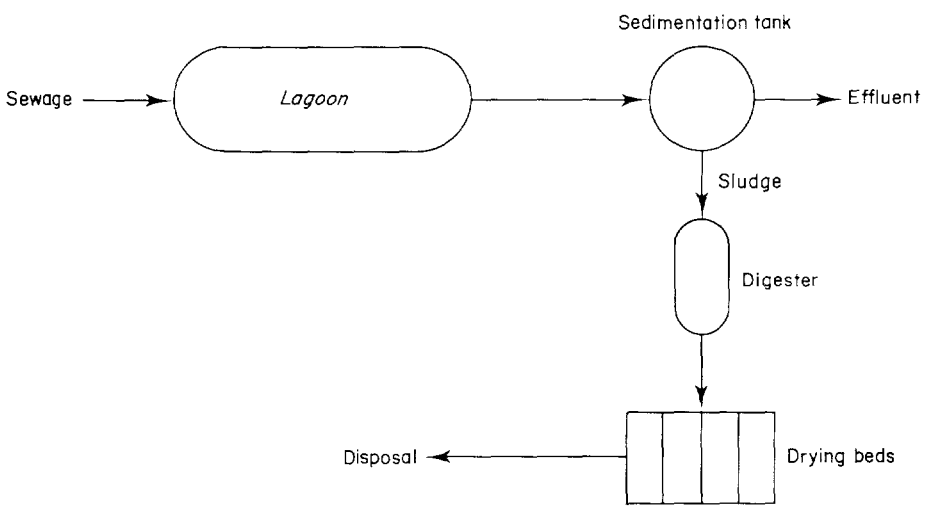



Figure 6-9. *Flow diagram for an aerated lagoon incorporating sludge digestion.* From Mara (1976)

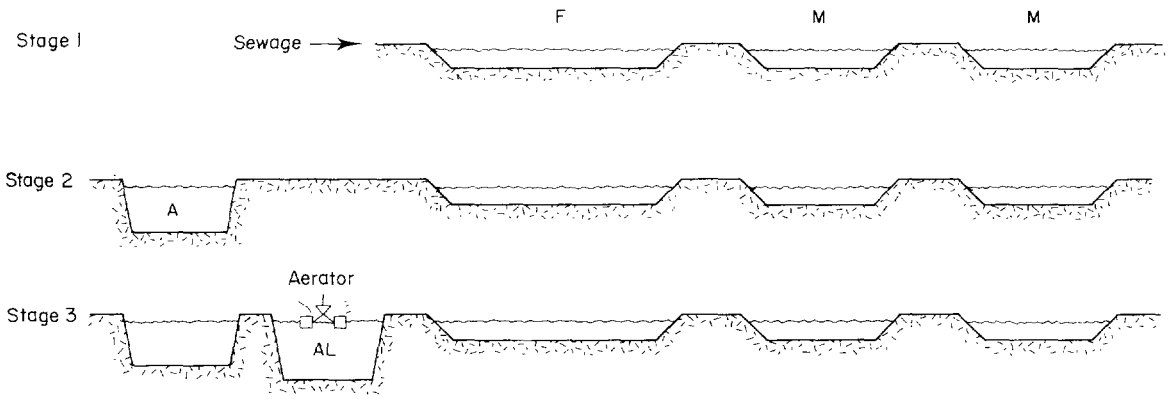



Figure 6-10. *Stages in development of a waste stabilization pond–aerated lagoon system.* F. Facultative pond; M maturation pond; A anaerobic pond; AL aerated lagoon. At stage 3 additional maturation ponds will probably be necessary. In some cases septic tanks may replace anaerobic lagoons (usually for populations below 10,000)

advantageous because it is often cheaper, easier to maintain, and more efficient in terms of removal of excreted pathogens. Aerated lagoons are often used to extend the capacity of existing waste stabilization pond systems (see figure 6-10).

*Pathogen survival*

In the aerated lagoon itself there will be incomplete removal of excreted pathogens, although as a result of the longer retention times the removal achieved is better than that obtained in the conventional activated sludge process. In the settling pond there will be complete removal of excreted protozoa and helminth eggs, but schistosome and hookworm larvae may appear in the effluent, which will also contain pathogenic bacteria and viruses. The effluent may, however, be treated in one or more maturation ponds to achieve any desired level of pathogen survival.

## Oxidation Ditches

In addition to the aerated lagoon, the oxidation ditch is another modification of the activated sludge process.

*Technical description*

Screened sewage is aerated in, and circulated around, a continuous oval ditch by one or more special aerators (called "rotors") placed across the ditch (see figure 6-11). The ditch effluent is settled in a conventional secondary sedimentation tank, and

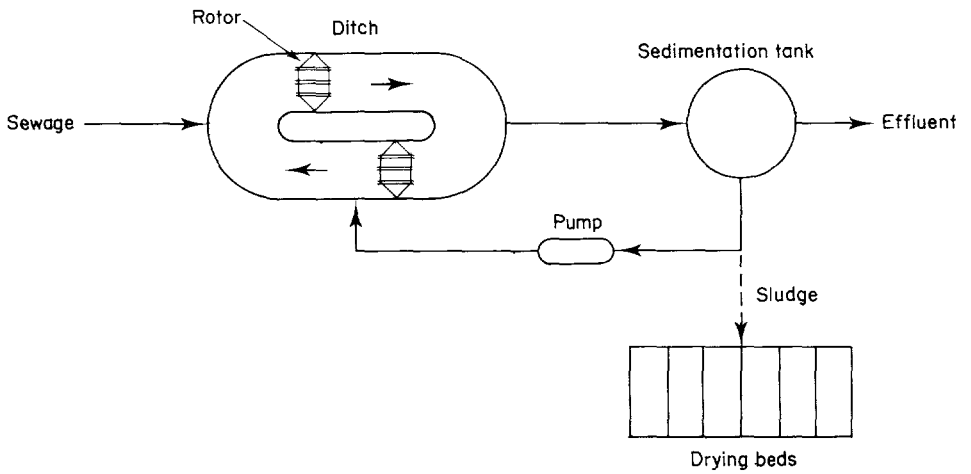



Figure 6-11. *Flow diagram for an oxidation ditch.* From Mara (1976)

almost all the sludge (>95 percent) is returned to the ditch. The small quantity of excess sludge is placed directly on sludge drying beds. The hydraulic retention times are 1–3 days in the ditch and 2 hours, minimum, in the sedimentation tank. Because a high proportion of the sludge is recycled, the mean retention time for solids is 20–30 days; as a result there is only a small production of excess sludge, which is highly mineralized and requires only dewatering on drying beds. The main engineering advantages of the process are that primary sedimentation is eliminated and that sludge production and treatment are minimal.

*Pathogen survival*

The effluent from the sedimentation tank has a pathogen content similar to that of the effluent produced by a conventional activated sludge process, although as a result of the increased retention time slightly lower survivals are achieved. The small quantity of sludge produced is similar in quality to that produced by an anaerobic digester and contains the same range of excreted pathogens.

## Tertiary Treatment

Tertiary treatment methods are increasingly used in Europe and North America to improve the quality of effluent produced by conventional treatment works. Sophisticated systems designed to reclaim effluent for potable water, such as the one used at Windhoek, Namibia (Stander and Clayton 1977), are not intended by the term, but rather those treatment processes used to upgrade the physicochemical quality of an effluent prior to discharge. Tertiary treatment processes originally were not designed primarily for pathogen removal, but some of them do have good pathogen removal characteristics.[9]

*Rapid sand filtration*

This is perhaps the most common tertiary treatment found in larger treatment works. High loading rates (200 cubic meters per square meter daily) and frequent backwashing (1–2 days) prevent the build up of much biological activity in the filter. Some viruses will be adsorbed to solids and some bacteria retained. Protozoal cysts and helminth eggs may be retained because of their size. In short, the pathogen content of the effluent may be reduced but not substantially, and probably insufficiently to justify investment in this filtration method by the health benefits it yields.

*Slow sand filtration*

This method may be used in small treatment works. The low loading rates of the filters (2–5 cubic meters per square meter daily) causes them to occupy a large land area. Substantial biological activity builds up, especially in the upper layers of the filter, and pathogen removal may be very high. Removal of 4 $\log_{10}$ units of excreted viruses and bacteria may be expected from a well-run unit, with virus removal a little higher than bacteria removal. Complete retention of protozoal cysts and helminth eggs has been recorded. Slow sand filters are therefore highly effective in removing pathogens from a secondary effluent, but their land requirement makes them suitable only for small treatment works.

*Land treatment*

Secondary effluents may be applied to land in three ways; application to land for deep percolation and groundwater recharge, application to land for collection in underdrains, and application to sloping grass plots for collection in downslope channels. The first two systems can have extremely high pathogen removal performances,[10] whereas the grass plot system is less effective because some of the effluent runs over the surface of the soil, rather than through it. There is little or no information about the application of these processes in the tropics or in developing countries. If poorly managed, they will probably lead to the creation of a foul and unsanitary bog. In addition, all land application systems pose the potential threat of groundwater contamination.[11]

*Maturation lagoons*

Conventional effluents can be upgraded in maturation lagoons. The principles involved are exactly as described for waste stabilization ponds (see the section of that title, below, and figure 6-10). If two or more maturation ponds are used, with perhaps 5 days of retention in each, total removal of protozoal cysts and helminth eggs will be achieved. High levels of virus and

9. The effect of tertiary processes in removing excreted pathogens from secondary effluents is reviewed in Part Two.

10. See chapters 9 and 13 and Uiga and Crites (1980).

11. See chapter 7, the section "Effluent Discharge. To groundwater."

bacteria removal are also effected, and a pathogen-free effluent may be produced by adding sufficient ponds.

*Other tertiary treatment processes*

Several other tertiary treatment processes are in use or under experimentation, including coagulation, carbon adsorption, irradiation, and ozonation. The effects of these on enteroviruses and fecal indicator bacteria are reviewed in chapters 9 and 13. These processes are, in general, too technically complex and costly to be appropriate for sewage treatment in developing countries.

## Effluent Chlorination

The chlorination of sewage effluents is commonplace in only a few countries (notably the USA, Canada, and Israel). Its purpose is to reduce the pathogen content of conventional effluents. As discussed earlier, it represents the borrowing from the water treatment industry of a technology that might overcome the poor pathogen removal characteristics of conventional treatment systems.[12] Effluent chlorination has a number of serious limitations, the principal one being that in some senses it does not work. At best, chlorination is complex and difficult to control. Chambers (1971) writes that

> Chlorination of wastewater effluents is a vastly more complex and unpredictable operation than chlorination of water supplies. It is extremely difficult to maintain a high, uniform, and predictable level of disinfecting efficiency in any but the most efficiently operated waste treatment plants.

For these reasons it should be rejected except where the highest levels of management and process control are guaranteed.

Chlorine has to be applied in heavy doses (10–30 milligrams per liter) to achieve coliform concentrations of less than 100 per 100 milliliters of effluent. These levels of chlorine will also kill pathogenic bacteria if the chlorine demand of the effluent is not too high, if the chlorine and the effluent are well mixed, and if adequate contact time (at least 1 hour) is allowed. But regrowth of coliforms and *Escherichia coli* following chlorination has been widely reported (for instance, Shuval 1977), and the regrowth of pathogenic bacteria has not been fully ruled out. Moreover, all bacteria in the effluent are affected by the chlorine, many of which are essential for the effluent's natural self-purification. If the effluent is discharged into a river or lake, the chlorine may adversely affect the ecology of the receiving water and hinder its natural oxidation processes. Further, the chlorine will be present in such forms as chlorinated organic compounds, which are less biodegradable than their parent compounds and are directly toxic to fish and other aquatic life (Water Research Centre 1979).

Excreted viruses are more resistant to chlorination than bacteria.[13] Chlorine doses of 30 milligrams per liter and above have been recommended; even so, complete viral removal may not be achieved (Melnick, Gerba, and Wallis 1978). It appears, at least from South African experience (Nupen, Bateman and McKenny 1974), that chlorination beyond the breakpoint—with resultant free, residual chlorine as HOCl—may be necessary to effect viral removal. Depending on the chlorine demand and pH of the effluent, breakpoint chlorination may require high doses and will always require efficient and vigilant process control.

It is most unlikely that chlorination of effluents will be effective in eliminating protozoal cysts because they are more resistant than either excreted viruses or bacteria. Most helminth eggs will be totally unharmed by effluent chlorination.

It is evident from these shortcomings that effluent chlorination may not be particularly effective in removing pathogens from conventional effluents. That chlorination may have deleterious environmental consequences—including the proliferation in water supplies of carcinogenic chlorinated hydrocarbons, which are formed by the reaction of chlorine with organic material—must also be considered (Buxton and Ross 1979; Carlo and Mettlin 1980; Deinzer, Schaumburg, and Klein 1978; Grabow 1979; Hais and Venosa 1978; Wilkins, Reiches, and Krusé 1979). Nupen and Morgan (1978) write, regarding effluent chlorination below the breakpoint in South Africa, that

> Present findings indicate that the practice not only fails to provide an effective barrier to the spread of diseases but ignores the environmental impact on receiving waters ... Under no conditions can this type of chlorination be considered as a substitute for the adequate treatment of wastes.

12. See chapter 4, the section "Objectives of Night Soil and Sewage Treatment."

13. Inactivation of enteroviruses and fecal indicator bacteria by effluent chlorination is reviewed in chapters 9 and 13.

## Waste Stabilization Ponds

Waste stabilization ponds are the most economic method of sewage treatment wherever land is available at relatively low cost (Muiga and Reid 1979). Thus, they are widely used in North America. But their principal advantage in warm climates is that they achieve low survival rates of excreted pathogens at a much lower cost than any other form of treatment, with maintenance requirements simpler by several orders of magnitude. In fact, a pond system can be designed to ensure, with a high degree of confidence, the total elimination of all excreted pathogens. This is not usually achieved in practice because the incremental benefits resulting from achieving zero survival, rather than low survival, are less than the associated incremental costs. Yet waste stabilization ponds are the best form of treatment in tropical, developing countries because they can achieve any level of pathogen removal desired. From a strictly health-directed viewpoint, the fact that ponds can do this at lowest comparable cost is an additional advantage.

### *Technical description*

Waste stabilization ponds are large, shallow ponds in which organic wastes are decomposed by micro-organisms in a combination of natural processes involving both bacteria and algae. The waste fed into a stabilization pond system can be raw sewage, aquaprivy effluent, or diluted night soil (figure 6-10). There are three kinds of ponds in common use:

- Anaerobic pretreatment ponds, which function similarly to open septic tanks; they have retention times of 1–5 days and depths of 2–4 meters.
- Facultative ponds, in which the oxygen necessary for biooxidation of the organic material is supplied principally by photosynthetic algae, which grow naturally and with great profusion in them; they have retention times of 10–40 days and depths of 1–1.5 meters.
- Maturation ponds, which receive facultative pond effluent and are responsible for the quality of the final effluent; they have retention times of 5–10 days and depths of 1–1.5 meters.

Anaerobic and facultative ponds are essentially designed for biochemical oxygen demand (BOD) removal, whereas the function of maturation ponds is the destruction or removal of excreted pathogens. These three ponds are complementary and should normally be used in conjunction with one another to form a series. Although it is all too common to find only a single facultative pond treating domestic wastes, this represents a false economy when health is considered. Maturation ponds are necessary to ensure low pathogen survivals. Good designs (see figure 6-10) incorporate a facultative pond and two or more maturation ponds; for strong wastes (biochemical oxygen demand of $>400$ milligrams per liter), the use of anaerobic ponds as pretreatment units ahead of facultative ponds is often advantageous because they minimize the land requirements of the whole pond system.

### *Pathogen survival*

Several authors have reported the fate of fecal indicator bacteria in ponds (see chapter 13).[14] High removal rates of 99.99 percent or better have been reported for series of three, four, or more ponds. Complete elimination of *Salmonella* and other enteropathogenic bacteria can be achieved in pond systems with long retention times (30–40 days), particularly if ambient temperatures are above 25°C (see chapters 13 and 15). It is known from both theoretical considerations and field experience that a series of ponds will perform far better in removing BOD and excreted bacteria than will a single pond with the same overall retention time. A series of five to seven ponds, each with a retention time of 5 days, can produce an effluent containing 100 fecal coliforms and fecal streptococci per 100 milliliters. Such an effluent can be safely used for unrestricted irrigation.

Little is known at present about the fate of viruses in ponds in warm climates or developing countries (see chapter 9). Viruses adsorb to solid particles that may settle to the sludge layer, and other biological and physical factors may be specifically virucidal; for instance an increase in pH to $\geq 9$ caused by blooms of algae. Irrespective of such effects, inactivation of excreted viruses will proceed rapidly in warm waters, and may be 1–2 log units per 5 days in ponds at $>25$°C. A pond system with an overall retention of 30 days in a warm climate should therefore achieve a reduction of excreted viruses of not less than 6 log units (99.9999 percent).

Reports on the effect of ponds on protozoal cysts and helminth eggs (see chapters 20 and 23) indicate 100 percent removal in all cases in which well-designed, multicelled ponds with a total retention time of $>20$

14. A compilation of original sources and findings on pathogens in waste stabilization ponds is given in Part Two, especially chapters 9, 13, 20, and 23.

days were investigated. Hookworm larvae may survive for up to 16 days in aerobic ponds. Because of this fact, hookworm larvae have been reported in the effluent from ponds with an overall retention time of < 10 days; they have not, however, been reported in the effluent of ponds with retention times of > 20 days. The majority of schistosome eggs in an aerobic pond will settle; in a facultative pond they will either settle or hatch into miracidia. Miracidia will either die or infect an intermediate snail host if the correct snail species is colonizing the pond (as may be the case in badly maintained and vegetated ponds). Even if cercariae emerge, they should not find a human host to invade and will die within 48 hours.

An important consideration in the design and operation of waste stabilization ponds is that they may become sites for mosquito breeding. The most common mosquitoes found breeding in ponds belong to the *Culex pipiens* complex, which favors polluted water. The distance between the town producing the sewage and the pond system treating it is usually well within the flight range of the mosquitoes, which may be as great as 10 kilometers. Any large outbreak of mosquitoes will thus be a nuisance (depending on the weather conditions at the time). Moreover, because the mosquitoes can serve as vectors for disease, it is essential to attempt to keep waste stabilization ponds free of mosquitoes. Studies on mosquitoes in ponds (reviewed in chapter 36) indicate that emerging and encroaching vegetation are important in encouraging breeding. It is easy in practice to discourage vegetation growth in ponds by making the ponds > 1 meter deep and using concrete slabs, rip-rap, or soil cement on the embankments at the surface water level. Reinforcing the pond's banks not only prevents vegetation from growing down the embankment but also halts erosion of the embankment by wave action. Any residual vegetation problem may be dealt with by the well-supervised staff of laborers who should be employed on all waste stabilization pond plants. Mosquito breeding in ponds can thus be largely circumvented by good design and good maintenance.

In summary, well-designed pond systems—incorporating a minimum of three cells, and having a minimum total retention time of 20 days (see figure 6-12)—produce an effluent that will contain only small concentrations of excreted bacteria and viruses. Excreted helminth eggs and protozoal cysts will be completely eliminated. Bacterial or viral pollution can be further reduced (or eliminated) by adding more ponds to the system. The effluent is suitable for direct reuse or discharge into receiving waters.

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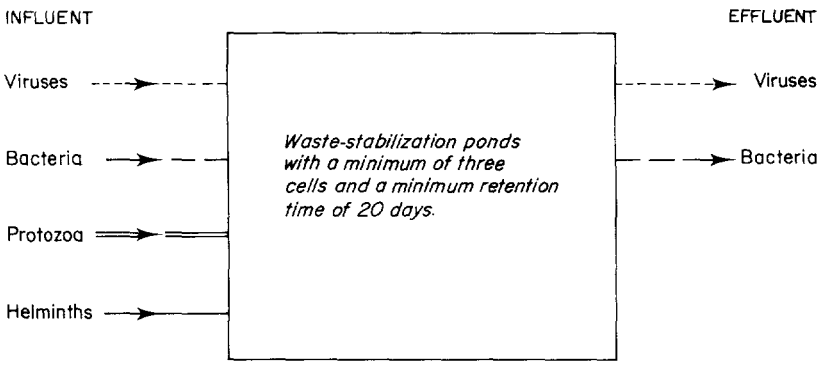


Figure 6-12. *Pathogen flow through a waste stabilization pond system*

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# 7

# Reuse of Excreta and Discharge of Effluents

HUMAN EXCRETA should be regarded as a natural resource to be conserved and reused as night soil, as sewage, or as the effluent or sludge from a sewage treatment works rather than discarded. Excreta may also be composted with organic material (such as urban refuse), which provides the carbon necessary for the composting process (discussed in chapter 5).

In all forms, excreta can provide a rich source of nitrogen and other nutrients necessary for the growth of terrestial and aquatic plants. If excreta are reused as sewage or as sewage effluent, recycling simultaneously provides valuable water, an added agricultural benefit in arid regions. When excreta are broken down anaerobically by microbial action, methane is produced and can be used as energy for heating, lighting, and other purposes.

There are, however, situations in which sewage effluents should be discharged without reuse. These may occur when there are no appropriate opportunities for reuse, when there is no local demand for the product of the reuse process, or when discharge is economically the most attractive option.

The purpose of this chapter is to explore the health implications of excreta reuse and effluent discharge. For a literature review and technical assessment of the subject, the reader should consult Rybczynski, Polprasert and McGarry (1978) and Kalbermatten and others (1982). The practices considered below are agricultural reuse, aquacultural reuse, biogas generation, and effluent discharge.

## Reuse in Agriculture

The common, and in some ways most attractive, form of waste reuse is agricultural—the application of sewage, sludge, or night soil to the land. The method of application depends in part upon the solids content of the material, and each of these fecal products may be used raw or after varying degrees of treatment. These materials, when applied to farming land, are important soil conditioners and often provide additional plant nutrients. Sewage and sewage effluents will also provide water, which may be a very scarce resource in arid areas. The health hazards associated with reuse are of two kinds: the occupational hazard to those employed to work on the land being fertilized, and the risk that contaminated products from reuse may subsequently infect humans or animals through consumption or handling. The occupational hazard is described in a separate section below, and it is only the risk from contaminated products that will be considered here. Such risk depends largely on the type of product, three categories of which are examined: foodstuffs for human consumption, foodstuffs for animal consumption, and agricultural products put to other uses.

### *Foodstuffs for human consumption*

The direct, agricultural application of raw night soil to food crops has been widely practised in many countries for centuries. There is no doubt that this reuse contributes significantly to the transmission of a broad variety of human infections. It is therefore condemned by most, if not all, health authorities and advisory agencies, and attention is now being directed to the reuse of treated effluents, sludges, and night soil to enhance agricultural production.

HEALTH ISSUES. The health problems associated with waste reuse in the production of human food may be broken down into a series of questions:

- How many pathogens and of what kind reach the field or crop?
- Are pathogens likely to survive in sufficient numbers and for sufficient time to cause subsequent infection?

- How significant is this infection route compared with all other potential infection routes?

The concern here is with the health risks to those who handle, prepare, or eat the crop after it has been harvested.

PATHOGENS REACHING THE FIELD. All pathogens in the reused waste may reach the field. Different treatment technologies will remove different pathogens to varying degrees, as discussed in chapters 5 and 6. Where effluent is used, the only treatment processes that will produce an effluent free or almost free of pathogens are waste stabilization ponds or conventional treatment followed by maturation ponds, land application, or sand filtration. Where sludge or night soil are used, the only processes which will yield a totally pathogen-free product are batch thermophilic digestion, thermophilic composting, or drying for at least 1 year.

PATHOGEN SURVIVAL. If pathogens are not removed by these processes, they will arrive at the field. Survival times of excreted pathogens in soil are summarized in table 4-5 and extensively reviewed in Part Two.

Whether or not the pathogens become attached to the surface of the crops depends upon the method of application and the crop. Crops grown on or near the ground are almost certain to become contaminated. Where wastes are sprayed or poured on fields with growing crops, contamination is also certain. Crops may be protected by subsurface irrigation, by drip or trickle irrigation where crops are not on the ground, by irrigation in furrows not immediately adjacent to the crops, or by similar techniques. Alternatively, wastes may only be applied prior to planting, or application may be discontinued one month before harvesting (with the hope that all pathogens will die before the harvest). All these methods may be effective in preventing crop contamination when the waste applied has been treated. When a waste rich in pathogens is used, however, pathogens are likely to reach the crops despite these protective strategies.

Once pathogens are on the crop, their survival is not long compared with survival in soil (table 4-6). The factors most lethal to pathogens are desiccation and direct sunlight. Survival may be expected to be much shorter in dry, sunny climates than in humid, cloudy ones.

Survival rates are quite sufficient, however, for viable pathogens (except, perhaps, protozoa) to be transported into markets, factories, and homes and subsequently to infect those who handle, process, prepare, or eat the contaminated crops. A distinction is sometimes made between crops eaten raw (tomatoes, for instance) and those normally cooked (such as cabbage). Conservative and appropriate public health policy regards these similarly because, even if a food crop is eventually cooked, those who handle and prepare it are still at risk.

PATHOGEN TRANSMISSION. The epidemiological literature reviewed in Part Two indicates that, wherever an infection is highly endemic in a community wherein poverty and squalor are also found, the introduction of the particular pathogen into the home on contaminated vegetables or other crops may have a negligible effect on transmission. In contrast, wherever an infection is not widespread in a community that has improved its standards of hygiene and housing, the introduction of contaminated crops into the home may be the major transmission route for some excreted pathogens. Thus, the significance of contaminated crops in disease transmission has mainly been emphasized in countries such as Japan, Israel, South Africa, or Germany in postwar periods, when use of sewage or excreta on crops was combined with a relatively high level of hygiene and housing.

This state of affairs can be illustrated by a hypothetical example. Imagine a town of moderately wealthy people who live in houses with water connections and flush toilets. Outside this town is a village where people are extremely poor, houses have earth floors, water is drawn from an open well, and no adequate excreta disposal system exists. The main source of income for the village is the cultivation of vegetables for sale to the town; vegetables are also used by the villagers as subsistence crops. These vegetables are fertilized by night soil collected in the village and by sewage sludge obtained free of charge from the treatment works on the outskirts of the town. The prevalence of roundworm (*Ascaris lumbricoides*) infection in the town is only 8 percent, and the principal means of entry to the home of viable *Ascaris* eggs is on the vegetables bought from the villagers. Transmission among the wealthy townsfolk does not take place because their excreta are flushed away and high standards of hygiene prevail. The prevalence of ascariasis in the village, however, is 68 percent, and transmission occurs intensively, particularly in the home. The floors and yards of the village houses are contaminated with viable eggs from the feces of infected children. Most transmission is quite unrelated to the contaminated vegetables that the villagers eat. If the supply of contaminated vegetables from the village suddenly ended, the transmission of ascariasis in the

town would be reduced very substantially, but transmission in the village would be unaffected.

Ascariasis was selected for this example because as an infection with high persistence it illustrates the point most effectively. For other pathogens, transmission may be more complex but the same principles may apply. For instance, if cholera were introduced to the area envisioned above and the crops were contaminated with *Vibrio cholerae*, the contaminated vegetables might cause an epidemic in the town and might be the major route of transmission. The village would, in all probability, experience a cholera outbreak in any case, and the vegetables might be only a slight contributing factor. An outbreak of cholera in Jerusalem in 1970 manifested epidemiological characteristics similar to this hypothetical example (Cohen and others 1971).

### *Foodstuffs for animal consumption*

A widespread use of sewage effluents, sludge, and night soil is in application to pastures or fodder crops subsequently fed to animals. In the United Kingdom, for instance, 74 percent of all sewage works sludge is disposed of on land, the remainder dumped at sea. Of the sludge disposed of on land, 21 percent is spread on grazing land, 35 percent on general arable land, 33 percent is dumped, and the remainder is used in horticulture, forestry, and land reclamation. Of the sludge applied to grazing land, 29 percent is applied raw and 71 percent is applied following digestion (Standing Committee on the Disposal of Sewage Sludge 1978). A wide variety of animal pathogens may be encountered in sewage sludge, and night soil, including:

| *Viruses causing:* | *Bacteria causing:* | *Helminths causing:* |
|---|---|---|
| Foot and mouth disease | Anthrax | Beef tapeworm infection |
| Porcine encephalomyelitis | Brucellosis | Pork tapeworm infection |
| Rabies | Leptospirosis | |
| Rinderpest | Salmonellosis | |
| Swine fever | Tuberculosis | |

HEALTH ISSUES. Despite this alarming array of infections, it is clear that, in most cases, the sewage or sludge will contain an insignificant number of these pathogens and will have a negligible effect in transmitting these diseases. There are three exceptions, however, in which the use of human wastes on pastures or fodder crops may promote the transmission of diseases of significant human or veterinary importance: beef tapeworm infection, salmonellosis, and tuberculosis.[1]

BEEF TAPEWORM INFECTION. Beef tapeworm (*Taenia saginata*) is by far the most important of these exceptions (it is described in detail in chapter 34). This helminth circulates between humans and cattle, but infection only continues when cattle eat *Taenia* eggs excreted by humans. Any treatment, disposal, or reuse technology that brings cattle into direct contact with human excreta may therefore promote the transmission of the disease unless adequate waste treatment is provided. *Taenia* eggs are hardy and are surpassed only by *Ascaris* eggs in their ability to survive outside the hos[t] (they may survive in soil or on pasture for $> 6$ months). Their removal from effluent will require either the use of waste stabilization ponds or tertiary treatment in the form of sand filtration, land application, or lagooning. Removal of *Taenia* eggs from sludge requires either a thermophilic process or retention for over a year. *T. saginata* infection in humans is not a major public health problem in most countries. The importance of controlling the infection lies in its consequences for the beef industry. Carcasses found to contain the cysts of *T. saginata* are condemned in whole or in part and the economic loss is substantial in areas of high transmission.

SALMONELLOSIS. Sewage effluents, sludges, and night soil from all large communities in both rich and poor countries will contain substantial numbers of salmonellae. Figures of $10^4$ organisms per liter of raw sewage and of raw sludge are not uncommon in Europe. These salmonellae may reach pastures or fodder crops and may infect animals and animals may subsequently infect people. The infective doses required are high, however, and *Salmonella* infections are transmitted among cattle by many ways other than contaminated fodder. There is no clear evidence that cattle grazed on pastures fertilized with wastes are at more risk from salmonelloses than other cattle (see chapter 15).

TUBERCULOSIS. Wastes from institutions treating tuberculosis patients, or from industries such as dairies and abattoirs that handle tuberculous animals, will almost certainly contain *Mycobacterium tuberculosis.* Studies in Denmark (Jensen 1954) showed tubercle

1. Pork tapeworm (*Taenia solium*) infection has been omitted from this discussion because, although the use of human wastes on fodder crops fed to pigs would undoubtedly promote the transmission of this helminth, in practice its life cycle usually depends on pigs gaining direct access to human feces, which they eagerly eat.

bacilli in the sewage produced by 5 towns with tuberculosis sanatoria. Tubercle bacilli were also demonstrated in the effluent, digested sludge, and 5-week-old dried sludge from the treatment plants of these towns.

Chlorination will remove tubercle bacilli from sewage effluent, although they are more resistant than *Escherichia coli*. In one experiment an applied dose of 10 milligrams per liter of chlorine removed tubercle bacilli from an effluent having a $BOD_5$ of 11–63 milligrams per liter (Jensen 1954). Greenberg and Kupka (1957) concluded, however, that a chlorine dose of 20 milligrams per liter and a contact time of at least 2 hours were required to remove tubercle bacilli from a well-oxidized effluent. Sludge has been recorded as containing at least $7 \times 10^5$ tubercle bacilli per gram of dry matter (Heukelekian and Albanese 1956), and 15 months on a drying bed were required to remove these in Denmark (Jensen 1954). Sludge may also be disinfected by thermophilic processes, in which tubercle bacilli are killed after 20 minutes at 66°C.

In summary, tubercle bacilli may be numerous in sewage, sludge, and night soil, and they are more persistent and resistant to disinfection than the enteric bacteria. The epidemiological significance of this is unclear. There is a case reported of tuberculosis in children who fell into a river polluted by sanatorium wastes (Jensen 1954). It remains most doubtful, however, that transmission of either human or bovine tuberculosis is significantly affected by exposure to wastes or polluted water.[2]

### *Other agricultural products*

Fecal wastes may also be used to produce crops not intended for consumption by animals or humans. Examples are tree cultivation for timber production, beautification, or the control of desertification; the irrigation of parks; and the cultivation of commercial crops such as cotton or coconuts (Sundaresan, Muthuswamy and Govindan 1978). These reuse technologies pose health hazards mainly of an occupational kind. Workers in the fields and in the factories where the crops are processed are at risk (see the next section).

One reuse system worth special mention is the practice, now widespread in the Middle East and elsewhere, of using effluents to irrigate parks, lawns, central concourses or medians of highways, and other open amenity areas. Effluents are sometimes brought in tankers from the treatment works to the city center for this purpose. Where conventional treatment works without tertiary processes are operating, this practice involves great risk to the public health and should be condemned. It is only acceptable to use the effluents from waste stabilization pond or tertiary treatment processes and, even then, very careful monitoring of the pathogen content in these effluents is required. Compared with other reuses described in this chapter, the irrigation of amenity areas is a high-risk activity.

### *Occupational hazards*

A health hazard common to all the agricultural reuse practices considered above is the risk to those who actually work in the fields. Although there is very limited epidemiological evidence to demonstrate the fact, it is likely that those who work in fields contaminated by excreted pathogens are at greater risk than others. If fieldworkers bring these infections back into their homes and subsequently infect their families, then a measurable difference in their health compared with that of nonagricultural workers and the whole community may not be apparent. Moreover, in many agricultural communities practically the whole population works in the fields at some time of the year, and so all may be exposed to the risk (although not equally).

The only sure way to protect the health of the agricultural workers is to use only wastes that are pathogen free or nearly so.[3] Once again this means only effluents that have undergone waste stabilization pond or conventional treatment followed by land application, sand filtration, or lagooning. Similarly, sludges or night soil require batch thermophilic processing, protracted drying, or storage for over 1 year.

A special problem affecting the health of agricultural workers is spray irrigation using sewage effluent. Aerosol droplets containing excreted viruses and bacteria may travel several hundred meters downwind, and excreted bacteria may be more infective (that is, have a lower infective dose) when inhaled than when

2. Tuberculosis has not been considered in Part Two. Those wishing to read further may consult Greenberg and Kupka (1957); Heukelekien and Albanese (1956); Jensen (1954); Maddock (1933); Pramer, Heukelekien and Ragotzkie (1950); Viraraghavan and Raman (1967); and Williams and Hoy (1930).

3. This recommendation, with some others in Part One of this book, concerns ideal practice and is directed to those contemplating the establishment of new waste treatment and reuse projects. For those trying to upgrade existing systems, it should be noted that any measurable reduction in the pathogen content of a waste is likely to improve public health.

otherwise ingested.[4] There is therefore some cause for concern that aerosol-disseminated excreted viruses and bacteria can infect, by inhalation, those who work in, or live near to, spray-irrigated fields. A quite different potential hazard of spray irrigation is that it often causes ponding of effluent, and this might lead to increased populations of *Culex pipiens*, and other mosquitoes breeding in dirty water (Sorber and Guter 1975).

A study in Israel (Katzenelson, Buium, and Shuval 1976) showed that people in kibbutzim (cooperative agricultural settlements) practising spray irrigation with waste stabilization pond effluent had a higher incidence of shigellosis, salmonellosis, typhoid, and infectious hepatitis than people in kibbutzim practising no form of wastewater irrigation. This could be attributed either to the agricultural use of wastewater or specifically to the spray technique promoting aerosol transmission. Subsequent debate, and new studies in Israel (Shuval and Fattal 1980), have cast doubt on these findings. There is no conclusive epidemiological evidence of adverse health effects caused by exposure to wastewater aerosols at spray irrigation sites or sewage treatment plants (Pahren and Jakubowski 1980). Such health effects, if they do exist, are less likely in dry, sunny climates than in temperate climates because viruses and bacteria in aerosols are rapidly inactivated by warm temperatures, low humidity and bright sunlight.[5]

A specific occupational hazard in the agricultural reuse of excreta is schistosomiasis. Of the various species, the one whose transmission has been related to deliberate reuse rather than incidental pollution is *Schistosoma japonicum*. The eggs survive in feces for over a week, so that when excreta are applied fresh to irrigated rice fields containing the amphibious snail hosts, the snails may become infected. This occurs in several parts of Southeast Asia and, most notably, in China. After the schistosomes have developed within the snails, larvae that can bore through the human skin are shed into the water, thus creating the occupational risk to farmers. The snail-transmitted larvae of other flatworms encyst on vegetables or in fish and crabs, so that they infect the consumer rather than the agricultural worker. Excreta can be rendered free of live schistosome eggs by suitable treatment (see chapter 32).

4. Enteroviruses and fecal indicator bacteria in aerosol droplets are discussed fully in the relevant sections of chapters 9 and 13.

5. The costs of alternative methods of reducing any health hazards associated with spray irrigation are reviewed by Young (1980).

*Pathogen control in agricultural reuse*

There is now a substantial literature on the health implications of the agricultural reuse of excreta, much of which is reviewed in Part Two. Several reviews of the topic, which some readers may find of additional value, are also available.[6]

It is clear from the discussion above that a desirable public health policy would be to require the highest quality standards for all wastes reused in agriculture. For effluents, this standard might be expressed in terms of a fecal coliform count of less than 100 per 100 milliliters (World Health Organization 1973). Such a standard, however, may tell little about the effluent content of viruses, protozoa, and helminth eggs, especially following the chlorination of the effluent, a process considerably more lethal to excreted bacteia than to other excreted pathogens (see the previous chapter). As discussed in chapter 4, *E. coli* is also an inappropriate indicator for the quality of treated sludges or night soil. For these materials the concentration of *Ascaris* eggs is a better guide to overall pathogen content.[7] Criteria for *Ascaris* have been adopted in China (McGarry and Stainforth 1978).

The imposition of stringent quality standards on effluents (for example, $<100$ fecal coliforms and fecal streptococci per 100 milliliters) restricts the range of treatment technologies considerably. It is fortunate that waste stabilization ponds are able to meet these standards and are a low-cost, appropriate form of waste treatment in hot climates (see chapters 4 and 6). Irrigation with waste stabilization pond effluent is therefore recommended.

The imposition of strict quality standards on sludges or night soil ($<10$ viable *Ascaris* eggs per 100 grams, for example) poses greater problems. Such standards can only be achieved by well-managed thermophilic digestion or composting, or by retention times of $>1$ year. A second-best choice, as indicated in figure 6-6, would be batch mesophilic digestion followed by several months on drying beds. An alternative for night soil reuse is its deposit in a facultative stabilization pond to produce a small effluent flow for irrigation or fish farming.

6. See, for instance Benarde (1973); Bryan (1977); Burge and Marsh (1978); Crook (1978); Engelbrecht (1978); Gerba, Wallis and Melnick (1975); Goldberg (1979); Hickey and Reist (1975); Pahren and others (1979); Petrik (1954); Rudolfs, Falk and Ragotzkie (1950 and 1951 *a–f*); Shuval (1977); Sorber and Guter (1975); Sorber and Sagik (1978); Wiley (1962); Wiley and Westerberg (1969) and World Health Organization (1973).

7. See Chapter 4, the section "Pathogen Indicators."

In conclusion, stringent quality standards may be set upon waste intended for agricultural reuse, and these standards can be achieved by relatively simple and low-cost technologies. Major problems in pathogen removal will only be encountered where conventional sewage treatment plants are in use. Such plants produce both an effluent and a sludge that are rich in pathogens and that require expensive additional treatment (see the previous chapter) before they can be recommended for unrestricted agricultural reuse.

## Reuse in Aquaculture

Human excreta may be reused to promote the growth of aquatic flora and fauna, a practice known as aquaculture. Three principal kinds of aquaculture are common: fish farming, algae production, and macrophyte (macroscopic aquatic plant) production.

### *Fish farming*

The raising of fish in ponds enriched with human and animal excreta has a long tradition. In China and elsewhere in Asia it has been operating continuously for centuries; it was practised in ancient Egypt and was widely used by European monasteries in the Middle Ages.

The controlled addition of wastes to ponds causes a large population of bacteria to thrive; these organisms in turn promote communities of phytoplankton (algae) and zooplankton, which then graze on the algae. With this rich food chain available, some fish, notably carp and tilapia, grow rapidly. Different fish species occupy different ecological niches—some feeding on large algae, some on small algae, some on zooplankton, some in the bottom layers, and some nearer the surface. For this reason, polyculture (the growing of several species in the same pond) is widely practiced (Muthuswamy and others 1978) because it greatly increases the total fish yield.

Fish may be grown in ponds enriched with sewage or night soil. Where sewage is used, it is usually pretreated, diluted, or both. An appropriate system is to grow fish in the maturation ponds of a chain of waste stabilization ponds (see figure 6-10); fish (except the air-breathing varieties) cannot be grown in facultative ponds because the biochemical oxygen demand (BOD) may exceed the oxygen supply, with the result that the water becomes deoxygenated, and the fish die. Night soil is commonly added to ponds either by locating latrines directly over them or by delivering night soil to them in carts or trucks.

In addition to promoting productivity, growing fish in waste-enriched ponds has other advantages. With reference to sewage treatment, nutrient removal is improved because nitrates and phosphates concentrate in the food chain and are thus removed during harvesting of the fish (Wert and Henderson 1978). The bacteriological quality of the sewage may also improve because the presence of fish appears to raise the oxygen levels and the pH (generally, to over 8.5) of the ponds, and both of these effects increase the death rate of enteric bacteria. Furthermore, there is some evidence that fish reared in sewage are less prone to disease than others.

HEALTH ISSUES. There are three distinct health problems associated with fish farming in excreta-enriched ponds:

- Passive transference of animal pathogens by fish contaminated by polluted water.
- Transmission of helminths whose life cycles involve fish as intermediate host.
- Transmission of other helminths with life cycles involving other pond fauna, such as the snail intermediate hosts of schistosomes.

The first of these problems is a cause for concern throughout the world, whereas the second and third apply only in areas where particular eating habits are found, where the helminths concerned are endemic, or both.

PASSIVE TRANSFERENCE OF EXCRETED PATHOGENS. Fish may passively carry excreted human pathogens in their intestines or on their body surfaces, and these pathogens may subsequently infect people who handle, prepare, or eat these fish. There is little risk to fish eaters except in areas where fish are eaten raw or partially cooked. Thorough cooking will destroy all excreted pathogens. The risk to those who handle or prepare the fish, however, is unaffected by the local eating habits.

Most studies on pathogen carriage by fish are related to fish caught in sewage-polluted seawater or rivers, but the principles of pathogen carriage will apply to fish farming as well. There is abundant evidence that the intestinal bacteria of humans and animals are not the normal resident flora of fish. Fish raised in contact with these bacteria may, however, acquire substantial numbers of them on their bodies and in their intestines. Fecal coliforms, fecal streptococci, and salmonellae are easily isolated from fish grown in polluted waters. A concentration effect is discernible, and concentrations of enteric bacteria in

fish intestines tend to be higher than in the water in which the fish live. There is even evidence of their ability to multiply in the intestines of some fish (see chapters 13 and 15).

It is quite possible for pathogenic bacteria carried by fish in this way to infect people. It is equally possible for the contaminated fish to infect (especially with *Salmonella*) the animals fed on fishmeal and the people who eat these animals. In practice, however, it is equally likely that the fish will become infected after harvesting and during handling, transport, and processing (Brown and Dorn 1977). The major outbreaks of salmonelloses in animals and man known to be associated with fish have been associated with contamination after harvesting. It remains quite possible for fish to carry bacterial pathogens passively from enriched ponds to humans and thereby to cause infection. The survival of excreted bacteria in fish entrails or in fish transferred to clean water is generally reported as less than 14 days (see chapter 13), although some data suggest that salmonellae may survive for 2 months in fish guts (see chapter 15).

There is little information about the carriage of nonbacterial pathogens by fish. One must assume that viruses, protozoal cysts, and helminth eggs can all be carried, and even concentrated, in or on fish and thereby infect the eaters or handlers of fish. Helminth eggs will tend to settle to the pond bottom and therefore may only be ingested by bottom-feeding fish (such as the common carp, *Cyprinus carpio*).

HELMINTHS HAVING FISH AS INTERMEDIATE HOSTS. The second, and quite distinct, health problem associated with fish farming is the transmission of worms parasitic to man that have fish as intermediate hosts. The major helminths of this kind are: *Clonorchis sinensis* (Chinese liver fluke), *Diphyllobothrium latum* (fish tapeworm), *Heterophyes heterophyes*, and *Metagonimus yokogawai*. Of these, *Heterophyes* and *Metagonimus* are of no major public health importance (they are primarily parasites of dogs and cats, and *Heterophyes* only infects fish in brackish water—see chapter 30). *Diphyllobothrium* infects pike, perch, turbot, and other fish found in lakes or rivers and is not associated with enriched ponds (see chapter 25). *Clonorchis sinensis* and the related species of cat liver flukes, *Opisthorchis viverrini* and *O. felineus*, however, are associated with excreta-fed fishponds and are intensively transmitted where fish are eaten raw or partially cooked. Infection occurs principally in China, Korea, Taiwan, Thailand, and Vietnam, and the local prevalence can reach 60 percent. Cooking of fish must be thorough to kill the encysted larvae, and most preservative and pickling techniques for fish have little effect (see chapter 24).

Where fish are grown in pretreated or presettled sewage, *Clonorchis* eggs will have settled. Transmission is therefore associated with the direct enrichment of ponds with night soil or raw sewage. *Clonorchis* eggs are fragile and die if stored for a few days in night soil. Seven-day storage of night soil prior to pond enrichment is therefore a sound strategy for the control of this infection, but it must be noted that this helminth has other vertebrate hosts (such as dogs and cats) besides man and that the control of human excreta may only partially reduce transmission.

HELMINTHS WITH OTHER AQUATIC INTERMEDIATE HOSTS. Third, it is possible that schistosomiasis transmission—through the presence of the appropriate species of snails as intermediate hosts—may occur in the ponds and infect fishermen. This requires that fresh eggs or miracidia are reaching the ponds, an event that can be prevented by using only sewage treated in stabilization ponds or stored night soil (see chapter 32).

PATHOGEN CONTROL. In summary, fish farming that uses excreta or sewage carries with it the hazards of passive carriage of a range of pathogens and of transmission of *Clonorchis* and schistosomes in some parts of the world. Pathogen control may be accomplished by:

- Enriching ponds only with treated sewage, stored night soil, or sludge
- Allowing fish to reside in clean water for several weeks prior to harvesting
- Clearing vegetation from pond banks to discourage the molluscan intermediate hosts of *Clonorchis* and the schistosomes
- Promoting good hygiene in all stages of fish handling and processing
- Discouraging the consumption of undercooked fish.

### *Algal culture*

Instead of growing fish in waste-enriched ponds with large algal populations, it is possible to harvest the algae directly. This is as yet only an experimental technique, but it may well find large-scale application in the coming decades. The advantage is that harvesting at a lower trophic level ensures far higher yields of biomass and protein. For instance, the yields to be hoped for from sewage-enriched fishponds are in the order of 10,000 kilograms per hectare yearly

(Muthuswamy and others 1978), whereas algae production in high-rate ponds may be up to 150,000 kilograms per hectare yearly. The algae are approximately 50 percent protein, and thus protein yields of 75,000 kilograms per hectare yearly are achieved. This compares favorably with protein yields from rice (56 kilograms per hectare yearly), corn (270 kilograms per hectare yearly), and soybeans (650 kilograms per hectare yearly) (McGarry 1971).

Algae may be harvested by flocculation with lime or aluminum sulfate followed by flotation (McGarry 1971), or by partial removal by microstraining. Oswald and others (1978) reported that algae are harvested from shallow ponds in the Philippines by simple sedimentation, with a production of 47,000 kilograms per hectare yearly. These various methods produce an algal paste or sludge containing 8–10 percent solids, which is then sun dried. An overview of the engineering and economic aspects of algae production in high-rate ponds is given by Lee and others (1980).

HEALTH ISSUES. High-rate ponds have a short retention time of around 1 day. Pathogen removal is therefore minimal, and the harvested algae will be rich in excreted viruses, bacteria, protozoa, and helminth eggs.

PATHOGEN CONTROL. The most effective removal process is sun drying. If the algae are dried to less than 5 percent water, pathogen removal will be complete. If not, pathogens will survive to a degree dependent upon drying time, final moisture content achieved, and sunlight intensity. There are no data on pathogen survival on drying algae, but it may be assumed that protozoa will be rapidly removed (in a few weeks) and that bacteria may be killed by algal toxins and other factors. Viruses and helminth eggs will be long-term survivors, with the latter enduring for a year or more if moisture content in the algal sludge stays above 10 percent.

The health hazards involved in the reuse of this algal product will vary. If the algae are fed to cattle, the major requirement will be the elimination of *Taenia saginata* eggs, *Salmonella* spp., and *Mycobacterium tuberculosis* (see above). If they are fed to chickens, the major requirement may be removal of *Salmonella* and *Campylobacter*. If they are fed to people (as in Japan), they will require thorough disinfection prior to packaging and marketing.

### *Macrophyte culture*

Around the world, but especially in Southeast Asia, many water plants are used for human or animal food. Some are harvested wild, and some are cultivated; they include water spinach (*Ipomoea aquatica*), water chestnut (*Eleocharis dulcis* or *E. tuberosa*), water hyacinth (*Eichhornia crassipes*), water bamboo (*Zigania* spp.), water calthrop (*Trapa* spp.), and lotus (*Nelumbo nucifera*). Some of these plants (for instance, water spinach) are intensively fertilized with human and animal wastes, whereas others are grown in water that may be incidentally contaminated (National Academy of Sciences 1976).

Attention has recently focussed upon the use of water hyacinth in waste treatment and recycling systems (Dinger 1978*a*, 1978*b*; Wolverton and MacDonald 1979). Water hyacinth removes nutrients, metals, and phenols from wastewaters (Cornwall and others 1977). The hyacinth can be harvested and used as animal feed, processed to produce fertilizer, or used to generate methane (see the section on biogas below). If water hyacinth is introduced, however, the ecological consequences of its escape into irrigation systems (it grows rapidly and can clog waterways) must also be considered. Such systems for intense recycling of wastes are usually fed by sewage but could be fed by night soil or sludge.

HEALTH ISSUES. The health hazards associated with these aquacultural practices are of three types.

First, there is the occupational risk to those who work in the water, especially where intensive use of night soil occurs. These workers may accidentally swallow pathogens or carry pathogens back to their homes on their clothing or bodies, and they may become infected percutaneously with schistosomiasis in areas where the disease is endemic and the intermediate host snails reside in the ponds or flooded fields.

Second, the harvested plants may be heavily contaminated with pathogens and may infect those who harvest, handle, prepare, or eat them. Some of these plants, such as water chestnut in China, are eaten raw.

Third, the parasitic fluke *Fasciolopsis buski* is locally important in some parts of Asia and may infect 10 million people. This worm has a life cycle that moves from man (or pig or dog) to snail to water plant to man. Animals or people become infected by eating the encysted metacercariae on water plants, especially *Eleocharis*, *Eichhornia*, *Trapa*, and *Zigania* (see chapter 28).

PATHOGEN CONTROL. Control of these health problems depends chiefly upon the treatment of night soil and other wastes prior to their discharge or prior to their use as fertilizer for aquatic plants. The health

requirements for a particular plant production system must derive from a consideration of exactly what kinds of process are being used to grow which crops, and the degree of mechanization incorporated. In addition to adequate treatment of the fecal wastes used, attention to crop harvesting and marketing techniques and education for both producers and consumers are important preventive health strategies.

## Reuse for Biogas Production

When organic wastes are digested anaerobically, a mixture of methane, carbon dioxide, and other gases is given off. This gas has become known as "biogas" and can be produced in various quantities by different technologies. In conventional sewage treatment works, anaerobic sludge digestion produces biogas that is sometimes used to heat the digesters or for some of the other energy needs of the works. Biogas production usually refers to the production of methane on a small scale by individual farmers, communes, or rural institutions in hot climates.

### *Technical description*

Biogas digesters have been installed in large numbers in China, and it is probably there that the technology has become most developed (McGarry and Stainforth 1978) (see figure 7-1).[8] Significant numbers also operate in India, Korea, and Taiwan. The biogas plants are fed with diluted animal feces, with or without human excreta and with or without vegetable refuse. The effluent slurry is reused in agriculture[9] and can also be used to enrich fishponds; the biogas is used primarily for domestic cooking and lighting. The dung from one medium-size cow or similar animal may produce around 500 liters of gas daily, and the calorific value of this gas may be around 4–5 kilocalories per liter (McGarry 1977). In contrast, human excreta only produce 30 liters of biogas per person daily. The process is very sensitive to temperature. In the mesophilic range, optimum gas production occurs at around 35°C, but in rural applications digesters are not heated (although they may be lagged or buried), and so they operate at around ambient temperatures. Gas production falls off considerably at lower temperatures and may be negligible below 15°C.

### *Pathogen control in reuse of biogas plant slurry*

The health problems associated with biogas plants come entirely from the reuse of the slurry because the gas production itself has no health implications (that is, unless the digesters explode or the gas starts a fire). Average retention times in biogas plants are commonly short (5–30 days), and the operation is usually continuous, rather than batch. Pathogen removal will therefore be considerably less effective than in conventional sludge digestion processes.[10] Protozoal cysts should not survive, but pathogenic viruses, bacteria, and helminth eggs may be expected to be present in the effluent slurry in considerable concentrations.

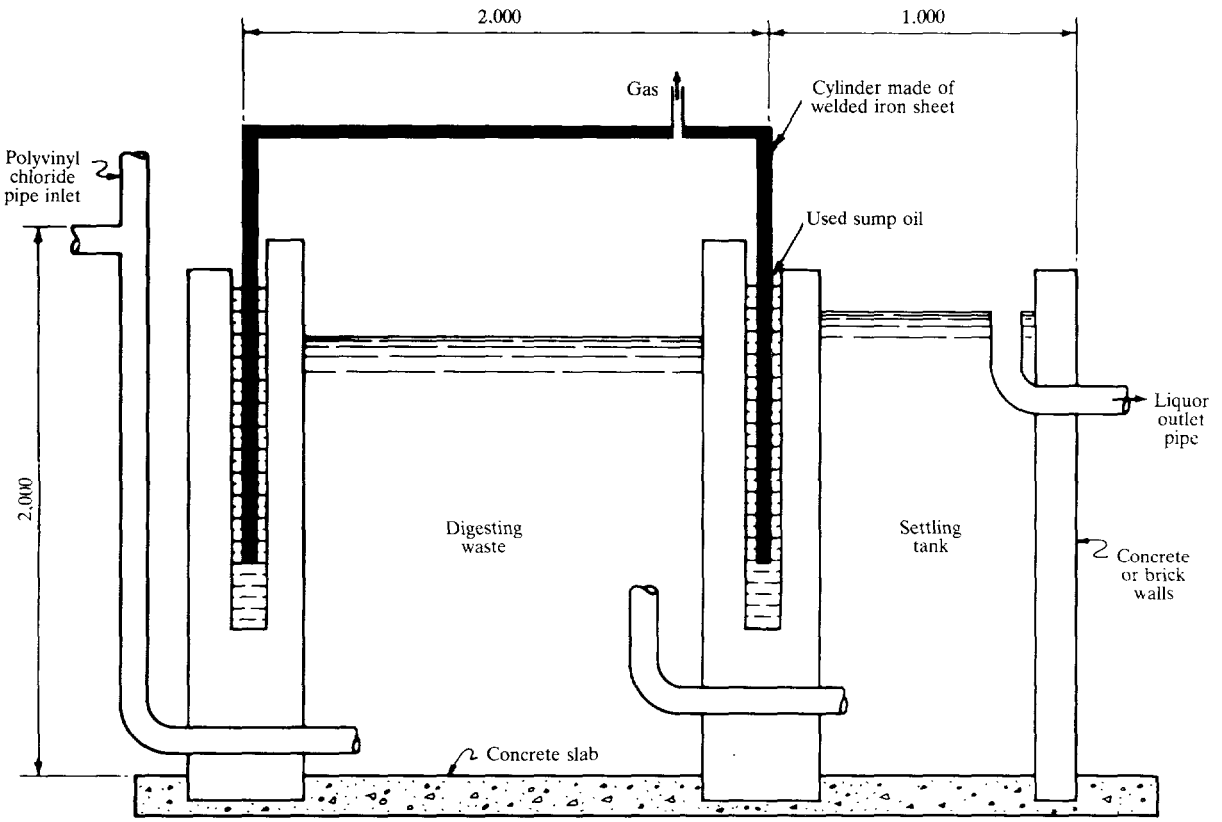
There is little information from the field on the quality of effluent from biogas plants. Data from China (McGarry and Stainforth 1978) indicate an average of 15,000 helminth eggs, 4 hookworm eggs and $8 \times 10^7$ *E. coli* per liter of biogas-plant effluent. In the same report the authors found that survival times for *Salmonella*, *Shigella*, spirochetes, schistosome eggs, and hookworm eggs in the anaerobic environment of the biogas tank are up to 44 days, 30 hours, 30 hours, 40 days, and 75 days, respectively. Therefore, for a plant with a retention time of 10–30 days, it can be expected that salmonellae, schistosomes, and hookworms will be in the effluent, but that shigellae or spirochetes will not. *Ascaris* eggs will survive considerably longer than hookworm eggs and therefore will also be present. (It is likely that the major proportion of the 15,000 helminth eggs per liter reported in the study mentioned above were *Ascaris*.) In another investigation in China (Research Institute of Military Medical Sciences 1977), inflow to a biogas plant contained $5.4 \times 10^6$ *E. coli* per liter, whereas the outflow contained $1.4 \times 10^4$ (a 99.7 percent reduction). *Shigella flexneri*, kept in conditions simulating those of a biogas tank, survived for up to 13 days, thus contradicting the study reported above.

It is clear from the data above that the effluent slurry from a biogas plant is unlikely to be significantly less pathogenic than raw sludge. Its direct reuse on crops is therefore not advised (see "Reuse in Agriculture," above). It may, however, be reused in agriculture following prolonged drying (>1 year) or after

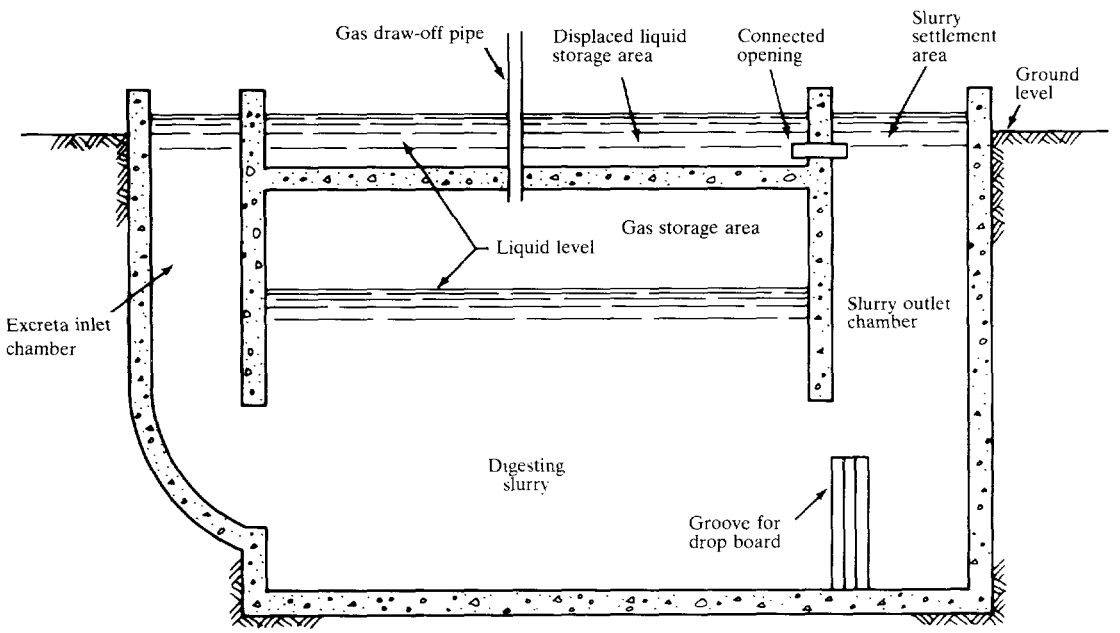
8. In addition to the two designs of biogas units shown in figure 7-1, the reader will find further details of the technology in Barnett, Pyle and Subramanian (1978); Freeman and Pyle (1977); McGarry (1977); McGarry and Stainforth (1978); Rybczynski, Polprasert and McGarry (1978); Subramanian (1977); and Van Buren (1979).

9. It is reported from China that biogas slurry increases the yields of corn and wheat by 19 percent and of vegetables by 50–60 percent (Research Institute of Military Medical Sciences 1977).

10. See the previous chapter, the section "Conventional Sewage Treatment" and figures 6-5 and 6-6.




**South Pacific design**




**Chinese design**

Figure 7-1. *Typical biogas digesters* (dimensions in millimeters). From Kalbermatten and others (1982); top, from a design by G. L. Chan

composting (see chapter 5; the area of land required for prolonged drying will be so great that composting will generally be the preferred treatment method). Biogas plant effluent may also be used to enrich fishponds. *Clonorchis sinensis* eggs will be eliminated in the plant, and the health hazard involved is the passive transmission of other pathogens by harvested fish (see "Reuse in Aquaculture," above).

## Discharge of Effluents

This chapter began with an expression of the view that sewage effluent, sludge, and night soil are important natural resources to be reused if possible. There will be occasions, however, when the most economically or environmentally appropriate solution to disposal is not reuse but the discharge of wastes to rivers, lakes, the sea, or groundwater. The health implications of each of these alternatives are discussed in this section.

### *Into rivers and lakes*

The survival of pathogens in freshwater has been examined in chapter 4. Survival times are considerable for all groups of organisms, and they increase in the following order: protozoa, bacteria, viruses, and helminths. Moreover, pathogens may travel substantial distances after being discharged into freshwater. Pathogens discharged into rivers and lakes may contaminate fish in the same way described for marine discharge (below). Where discharge is to a river, pathogens may be carried to its mouth, where they may infect shellfish.

HEALTH ISSUES. There are two overriding health problems associated with discharge of effluents into rivers or lakes: pathogens may be ingested by waterside human populations who use the river or lakewater for domestic purposes; and discharge to freshwater may promote the transmission of those parasitic worms that have aquatic intermediate hosts.

WATERBORNE PATHOGENS. People who use a polluted river or lake for their drinking water may become infected by pathogens that have previously been discharged into their water supplies. Viral, bacterial, and protozoal pathogens may all be transmitted in this way—although, where these infections are endemic in the community, the magnitude of this waterborne transmission may be minor compared with other, more direct routes (see chapters 2 and 3). Poor and seasonally arid countries are especially at risk from river or lake pollution of this kind for two reasons. First, the waterside dwellers may have no alternative, potable water supply and therefore may be compelled to use the polluted water. Second, at some period of the year river flow may be low or nonexistent, so that the discharged effluent will receive little or no dilution. These factors make it essential to guard against substantial pathogen pollution of lakes and rivers.

HELMINTHS WITH AQUATIC INTERMEDIATE HOSTS. The excreted helminths that require one or more intermediate aquatic hosts are: *Clonorchis sinensis*, *Diphyllobothrium latum*, *Fasciola hepatica*, *Fasciolopsis buski*, *Gastrodiscoides hominis*, *Heterophyes heterophyes*, *Metagonimus yokogawai*, *Paragonimus westermani*, and *Schistosoma* spp.[11]

*Fasciola* is primarily a parasite of cattle and sheep and is present in wet pastures and small streams. *Fasciolopsis* and *Gastrodiscoides* are associated with the cultivation and ingestion of water plants (see "Reuse in Aquaculture," above). *Heterophyes* and *Metagonimus* are of limited public health importance and have a very restricted geographical distribution. It is thus *Clonorchis*, *Diphyllobothrium*, *Paragonimus*, and *Schistosoma* infections that are associated primarily with discharge of effluents to rivers and lakes.

*Clonorchis sinensis*—and the related helminth species *Opisthorchis felineus* and *O. viverrini*—are transmitted from human (or dog or cat or other fish-eating mammal) to snail to fish to human, and they are particularly associated with fish farming in ponds enriched with excreta. *Diphyllobothrium latum* is transmitted from human (or dog or bear or other fish-eating mammal) to copepod (minute crustacean) to fish to human. It is especially prevalent in lakeside areas of temperate countries. *Paragonimus westermani* is transmitted from human (or many other animals) to snail to crab or crayfish to human. These three parasites all may be controlled by preventing untreated human excreta from reaching bodies of water where the intermediate hosts are found and by persuading affected communities not to eat undercooked fish, crabs, or crayfish. In the case of *Clonorchis* and *Paragonimus*, asexual multiplication takes place in the snail, so that one viable miracidium infecting a snail can ultimately infect many fish or crabs and, thus,

11. A full account of the life cycles and distribution of these parasitic worms, and guidance on the treatment processes required to remove eggs from sewage, sludge, or night soil, will be found in Part Two.

many people. The discharge of the parasite eggs in the effluent must accordingly be cut to extremely low levels if transmission is to be reduced significantly. In all cases, animals other than man act as definitive hosts, and the management of human excreta alone can never guarantee the cessation of transmission. But keeping all untreated human wastes out of rivers and lakes should have a dramatic effect on transmission in most endemic areas.

Schistosome worms are transmitted from human to snail and directly to humans through the skin. The discharge of inadeqately treated wastes to rivers and lakes is a major factor in the transmission of these important parasites. Adequate treatment of all wastes before discharge should be helpful in the control of the fecal species (*S. mansoni* and *S. japonicum*). Waste treatment will have less effect on *S. haematobium*, whose eggs are passed in the urine, because people may freely urinate near water. Once again, multiplication takes place in the snail, so that a great reduction in the number of viable eggs reaching the water is necessary before a marked reduction in transmission can be expected.

### *Into the sea*

Night soil and raw sludge are often taken out to the open sea by boat and dumped; less commonly, these wastes are dumped from the shore. Dumping of night soil or sludge in the open sea should pose no significant health problems, but dumping from the shore is so offensive that it should never be a feature of any well-designed disposal system. Only the more usual practice of discharging effluents from sewage treatment facilities into the sea near the shore is discussed below.

HEALTH ISSUES. The discharge of sewage effluent into coastal waters can create two kinds of health problem: the risk of contaminating fish or shellfish, which may subsequently be eaten, and the risk of contaminating bathing areas and beaches.

PATHOGEN SURVIVAL. Enteric viruses and bacteria discharged into seawater survive for considerably shorter periods than they do in freshwater. Coliforms in seawater undergo a 90 percent reduction in 0.6–8 hours compared with 20–100 hours in freshwater. Fecal streptococci may survive a little longer than coliforms in seawater, and salmonellae longer still. Enteroviruses survive for longer periods in seawater than excreted bacteria—90 percent reductions in 15–70 hours—but this is still considerably shorter than their survival in freshwater.[12] Excreted viruses and bacteria are eliminated very much faster in warm seawater than in cool seawater. Protozoal cysts and helminth eggs do not experience any particular lethal effects in seawater, and their survival is similar to that in freshwater (table 4-3). They do tend to settle, however, and so present little health hazard.

SEAFOOD CONTAMINATION. Fish and shellfish in polluted seawater may be contaminated by human excreted viruses and bacteria. The spread of pathogenic pollution for more than a few kilometers from sewage outfalls is not normally reported, and fish caught in the open sea are therefore found to harbor no human pathogens. Fish caught in the littoral zone, however, may well have excreted viruses and bacteria on their body surfaces and in their intestines, a hazard examined in the subsection "Fish farming," above. Excreted viruses and bacteria may survive in fish guts for a few weeks and can infect humans who handle or eat them, and may also infect animals fed on fishmeal, which may in turn infect humans. However, a more common hazard is the contamination of fish after they are caught, and most fish-associated outbreaks of salmonellosis or typhoid have been linked to this form of contamination.

A more serious problem than fish contamination is the contamination of edible shellfish (Hughes, Merson and Gangarosa 1977). Mussels and oysters are grown along coasts and in estuaries where the salt concentration is 0.8–3 percent (compared with 3.5 percent in the sea). Shellfish therefore live in the marine environment most exposed to pollution from sewage outfalls and from contaminated riverwater. Because they filter water to feed, shellfish concentrate excreted bacteria and viruses in their tissues. *Salmonella* spp. (including *S. typhi*) and enteroviruses have frequently been isolated from shellfish at concentrations well above those of surrounding seawater (see chapters 15 and 9). Outbreaks of poliomyelitis, hepatitis A, and diarrheal diseases all have been associated with the ingestion of of shellfish originating in polluted water.

Shellfish can be decontaminated by placing them in clean water. Chlorinated water that has been dechlorinated is often used (chlorinated water is ineffective because it discourages the shellfish from

12. The survival of indicator bacteria, salmonellae, and enteroviruses in seawater are reviewed in chapters 13, 15, and 9, respectively. Rapid bacterial death rates may be due to the injury of bacterial cells by seawater, such that they cannot grow on standard laboratory isolation media, rather than to actual death (Dawe and Penrose 1978). These injured bacteria can be resuscitated by special techniques, but it is not known whether they are still infective.

pumping and feeding and so will not flush out viruses or bacteria lodged in their tissues). Studies reviewed in chapters 9, 13, and 15 indicate that 2 days in disinfected water may be sufficient to cleanse shellfish of *E. coli*, but that several days are required for elimination of enteroviruses and several weeks for elimination of salmonellae. Even small numbers of pathogenic bacteria remaining in the shellfish tissues may subsequently multiply in warm conditions and infect someone eating inadequately cooked shellfish.

A related problem is that of acute gastroenteritis caused by *Vibrio parahaemolyticus*. *V. parahaemolyticus* has been reported as a cause of acute diarrhea in several countries, and it may be the single most common cause of food poisoning in Japan (Miwatani and Takeda 1976). The bacterium occurs widely in nature and is not restricted to the animal intestine. It is a halophile and has frequently been isolated from seawater, estuarine water, brackish lagoon water, marine sediments, fish, shellfish, crabs, and prawns (Baross, Liston and Morita 1978; De and others 1977; Felsenfeld and Cabirac 1977; Franca and others 1980; Sirca and others 1979; van den Broek, Mossel and Eggenkamp 1979; and Vanderzant and Nickelson 1973). Outbreaks of *V. parahaemolyticus* diarrhea in humans have usually been associated with the ingestion of inadequately cooked seafood, and the organism may also be a pathogen of marine fish and shellfish. It remains unclear to what degree disease outbreaks are associated with *V. parahaemolyticus* deriving from sewage discharges into estuaries and coastal waters, rather than to naturally occurring aquatic reservoirs of *V. parahaemolyticus*. In this connection it is noteworthy that pathogenicity in humans is particularly associated with those strains of *V. parahaemolyticus* which produce a thermostable hemolysin (the Kanagawa phenomenon). Yet, in studies in the Andaman Islands (Lall and others 1979), Britain (Ayres and Barrow 1978), India (Chatterjee and others 1978; De and others 1977; Natarajan, Abraham, and Nair 1980), and Togo (Bockemühl and Triemer 1974), from 89 to 100 percent of environmental isolates were Kanagawa negative.

RECREATIONAL HAZARDS. An active debate continues about the magnitude of the health risk associated with swimming in fecally polluted seawater and the correct approach to water quality standards and legislation (Cabelli 1979; Evison and Tosti 1980; Moore, Perin and Maiden 1979). Recent evidence from Egypt and the USA (Cabelli 1979; Cabelli and others 1979) revealed a small but measurable difference in the incidence of gastrointestinal illness between swimmers and nonswimmers at polluted beaches. The recorded risks of swimming in seawater containing $10^2$–$10^3$ fecal coliforms per 100 milliliters were an additional attack rate of one to two cases of gastrointestinal illness per 100 people in the 8–10 days following the visit to the beach. It must be kept in mind, however, that especially in developing countries the infections that may be transmitted to swimmers at polluted beaches will usually be highly endemic in the community at large (the community producing the wastes that are polluting the sea), and swimming may constitute a negligible additional risk. Set against this is the possibility that swimmers from high socioeconomic strata (who experience a low risk at home owing to adequate water supply, sanitation, and hygiene) may be exposed to a substantially increased risk of infection when they bathe in seawater polluted by the wastes of all socioeconomic strata. The same level of additional risk may apply to tourists—who are usually either local residents from upper socioeconomic groups or foreign visitors. A perceived risk to tourists, whether it is real or imaginary, may have serious economic consequences.

*To groundwater*

Effluents and liquid wastes are frequently discharged to groundwater. This usually occurs unintentionally—when soakaway effluent or pit latrine seepage percolates down to reach the water table, for instance. It can also occur through seepage losses from the base of waste stabilization ponds or, in arid areas, when effluents are discharged into low- or no-flow streams that are losing flow to the ground. In some countries in which groundwater resources are being deliberately conserved or augmented, treated effluents may be recharged to groundwater as a means of indirect recycling. [See note on page 116.]

HEALTH ISSUES. There are two central questions in considering the health implications of waste discharge to groundwater: how far do the pathogens move vertically and horizontally from the point of discharge, and for how long are they able to survive? The movement of protozoan cysts and helminth eggs can be expected to be limited because their size will cause them to be retained in soil. It is therefore viral and bacterial movement and survival that are of interest, and inadequately treated groundwater is a major cause of outbreaks of diarrhea (both viral and bacterial) and hepatitis A in some countries (Craun 1979).

PATHOGEN TRAVEL. Studies on bacterial movement through soil and rock indicate normal maximum travel distances of up to 30 meters in sand and fine soils and up to several hundred meters in gravel or fractured rock (see chapter 13). Despite their tendency to become adsorbed onto soil particles, viruses may travel through soil for longer distances than bacteria (see chapter 9). Retention does not necessarily imply inactivation. It must be noted that, when moving through soils, the great majority of bacteria and viruses are retained in the first meter and that only a small fraction is able to travel more than 10 meters.

PATHOGEN SURVIVAL. Excreted bacteria and viruses are likely to survive for longer in groundwater than in surface water (table 4-3) because groundwater is cooler, not exposed to sunlight, and has less microbial and biological activity. Bacterial survival in groundwater may be up to 5 months, with most reduction taking place in the first few days. Fecal coliforms survive longer than salmonellae and can multiply in the presence of nutrients (for example, when effluent is reaching the groundwater; see chapters 13 and 15). Virus survival may be similar or somewhat longer (see chapter 9).

PATHOGEN CONTROL. In areas where there are many pit latrines, soakaways, unlined stabilization ponds, or a recharge system, there will always be a risk of pathogenic viruses and bacteria reaching groundwater. In pit latrines, soakaways, and ponds the waste-soil interface quickly becomes clogged with solids and thus more effectively retains these microorganisms. The risks to health occur when the contaminated groundwater is used as a source of drinking water. The pathogen content of polluted groundwater will, in general, be much lower than that of surface waters in the same area. Where untreated water is being used for domestic purposes, there will therefore be a lower risk from wells than from nearby streams or ponds. Where water is chlorinated, the bacterial pathogens will be effectively destroyed.

Special vigilance is required wherever dense populations use untreated wellwater as their only domestic source and wherever there is widespread use of soakaways or pit latrines. If routine water quality monitoring demonstrates a significant pollution problem from groundwater, it is necessary to supply piped water of better quality or to change the excreta disposal method in use. The former solution will in general be less costly and more practicable than the latter.

*Nitrates from effluents*

This study concerns health problems related to biological agents contained in excreta. It would be inappropriate, however, not to mention one chemical pollution problem, the problem of nitrate accumulation, which can occur as a result of waste discharge to rivers, lakes, or groundwater. Nitrates are an end product of the oxidation of many nitrogenous compounds. Nitrate levels may be high in lakes and groundwater receiving continuing discharge of raw or treated sewage or wastewaters leaching from pit latrines, soakaways, or garbage dumps (Berwick 1979; Brooks and Cech 1979; Nicholson 1979; Schalscha and others 1979). Domestic and industrial effluents may cause high nitrate levels in receiving rivers during periods of low rainfall when the rivers are providing inadequate dilution. High nitrate levels in surface and groundwater may also derive from surface runoff water that has picked up organic material and nitrates from soil or agricultural fertilizers.

The reduction of nitrite and nitrate levels in wastewaters prior to discharge is normally achieved by the action of denitrifying bacteria under anaerobic conditions. Such a process may be included as a tertiary treatment stage in a sewage treatment plant (Anderson and Ibrahim 1978), but its expense and sophistication makes it inappropriate in most circumstances in developing countries. In hot climates, denitrification sometimes occurs in secondary sedimentation tanks that have become almost anaerobic. The consequent release of gas ($N_2$ and $N_2O$) seriously interferes with the settlement process within the tanks.

HEALTH ISSUES. Nitrate levels of over 100 milligrams per liter of $NO_3$ have been associated with clinical methemoglobinemia in bottle-fed infants (Winton, Tardiff and McCabe 1971). The nitrates are reduced to nitrites in the intestine and thence enter the bloodstream, where they oxidize hemoglobin to methemoglobin. This molecule is unable to transport oxygen; thus if too great a proportion of methemoglobin is created, serious and sometimes fatal anoxia and cyanosis may ensue. This condition is rare and apparently restricted to infants (chiefly those under 3 months of age). Exposure to excessive nitrite may also cause methemoglobinemia in animals and fish (Raju and Rao 1979).

Methemoglobinemia is particularly associated with bottle-fed infants ingesting powdered milk formula made up with high-nitrate water. However, Shuval and Gruener (1977) studied the liquid intake of 104 infants (1–5 months old) in Israel and showed that "while

during the cool months 90 percent of the total liquid intake is made up of milk, as much as 50 percent can be in the form of tap water supplements during the hottest month." Thus, in hot climates, even young breast-fed infants may be exposed to a considerable intake of high-nitrate water. The same study detected raised levels of methemoglobin in bottle-fed infants whose water supply contained 45–55 milligrams per liter of $NO_3$ (the usual accepted standard for $NO_3$ in drinking water is $<45$ milligrams per liter of nitrate or 10 milligrams per liter of nitrate-nitrogen).

Several factors operating in hot climates and developing countries may have the effect of increasing the probability of an infant's developing methemoglobinemia. These factors include high fluid intakes due to heat; the practice of boiling water of uncertain microbiological quality, which increases the nitrate concentration; and the high incidence of infant diarrheal disease, malaria, and anemia, which may all act to compound any methemoglobinemia caused by high-nitrate water. In addition, some antimalarial drugs may induce an increased level of methemoglobin in the blood. Despite these theoretical dangers, and despite the fact that some communities in some developing countries (for instance, Botswana, Senegal, and Tanzania) habitually drink wellwater with several hundred milligrams per liter of nitrate, it remains undemonstrated that any significant amount of morbidity or mortality results.

High nitrate intake (from drinking water or food) has also been implicated in adult stomach cancers in Chile, Colombia, England, Japan and elsewhere (Cuello and others 1976; Drasar and Hill 1974; Haenszel and others 1976; Hill, Hawksworth and Tattersall 1973). It is hypothesized that nitrite (produced by the bacterial reduction of ingested nitrate), secondary amines, and bacteria may come together in the stomach of individuals with gastric achlorhydria (reduced stomach acidity), or in the bladder of individuals (especially females) with bladder infection, to produce dimethylnitrosamine—a potent carcinogen of the stomach. The health hazards associated with high nitrate ingestion have been reviewed more fully elsewhere (Fraser and Chilvers 1981; National Academy of Sciences 1978).

NITRATE CONTROL. These problems may be countered by surveillance of drinking water sources to identify the communities at risk. In any one country, communities affected by highly nitrate-contaminated water will probably be small in number and restricted in geographical distribution. If the nitrate problem derives from discharges or seepages of sewage or night soil, it may be possible to prevent these occurrences. Nitrates may, however, come from many other sources, particularly agricultural runoff, and it may be more practical to provide an affected community with piped water of low nitrate content. There is currently no simple and economic method of removing nitrate from drinking water (Adam 1980; Nicolson 1979), and so nitrate reduction is normally achieved by blending high-nitrate water with waters having lower nitrate concentrations.

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## Note added in proof

Since this chapter was written a comprehensive hydrogeological review of groundwater pollution by excreta and sewage has been published: Lewis, W. J., Foster, S. S. and Drasar, B. S. (1982). *The Risk of Groundwater Pollution by On-site Sanitation in Developing Countries.* Duebendorf, Switzerland: International Reference Centre for Wastes Disposal.

# 8

# The Human Element in Sanitation Systems

DURING THE DISCUSSION of the possible health benefits from improved sanitation (chapter 3) and the specific, alternative technologies for excreta disposal (chapters 5–7), some rather demanding stipulations about the social conditions under which maximum benefits can be achieved have been made. Several beneficial effects can only be expected to occur, for instance, if latrines are properly used and maintained. Changes in the public's knowledge and practices may be required before some systems are acceptable. Good maintenance of both the private and the public components of sanitation systems is vital. That there are many calls for health education or more effective program administration is a clear indication that the social prerequisites for effective sanitation are seldom achieved in practice. Yet the diagnosis of social ills has often taken a simply deductive form: if a sanitation technology fails, the fault must lie with the users. Careful analysis of these social factors may reveal that sometimes the public's response cannot reasonably be otherwise.

A recurrent theme in this study has been that excreta disposal systems must be suited to their environmental conditions (the climate, endemic diseases, water availability, or civic wealth), many of which are clearly beyond the control of public authorities. It is too often assumed, however, that society is within governmental control and that communities should simply change to accommodate a technology that has been introduced. The task is considerably more complex. Because of their low capital cost, several of the technologies that are appropriate for the urban or rural poor make heavy demands on the users (table 3-2). They may also levy considerable demands on the limited resources of finance and trained manpower of the public bodies that have responsibilities for operation and maintenance.

It is reasonable to hope for some social change from any sanitation intervention, but program designers should ask themselves which changes are really practicable and, conversely, how far social, administrative, or political factors should be viewed as constraints on policy options. To do this requires that a planner have a good "feel" for a society and the way in which sanitation is handled within it. Two questions form the basis of the discussion in this chapter: how do social values and understandings associated with health or defecation influence sanitation programs, and what possibilities are there for controlling excreta disposal through the activities of households, community groups or urban government? The questions are interrelated because understandings and values influence institutions, and the consequences of institutional behavior in turn influence individual understandings and values.

The social, behavioral and institutional aspects of excreta disposal, and of programs designed to change excreta disposal practice, are severely neglected areas of study. Very few good field studies or thorough project evaluations have been carried out. The writings of Curtis, Goyder, Kochar, and Streefland in Pacey (1978) are of interest, as are studies on the comfort stations in Ibadan, Nigeria (Ademuwagun 1975 and Pasteur 1979). Recent studies undertaken by the World Bank have yielded insights into behavioral and institutional aspects of sanitation programs in Latin America (Elmendorf 1980) and in Africa (Feachem, Mara and Iwugo 1980) and have led to a general review of current knowledge on these matters (Elmendorf and Buckles 1980). Some valuable work in this field has been carried out not only by those investigating social aspects of sanitation programs, but also by those investigating the social aspects of the transmission of particular excreta-related pathogens. For instance, Dunn has written on the behavioral aspects of intestinal parasitism (Dunn 1972), Bancroftian and Malayan filariases (Dunn 1976) and parasitic diseases in general (Dunn 1979), and Kochar (1978, 1979) has

*Note:* The first draft of this chapter was prepared by Dr. Donald Curtis, Institute of Local Government Studies, University of Birmingham, England.

done outstanding work on hookworm transmission in West Bengal. Other recent works of interest include a comprehensive literature review of community participation and education (Van Wijk-Sijbesma 1979) and contributions by Feachem (1980) and Jackson (1979).

## Relevance of Cultural Values and Attitudes

How people react to excreta disposal schemes or arrangements depends both upon deep-rooted cultural values and upon more mundane matters of cost, convenience, and comfort. Each of these may affect user preference or acceptance, and each should be explored in every project in which the acceptability of the technology is the least bit in doubt.[1] Resistance to new latrines, for example, might be due to inadequate door catches (a mundane factor, yet indicative of a preference for individual privacy) or perhaps, for Muslims, inadvertent and inappropriate orientation of the facilities in relation to Mecca (an objection implicating values and conventions; Goyder 1978).

Cultural interpretations of excreta and defecation underlie people's responses both to the deposition technologies and to removal and reuse processes. Excreta usually have a rather special psychosocial status. In many societies excreta are only referred to in everyday speech with calculated disrespect for the values of society. Excrement is a thing apart, despised, taboo. How deeply this view prevails varies: for some peoples, excrement is simply dirty, but for others it is dangerous, a matter for personal defilement or for evil uses, to be scrupulously avoided or carefully disposed of (Curtis 1978). There are in fact many interpretations of the significance of excreta besides that of modern science, with its concern for the pathogens that excreta contain.

These culturally relative interpretations are reflected in the principles and practices of personal hygiene found around the world. Many hygienic practices have little to do with pathogen avoidance (for instance, the doctoring of a house against witchcraft), and many substances that are of little interest to modern science (such as fingernails or hair clippings) may be regarded as dangerous. Yet in most cases there is a large element of common ground—if not in interpretation, then at least in practice—between science and these other beliefs. The ancient Israelites, for example, were instructed to take a stick with them on their early morning journeys from the camp to the bush, the stick to be used for burying their feces. This is an effective sanitary prescription in the modern sense, but it is clear from the context that the instruction had more to do with the ritual cleanliness of warriors before battle than with disease transmission as such (Deuteronomy 23:12).

Mary Douglas, seeking an explanation of the universal existence of taboos, suggests that those things become taboo which are difficult to classify culturally (Douglas 1966). A corollary to this idea is that most societies prefer to maintain a clear distinction between man and animal: man is the thinker, tool user, made in the image of divinity, and so on, whereas animals are instinctive, confined to their creature strengths, and of a lower order of existence. But this distinction is difficult to maintain, particularly in relation to bodily functions. Defecation and excreta are taboo because they reveal to man an aspect of his animal existence that he would prefer to forget. This anthropological observation may have sufficiently widespead relevance to explain why man seeks privacy to defecate, defecation is confined to the bush, and excreta are, if possible, avoided.

Interpretation aside, a number of fairly universal and deeply felt human reactions to the phenomenon of defecation exist, all of which can be utilized to promote practices conducive to improved hygiene in the scientific sense of the word. Privacy, apartness, and dirt avoidance are all values that lend themselves to the use of modern excreta disposal technologies. Beyond these there are a range of widely shared values: smell avoidance, household cleansing, sweeping, clothes washing, and so on that contribute to a reliable common basis for domestic sanitation programs. Effective excreta disposal may, of course, require that people come to have some new understandings of the health hazards from excreta and of the measures that can be taken to avoid these hazards. There will be some situations in which traditional understanding and practice—for example, defecating into rivers that are also water supplies—is strongly contraindicated by modern interpretations of health and disease. In these instances, authorities may have to assume didactic roles, but they can nearly always do so by building upon traditional culture rather than by starting from scratch.

The widely shared cultural evaluations of excreta have an equally common but regrettable side effect:

1. The study of community reaction to a proposed sanitation project, and the elicitation of support and acceptance, are valuable activities in all countries. Krauss (1979) described the replacement of a malfunctioning tertiary sewage treatment plant in Greenville, Maine, USA, by an innovative land application system after extensive public consultation and participation. An immediate practical outcome was an increased willingness to pay sewer rates on the part of households connected, and an increased demand for connections on the part of those households unconnected.

people who, by their occupation, come into regular contact with excreta become themselves persons to be avoided. In many towns throughout the world, sweepers and night soil removers are drawn from disadvantaged minority groups living in segregated communities within the towns, and their occupation tends only further to reinforce their segregation. This is a rather intractable problem wherever some kind of cartage system is necessary for night soil removal.

## Influence of Social Structure and Organization

Any excreta disposal system is a complex social activity involving planners, administrators, politicians, and corporation workers as well as the individual user. Officials, for their part, can plan improved systems but may face difficulties in raising the necessary resources, cooperating with other agencies, delivering the goods, and, crucially, building up routine services for maintenance. There are additional problems in securing political support for low-income schemes when upper-income groups, who can better afford to pay and have more political weight, themselves clamor for higher standards of service.

Politicians face the full brunt of deciding priorities in urban development and, if they have to recruit public support to keep themselves in office, they often face pressures to employ more sweepers or to favor particular parts of the community. Workers, such as the operatives of cartage systems, will also have a number of preoccupations besides service to the city. They must secure for themselves a living wage and tolerable working conditions, and in their struggles with the authorities or with a public unwilling to see taxes increased they will use what sanctions they have at their disposal, chiefly the disruption of services.

In short, whatever high ideals about the quality of human life may be embodied in sanitation programs, such programs cannot escape being a part of the complex social system of a city, and any attempt to make them work better has to take this complex system into account. The following sections examine these social values and organizational issues in relation to the deposition, transport, and reuse of excreta.

## Social and Behavioral Aspects of Latrine Design

It is difficult to predict how people will respond to technical innovations because many factors enter into their choice. But much can be gained by the planner's appreciating the position of the user and looking at innovations from the user's point of view. For the user, the toilet itself is a most important element in the excreta disposal system. He may have to decide whether to invest in one, and he has daily to face using it. Even the most hygiene-conscious people will take more than cleanliness into account in making these decisions, and disadvantages may not have to be great before some people will opt out of whatever innovation is being proposed.

### *Cost*

The most obvious and perhaps most cogent of all social constraints on sanitation is the cost of latrines. The existing distribution of sanitary facilities (both nationally and internationally) is heavily skewed toward the rich to a large extent because sanitation is expensive. Many of the alternative technologies discussed in this book are cheaper (some much cheaper) in capital terms than the sewerage systems of the industrial West, but most of the savings occur in the cost *to the public authority* that is spared the expense of sewers.[2] The cost of the toilets themselves may still be considerable, and at some point down the scale of poverty it ceases to be reasonable to expect people to pay for their own installations. In many urban environments sanitation programs must be seen as attempts to overcome one of the multifarious effects of poverty; as such, they are bound to involve a degree of government intervention through subsidies. Where excreta have an economic value, some of the disposal costs can be balanced against the expected income from reuse, but this is more likely to defray the costs of cartage than the in-house costs of toilet fitments.

### *Convenience*

The location of latrines is important and must balance advantages. Sometimes a technology constrains the choice of location, but, assuming that all options are open, toilets may be sited inside the house or compound or some distance away. People may be sensitive about such matters as the prominence of the toilet to public view, and such factors must be evaluated in detail for each situation. Some general principles, however, may be postulated.

If the latrine is sited at some distance from the living quarters, people may be discouraged from using it on

2. See Kalbermatten, Julius and Gunnerson (1982) for an economic comparison of alternative sanitation technologies and for proposed "sanitation sequences" that enable users to improve their sanitation facilities to a level and at a pace they can afford.

dark nights or in inclement weather. Yet if it is close to the house, there may be a feeling that defecation is not adequately segregated from the rest of daily living. In a new tenement project in Madras where toilets were provided in each flat, housing officials found that some of these were filled with sand and the space used for other purposes. One explanation of this response is that defecation within these small apartments, even behind closed doors, was unacceptable to the occupants (Curtis 1978).

Sufficiently private locations for outside toilets may be difficult to find in urban environments. Draft plans for an urban site and service scheme in Africa made provision for the siting of latrines in the front corner of plots, where they could be conveniently linked to sewer lines along the roads. But there were considerable doubts as to whether this technical convenience would be socially acceptable—the first thing to confront household visitors would be the toilet. A privy should be private. Most societies have conventions relating to domestic space—for example, that the back of the house is private, the front public—and these conventions need to be discovered and respected.

People may be sensitive not only about the location of the toilet but about the journey to it as well. In Botswana it was found, through careful monitoring of a latrine program, that the act of carrying a container of water to the new privy (something quite acceptable in India) was an embarrassing announcement to the world at large of an individual's intentions. The design was subsequently modified to provide a water source at the latrine.

A major difficulty with toilets may be providing access to the right people at the right time. Householders may be inclined to keep outdoor latrines locked to prevent misuse by passers by, with the unfortunate consequence that they are then not available for children to use during the day. Similarly, in the tenement project in Madras, interior toilets were inaccessible to children while both parents were out seeking work during the day (Curtis 1978). Private toilets have to be carefully designed and located to secure both adequate access and adequate control. Counterbalancing these factors is the fact that most toilets provide a degree of privacy such that the time of day when defecation may conveniently take place is greatly extended from the dawn or dusk periods that are often favored by those with no facilities at all.

### *Comfort*

Comfort has been found to be a great selling point for latrine programs, but again the social requirements are difficult to predict. There are the well-recognized cultural preferences for sitting or squatting (the latter in part an act of avoidance of physical contact with possibly defiling surfaces), and there are also strong commitments to particular anal cleansing procedures that must either be accommodated by the new technology or, if necessary, changed. Additional design considerations are that children have anxieties about cavernous holes in squatting plates, that the aged may have special needs (the surroundings must not be slippery, for example), and that hot and malodorous latrines discourage all users.

A vital aid to comfort has been the inclusion of personal washing facilities in toilet installations, as in the comfort station program in Ibadan, Nigeria (Ademuwagun 1975; Pasteur 1979). Facilities for hand washing at the place of defecation are highly desirable in any case, but total body washing in privacy could also be much appreciated. In India, for example, customary sanitary prescriptions require a bath to follow defecation (Kochar 1978); in these circumstances, linked bathing and toilet facilities would greatly encourage use of both private and public latrines. Comfort concerns both physical conditions and the fulfillment of conventional expectations. Householders in Botswana found the ventilation gap left at the bottom of doors to their outside toilets disconcerting because people could see their feet. Conventional expectations may be numerous, and the only way to discover them is by carefully monitoring reactions to new designs in each situation.

In many rural areas latrine programs may face the problem that people find the bush more acceptable and more comfortable than pit latrines or even some more sophisticated technologies. This choice may reflect both that defecation is often regarded as a shameful activity properly confined to the wilds [as Muhondwa (1976) found in Tanzania] and that the latrines may be hot, malodorous, and fly ridden (an acknowledged tendency of many pit latrines). The first problem decreases when the bush becomes inaccessible, as in town, or is so diminished that it constitutes highly contaminated spinneys or copses in areas of intensive cultivation. At this point the population presumably becomes susceptible to new interpretations of what constitutes an appropriate environment for defecation. Latrines can be presented as answers to the problem of privacy, and an analogy with the bush may be maintained by siting the facility at a suitable distance from the house. Crowded urban environments present opportunities for creating new conventions, practices, and concepts of comfort that program directors should seize upon.

*Group or communal toilets*

Private, domestic latrines have so many advantages to the user over any arrangements by which members of different households share their facility that they are always preferable wherever people can afford them and space is available. High costs and problems of land scarcity, however, may oblige authorities to select communal facilities even though, from past experience, their success is highly problematical. The difficulty in all cases is maintenance. Public toilets have a poor record in this respect and have inherent shortcomings. It takes only one misuser, perhaps a child avoiding the frightening squatting hole, to establish a chain of subsequent misuse for which no one is willing to take responsibility.

There are two possible responses to the problem of neglect of public facilities that public authorities can make: attendance by a cleaner, or the provision of public toilets for identified or self-identified groups of households. The first is an expensive proposition requiring the deployment of cleaning personnel on a large scale. In general, arrangements of this sort are common only in public places such as market areas or main thoroughfares, where provision must be made for large numbers of occasional users who are passers by. The additional expense of an attendant is often covered by a small charge to the users, which of course is not possible in toilets designed to serve the requirements of a resident population. The arrangement most likely to ensure sustained cleanliness is to have one cleaner constantly stationed at each public toilet. A cheaper alternative is to have a cleaner responsible for several public toilets, which he continually travels among. This latter option can work well if the cleaner has adequate water supply and equipment, so that he can cope with a toilet that has become grossly fouled since his last visit. This system is used to maintain public toilets in Beijing, China: the cleaner has a three-wheeled bicycle, with equipment and boxes, and water is available at each facility (Feachem, personal observation).

The potential for achieving better management of public toilets by associating them with an identifiable group of households is currently being explored in the Ibadan comfort station program in Nigeria where, in the old town, the indigenous social structure of family groups provides a framework for the social control of latrines (Pasteur 1979). The facilities, designed to serve between 350 and 700 people, are built by the authorities with the people themselves providing the land from family holdings and contributing to the cost of construction. The group, under the leadership of the traditional family head, then either appoints a cleaner and pays him from a communal fund or allocates responsibility for cleaning and maintaining the separate toilets to each of the participating households. The pilot scheme was monitored by a health education team who identified several problems (Ademuwagun 1975). Cleaners were often badly in arrears in their pay, and, where the people themselves undertook to carry out cleaning and maintenance, the constant attention of the health education team seemed to be necessary if standards were to be upheld. A basic problem appeared to be paying for water and electricity, and in some cases supplies were withdrawn. This last problem raises questions about how best to divide tasks between the public authority and the local groups. Voluntary groups often have difficulties in collecting money from their members on a routine basis (Feachem and others 1978) because defaulters encourage those who would otherwise be inclined to pay regularly to be similarly lax.

In most cases public facilities must be provided either by public authorities or by these authorities in conjunction with the users. Where night soil has a commercial value, however, there may be potential for the commercial organization responsible for reuse to provide the toilets themselves. In Indonesia fishpond owners, who stand to make a profit from the cultivation of fish, provide a number of latrines overhanging their ponds for the use of the neighborhood. Whatever virtues or vices attend this system, the great advantage is that management and maintenance by a public authority is minimized because the fishpond owner has to maintain the facilities in a manner that is attractive to the requisite potential users. It is not clear, however, whether in other circumstances—cartage systems, for instance - it is possible to push contractors beyond servicing into providing the latrines.

## Social and Organizational Aspects of Excreta Cartage Systems

It is perhaps regrettable that a sewerage system is not only a technically efficient removal system (given the massive financial outlays to introduce it) but, once constructed, that it is also the easiest to organize and run. The technology may in part be complex, but the need for servicing is limited. Instead of an army of sweepers required to empty buckets and pushcarts, a sewerage system may be run by white-coated technicians assisted by a few manual workers whose job is performed away from the public gaze,

either underground or beyond the urban bounds. The labor force required is small, elite, and dispensable for short periods. Breakdowns in a sewage system usually cause environmental pollution at the treatment works and beyond rather than any direct contamination or disruption of the domestic environment of the users. In other words, as is the case with many modern technologies (Dickson 1974), a sewerage system is more amenable to social control than any of the less automated technical alternatives.

By contrast, cartage in its simplest bucket-latrine form requires large numbers of workers carrying out routine collection of night soil from households by using buckets, boxes, or barrows that must be emptied into carts of some sort for conveyance to a disposal point. The buckets have limited capacity, and the system is prone to crises both from mismanagement and from collective action on the part of the workers. Civic authorities face on the one side citizens with various means at their disposal for insisting upon reasonable service; on the other, authorities face workers who wish to exercise what strength they have to get a reasonable reward for performing an unpleasant and socially degrading job. Which party gets the relative advantage depends upon the labor market, politicians' need for political support, and other factors; in any case, cartage systems often present organizational and political problems for civic authorities. If these authorities decide to change to sewerage systems, the decision may reflect a desire to escape from the organizational problems involved in cartage.

Direct handling of night soil in cartage systems leads to a situation in which it is often only groups of strangers, refugees, or other disadvantaged minorities of the urban community who are prepared to take the job. In the Indian subcontinent the work is associated with the sweeper castes, whose untouchable status the Indian government has been endeavoring to overcome. In practice this kind of social change has proved very difficult wherever there is continuing association of a caste or single group with occupations such as night soil removal, and eliminating a stigmatized occupation is a major additional incentive to changing an excreta disposal system. But even stigmatized occupations may be in strong demand if alternative sources of employment are unavailable. Operators in parts of cities covered by private cartage systems may have to purchase the rights to service a street (Streefland 1978), and municipalities are often under strong political pressure to expand the number of sweepers in their employ. For a sweeper there is perhaps only one thing worse than being of low social status in a lowly regarded occupation, and that is being of low social status without any occupation at all. Thus, from a social as well as economic point of view, whether night soil cartage systems are appropriate or not depends on the state of the wider economy. If there are equally well-paid or better jobs available, then it must be assumed that night soil removers will select these, and there will be a strong argument for changing the disposal system. If there are not other jobs available, there will be strong pressure from the disadvantaged groups themselves to maintain the cartage systems.

If hand-operated cartage systems remain necessary, something can be done to improve the social position of the operator by improving the terms and conditions of service. Low status frequently is reinforced by low pay, which, if improved, would somewhat counteract low status. It may be difficult, however, to alter the pay structure radically while there remains a reserve corps of unemployed sweepers without simply encouraging subcontracting. Government policy on public sector pay may also limit the options and create problems of its own. In some cases minimum wage legislation may set the scale for manual labor in the public sector above market rates, causing labor-intensive technologies to be uneconomic while there are still surplus workers willing to do the job. In other cases, as in Port Sudan, Sudan (Spencer 1978), rates of pay set at levels not competitive with private sector employment make it difficult to build up and train adequate staff. But even if it is difficult to make major changes in pay, working conditions can be improved in other ways. Where work clothes are issued, they often are similar in appearance to those of convicts and serve to set the users socially apart more than to protect their bodies. Equipment is also often poorly designed and badly maintained, and facilities for washing and changing after work are inadequate or neglected altogether. Improvement in any of these dimensions will improve the social status of night soil removers.

Operators of vacuum trucks have a stronger bargaining position than workers in manual cartage systems because they are more skilled and, in any one town, fewer in number. Sealed vaults, because they have no treatment potential and limited capacity, also have a crisis point if they are not emptied on time, and organized labor can use this to its advantage. Septic tanks, in contrast, are less crisis prone and may for this reason be favored by authorities worried about the power of their organized labor force to make demands.

Improved technologies, requiring less direct handling of feces, may facilitate an upgrading of the status of night soil removers. Jobs with more skill will attract higher pay, enabling the workers to maintain a higher

standard of living. One seemingly counterproductive effect here is that, if the social stigma attached to night soil removal is effectively lifted by improved technology, these jobs may then be open and attractive to people outside the minority traditionally filling the occupation, so that this minority group loses its employment monopoly while keeping its low social status.

Because many towns will require improved cartage systems of one kind or another in the future, it is important to discover whether the social stigma attached to night soil removal can under any circumstances be removed. Evidence is hard to come by. Some reports from China (Streefland 1978) indicate that, because of the importance attached to health in that society, the status of night soil removers has improved since the revolution. In a society where reuse of excreta has always been practiced, however, it is unlikely that the job has ever carried the stigma that it does, say, in India, where the rituals of excreta avoidance are highly developed. Furthermore, the Chinese approach of involving the public in hygiene and sanitation improvement committees (Schwartz 1977), if tried elsewhere, would not necessarily lead to an improvement in the status of those people who are employed in night soil removal. As with attitudes towards excreta and waste disposal, the willingness of a society to participate in an organized way in this sector is culturally dependant. This remains an important area for future investigation.

In many societies where night soil is valued as a fertilizer, cartage is a private sector activity. Cartage contractors make their money by selling the material to farmers, by being paid for the job of removal itself, or by a combination of both. In some towns, different areas are serviced by small-scale contractors who make agreements with individual householders for night soil removal. In others, larger-scale operatives undertake contracts with city corporations. Some operate simple cartage systems, others may service septic tanks with vacuum trucks. Private contractors may be difficult to control, particularly where they are numerous and stand to gain from dumping their loads in the nearest watercourse instead of removing them from the city to agreed disposal points. A good price for the product, however, is an effective incentive to efficient night soil removal.

## Social and Organizational Aspects of Excreta Reuse Systems

It is now widely accepted among agricultural and sanitation planners that reuse of wastes is a desirable objective if it can be hygienically achieved. This conclusion brings experts into line with the large part of mankind that has always favored reuse. In many parts of the world the problem is not reuse but how to persuade people that additional stages of treatment are sufficiently important for their health to warrant the increased time and expense that treatment requires. Elsewhere, however, the idea of reuse is not easily accepted culturally. Many people share the prejudice of the villagers in Zola's novel *La Terre* against the old lady who nurtured beautiful vegetables by night soil, thus relieving her poverty but placing herself beyond the bounds of social acceptance. However deep seated these prejudices may be, the situation is far from irredeemable. There are several reasons why the significance of cultural barriers to reuse is less than it might first appear. Processing can transform something that is socially unacceptable into something that is much more easily accepted. An analogy may be drawn to the universal practice of food preparation: an animal or vegetable, unattractive in the wild state, becomes appetizing when cooked, arranged on a plate, and served with a sauce; so may excreta, despite their malodorous nature and value-laden associations, become attractive when treated and moved to another environment as compost or fertilizer. Part of the art of treatment must be the achievement of this cultural transformation that would enable farmers to use a substance with pleasant texture and acceptable odor for the enrichment of their land.

Unlike the true subsistence farmer who experiences the whole cycle of agriculture from production to consumption and back to production, a commercial farmer produces for a distant and impersonal market and is better prepared to use any agricultural aids conducive to a good market return. The urban consumer, for his part, can only judge food by its appearance in the market stall and knows little of its origins. The separation of producer and customer is both geographical and institutional. Its positive aspect is the diminished significance of individual preferences and prejudices upon the production processes; its negative aspect is that the public must be protected from unscrupulous or unhygienic practices through bureaucratically administered controls upon these production and marketing processes. Thus, fish grown in oxidation ponds managed by city corporations under controlled conditions can escape any stigma because, in the marketplace, they cannot be easily identified. In India, for example, produce grown in sewage-irrigated fields enters the market unnoticed, although in parts of that country reuse of night soil is not a favored practice. In London, England, on December 23 and 24, housewives of slavonic extraction

(mainly Poles) buy imported carp for their traditional Christmas Eve feast, little suspecting that some of these carp have been raised in sewage ponds.

Finally, at least in the West (and the West as a great consumer of natural resources is very important in this respect), prejudices against reuse are being counteracted by a new consciousness of a need to achieve ecologically sound farming practices and patterns of human existence. This takes the form both of an awareness of the undesirability of polluting rivers and seaboards with untreated or inadequately treated sewage and of the need to find substitutes for the energy-consuming (often petroleum-derived) artificial fertilizers that are required in large volumes in agriculture. This transformation of values, coinciding as it does with the more structural changes described above, has now proceeded to the point that constraints upon effective reuse are more questions of cost and technical feasibility (particularly the problem of mixing domestic and industrial wastes in most urban sewerage systems) than questions of cultural predisposition. If there remain effective scruples regarding reuse, these are more likely to lie with policymakers than with the users themselves, and top managers are the people most exposed to the new ideologies about conservation and the need to manage resources effectively.

In summary, how successfully the reuse of urban wastes can be controlled depends upon organization. On the urban periphery, people may treat and reuse their own night soil in local fields or gardens, making it very difficult for local authorities to establish workable controls. Similarly, small-scale private contractors in night soil removal who service a number of households and sell their product to farmers in the countryside may easily escape bureaucratically administered control measures. If the municipality itself administers night soil removal or contracts it to large-scale commercial enterprises, however, the authority is then in a position to enforce suitable treatment before the product is made available for reuse.

## Improving the Management of Urban Sanitation Systems

The success of sanitation programs hinges largely on the capability of the municipal governments or other public authorities who must promote, control, and service the schemes. These authorities must not only understand the nature of the task but must also be able to exercise their authority to enforce routines and ensure that the public plays its part. The need for administrative discipline extends beyond the supervision of routine operations to the collection of dues and the control of access to services. Experience past and present indicates that this management ability is often the chief limiting factor in sanitation programs (Rybczynski, Polprasert and McGarry 1978). Not only are urban services often inadequate in extent (to be expected in rapidly growing cities), but existing systems also suffer from malpractices that add to their deficiency. Contractors dump night soil indiscriminately in rivers or drains. Workers gain political protection when attempts are made to enforce work routines. Members of the public get their houses preferentially connected to water supplies or sewer lines by paying "speed money" to minor officials. The poor pay their dues while the rich avoid payment.

These difficulties are unlikely to occur if the public at large is solidly behind the policies of their authorities and can effectively exercise some influence upon the course of events. It is noteworthy that in postrevolutionary China, where improved sanitation has high priority, urban public services are backed by voluntary committees, sponsored by the ruling party, that serve to keep the authorities on their toes, while at the same time mounting health improvement campaigns and other voluntary activities (Streefland 1978). Elsewhere, a major role for community development officials, health education teams, and civic leaders must be the generation of public support for and commitment to environmental improvement—not so much for the direct action that this can achieve as for the backing of the authorities attempting to carry out their proposals. No civic administration can maintain the integrity of its programs for long without active public support. Furthermore, because the kinds of sanitation schemes envisaged here require radical changes in the distribution and organization of services, radical changes in civic consciousness will also be required.

Such changes are not always forthcoming. In this imperfect world, realistic plans may need to accommodate existing interests and commitments and endeavor to promote change in spite of weaknesses in urban government and administration. Two different responses are currently in evidence. The first is to create special-purpose agencies beyond the influence of local interest groups to take responsibility for the development of a single city (as in the case of the urban development authorities found in most Indian cities), to look after the interests of a particular class of citizen, or to provide for one kind of service on a regional basis. There is a trend toward specialized water and sanitation authorities in many different parts of the world. The protagonists of these special-purpose agencies believe that such agencies will be more

effective development bodies than the traditional civic authorities because they are free to draw up rational plans and follow priorities. Yet these bodies often find themselves in a competitive position with other authorities with similar or overlapping responsibilities, and they still require constant political support to be effective.

The other approach is to rely upon technologies that require minimal municipal commitment and to ask the potential users to construct and maintain latrines through "self help." Pit latrines or on-site composting toilets require little municipal effort (see table 3-2) beyond grants or technical assistance as inducements, enforcement of bylaws if this is deemed necessary, and some long-term emptying arrangements.

Neither of these two approaches can be regarded as a substitute for getting wholehearted commitment to improved hygiene and sanitation, based upon a broad understanding of potential health and welfare benefits, from politicians and citizens alike. This chapter concludes with a discussion of the strengths and weaknesses of self-help schemes (which can be more than simple substitutes for municipal endeavor) in meeting these objectives, and of health education.

## Effectiveness and Limitations of Self-help Schemes

The potential of self-help programs lies in the willingness of individuals or groups, even among the poorest elements in society, to perform tasks such as laying pipes, digging pits, or improving their physical environment for themselves. Self-help schemes can take advantage of the spirit of self-reliance sometimes found in informal or squatter settlements; they may also work well where the ruling political party is active in urban management and can organize and control development, as in recent sites-and-services projects in Lusaka, Zambia. Carefully planned self-help exercises can totally transform a town, as in the case of Port Sudan, Sudan, where unplanned settlements have been rebuilt and provided with basic services through the authorities and the people working in unison for a few days in each quarter of the town. Critical evaluations of self-help schemes (Chambers 1974; Feachem and others 1978; Holmquist 1970; Lamb 1971; Schaffer 1969) reveal, however, that self-help often gets out of hand and ends in frustration for all parties. The potential hazards of self-help schemes in sanitation can be summarized as follows:

- If participation is voluntary, some households will not participate for one reason or another and, because some health benefits depend upon complete coverage of the population, incomplete coverage will frustrate the objectives of the program.
- There is no guarantee that those people who are most in need will be those who are most willing to participate. To encourage self-help, the authorities will be obliged to help those who are prepared to help themselves. Thus, self-help initiatives can curtail the authorities' ability to decide upon priorities.
- Self-help can become a popular movement, backed by politicians for whom it provides a following, through which government finds itself committed to providing a level of service it lacks the financial or manpower resources to meet.
- Self-help programs have shown themselves to be much more effective at generating capital in the form of "one-shot" projects such as classrooms, clinics, or dams than in *maintaining* services once they have been established.

Some of these difficulties can be overcome if authorities take a more rigorous approach to the organization of self-help projects from their inception.[3] For instance, they may need to:

- Enact by-laws requiring all households to provide themselves with latrines
- Stipulate what categories of households they are prepared to assist with grants or technical guidance and only help those who help themselves within these categories
- Ensure that the number of projects undertaken does not outrun the funds available by persuading political leaders of the dangers in overstimulating demand and by requiring local groups to register their intentions with the authorities before undertaking a project
- Limit the scope of a scheme to a size that can be adequately serviced by the authority in the future.

In summary, self-help can best be used for clearly defined and limited operations, such as urban cleanliness campaigns or the initial construction of public or private facilities, in which the people's contribution reduces costs and generates enthusiasm. It can also be conveniently linked with the broader task of health education.

3. The advantages and dangers of self-help strategies in rural water supply programs, which have many similarities with sanitation programs, are discussed in detail by Cairncross and others (1980). The case against self-help is set out by Feachem (1980).

## Appropriate Health Education

At the beginning of this chapter it was said that some values, attitudes, and understandings can be accommodated by sanitary engineers, whereas other social factors must be confronted and changed. In rural areas little progress can be made in cholera elimination while people continue to locate privies over rivers that downstream are other people's water supply. Health education campaigns have to address specific issues of this kind while simultaneously creating a general awareness of the potentials of new technologies for improving living conditions. Health education is, however, often disappointing both in design and in results. There is a tendency to lecture the public about good hygiene, or balanced diet, or birth control, repeating textbook prescriptions without considering how the ideas apply in the listeners' particular circumstances. This tendency to patronize not only minimizes the many real strengths in existing knowledge and practice, it is also ineffectual. It fails to explore the users' viewpoint or to reveal the genuine problems that technical innovations pose for them. Health education has to be, above all, a dialogue between officials and users if full benefits are to be obtained (Isely, Sanwogou and Martin 1979). A good example of this two-way communication is the health education program that accompanied the Ibadan comfort station pilot scheme (Ademuwagun 1975). Not only were the positive values of the users explored here, but practical problems in implementation and maintenance, such as finding suitable sites and paying for water, were clarified. Without this kind of detailed knowledge of the users' perceptual and organizational problems, campaigns instituted by the authorities are almost certain to founder in disenchantment and disorder. Health education has a critical, sensory role in community affairs. It cannot merely be the vocal chords of the sanitation authorities, it must be their eyes and ears as well.

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*Part Two*

# Environmental Biology and Epidemiology of Specific Excreted Pathogens

# SECTION I

## *Excreted Viruses*

# 9

# Enteroviruses, Poliomyelitis, and Similar Viral Infections

OVER 100 DIFFERENT VIRUSES are known to be fecally excreted by man. New viruses are still being discovered, and several have yet to be fully characterized. Typically they infect the alimentary canal and are shed in very large numbers by infected persons (Madeley 1979). Diseases caused by these organisms range from the trivial to the serious or even fatal. The occurrence and medical significance of excreted viruses in the environment is the most rapidly changing field of knowledge reported here. A combination of heightened environmental concern (especially in the USA), improvements in laboratory techniques, and the recent discovery of important new human viral pathogens (especially rotavirus) have caused a marked increase in scientific activity, with several hundred papers yearly now being published on aspects of excreted viruses (EPA 1978; WHO 1979).

Table 9-1 presents a classification of some excreted viruses similar to that recently proposed by the World Health Organization (WHO 1979). This classification will undoubtedly undergo changes over the next few years as new viral agents are characterized and the taxonomy is revised. For the purposes of this book, excreted viruses are divided into three groups:

- The enteroviruses (chiefly polioviruses, coxsackieviruses and echoviruses), described in this chapter, which also contains some information on adenovirus and reovirus, as these are often considered jointly with enteroviruses; (see figure 9-1)
- The hepatitis A virus described in chapter 10
- The viruses possibly associated with gastroenteritis (rotavirus, Norwalk agent, and others) described in chapter 11.

## Description of Pathogens and Diseases

The enteroviruses are an acid-stable subgroup of the small picornaviruses. They are a large group causing a wide variety of diseases (table 9-1). The polioviruses are probably the most important. They were the first enteroviruses to be fully investigated, and because they are relatively easy to culture they have been used in most experimental work.

### *Identification*

Poliomyelitis is unique in being the major permanently crippling disease of infectious origin. It is caused by the infection of the central nervous system by poliovirus or occasionally another enterovirus. It is usually recognized by a sudden and unexpected onset of tiredness and weakness in the limbs. Fortunately, the clinical symptoms of poliomyelitis occur in only a very small proportion of the persons infected, usually a maximum of 2 and often less than 1 percent of the total.

The clinical effects of infection range from the asymptomatic through nonspecific minor illness to meningitis, paralysis and possibly death. There are two basic patterns of symptoms. The first is a minor illness arising a few days after infection, lasting 1–2 days and characterized by mild fever, listlessness, sore throat and vomiting. The second, developing 3–4 days later but often occurring without the first phase, is much more serious. Symptoms of aseptic meningitis, fever, severe headache, and vomiting are followed by stiffness of the neck and back. In paralytic cases the disease usually leads to progressive weakness resulting in severe paralysis. Death, usually caused by respiratory failure, may occur.

The disease is short-lived and most people recover fully, but many of the most severely affected are permanently disabled. Mortality among the paralytic cases varies between 4 and 10 percent depending on the virulence of the virus, the degree of medical care and the age of the patient. Diagnosis in asymptomatic cases is dependent on laboratory facilities in which the virus can be cultured from throat swabs or feces. Serological

Table 9-1. *Human Excreted Viruses*

| *Chapter in which described* | *Virus group* | *Family* | *Size and composition* | *Number of types* | *Diseases or symptoms caused* |
|---|---|---|---|---|---|
| 9 | Enterovirus | Picornaviridae | About 20–30 nanometers diameter. Single-stranded RNA in a protein shell | | |
| | Poliovirus | | | 3 | Poliomyelitis, meningitis, fever |
| | Coxsackievirus A | | | 24 | Herpangina, respiratory disease, meningitis, fever |
| | Coxsackievirus B | | | 6 | Myocarditis, congenital heart anomalies, meningitis, respiratory disease, pleurodynia, rash, fever |
| | Echovirus | | | 34 | Meningitis, respiratory disease, rash, diarrhea, fever |
| | New enteroviruses | | | 4 | Meningitis, encephalitis, respiratory disease, acute hemorrhagic conjunctivitis, fever |
| | Adenovirus | Adenoviridae | About 70–80 nanometers diameter. Double-stranded DNA in a protein shell | >30 | Respiratory disease, eye infections |
| | Reovirus | Reoviridae | About 75 nanometers diameter. Double-stranded RNA in a double protein shell | 3 | Not clearly established |
| 10 | Hepatitis A virus | ? Picornaviridae | About 24–29 nanometers diameter. Single-stranded RNA | 1 | Infectious hepatitis |
| 11 | Rotavirus | Reoviridae | About 70 nanometers diameter. Double-stranded RNA in a double protein shell | ? | Vomiting and diarrhea |
| | Astrovirus | ? | About 28 nanometers diameter | ? | ? |
| | Calicivirus | ? | About 35–40 nanometers diameter. Single-stranded RNA in a protein shell | ? | Vomiting and diarrhea |
| | Coronavirus | Coronaviridae | Between 20 and 220 nanometers diameter. Pleomorphic with petal-shaped projections 20 nanometers long. Single-stranded RNA in protein shell and lipid envelope | ? | Common cold |
| | Norwalk agent and other small round viruses | ? | About 20–35 nanometers diameter | ? | Vomiting and diarrhea |
| Not described | Adeno-associated virus | Parvoviridae | About 19 nanometers diameter. Single-stranded DNA in protein shell | 4 | Not clearly established but associated with respiratory disease in children |

tests can also be used. Treatment is supportive in nature.

The other enteroviruses can cause a wide variety of symptoms (table 9-1). These viruses are generally less dangerous than poliovirus and like poliovirus they only cause significant disease in a small proportion of cases. They normally infect the alimentary canal or the respiratory tract, giving rise to gastroenteritis or influenza-like symptoms. More severe disease is often associated with the spread of the virus to other organs such as the liver or central nervous system. As with polio, the effects are generally short-lived, and treatment is supportive.

Diagnosis is either based on symptoms or on laboratory culture and identification. Diagnosis is complicated by the fact that the same virus may cause different symptoms in different patients and that different viruses may give rise to similar symptoms.

*Occurrence*

These infections occur worldwide and are very common. Some isolated communities have been known that were not infected with poliovirus but few or none now remain. The other enteroviruses have a similar distribution, but there are local variations in both virus types and in the virulence of various strains.

*Infectious agents*

Poliovirus is a small spherical particle 28 nanometers in diameter and is therefore not visible by normal light microscopy (figure 9-1). It occurs in three serotypes, numbered 1 to 3. A most important characteristic is the degree of neurovirulence, which is known to vary from strain to strain in all three types. The infective dose is small: probably as little as one virus particle. Poliomyelitis can occasionally be caused by coxsackie- and echoviruses.

Like poliovirus, the other enteroviruses are submicroscopic spherical particles with sizes ranging between 20 and 30 nanometers in diameter. The number of types in each group is given in table 9-1.

*Reservoir*

The reservoir of poliovirus is man. Chimpanzees have been known to catch the disease in captivity, and monkeys may also act as natural hosts, but nonhuman reservoirs have not been shown to be significant.

As with poliovirus, man is the main reservoir of infection for the other enteroviruses. A number have been isolated from pets and other animals associated with man, but it is unclear whether or not they were naturally infected. Reovirus appears to be an exception, having been isolated from a surprisingly large range of animal species. Even so, animals have not been shown to be a significant source of infection for man.

*Transmission*

Infected persons can shed very large numbers of virus particles—more than $10^6$ per gram of feces. Viruses are also present in throat secretions, especially during the early stages of infection. These particles are highly infectious and can remain viable for a considerable period under suitable conditions. Infection takes place when the virus is ingested, possibly in food or water. The primary sites of infection are the throat and the lower alimentary canal. Within a few days the virus spreads to the lymphatic system and the blood stream. This phase corresponds with the minor symptoms of infection. In the small proportion of severe cases the virus infects the central nervous system, possibly by spread along nerve fibers.

On infecting a suitable cell, the virus diverts the cell's metabolic activity to the production of large numbers of virus particles identical to the original virus. These are liberated when the cell breaks down, and they either infect other cells or, in the case of the cells lining the alimentary canal, are passed out in the feces. Transmission is mainly directly from person to person either by the oral-oral route or the fecal-oral route. There is some indication that in unhygienic conditions the latter route is the most common, with the former route being more important under more sanitary conditions. Children under the age of 2 years are the most potent disseminators by both routes.

There are indications that poliovirus is carried between family groups by young children who are both susceptible and mobile (2–6 year age group). Infection then spreads within the family downwards to nonmobile children and upwards to older children and adults. Up to 50 percent of persons having resistance from earlier infections may become reinfected, but in these cases the excretion of viruses is much reduced and symptoms absent.

The other enteroviruses are probably transmitted by the same route as poliovirus, whereas the adenoviruses, which are normally associated with upper respiratory tract infections, are mainly spread by an airborne route from contaminated throat secretions.

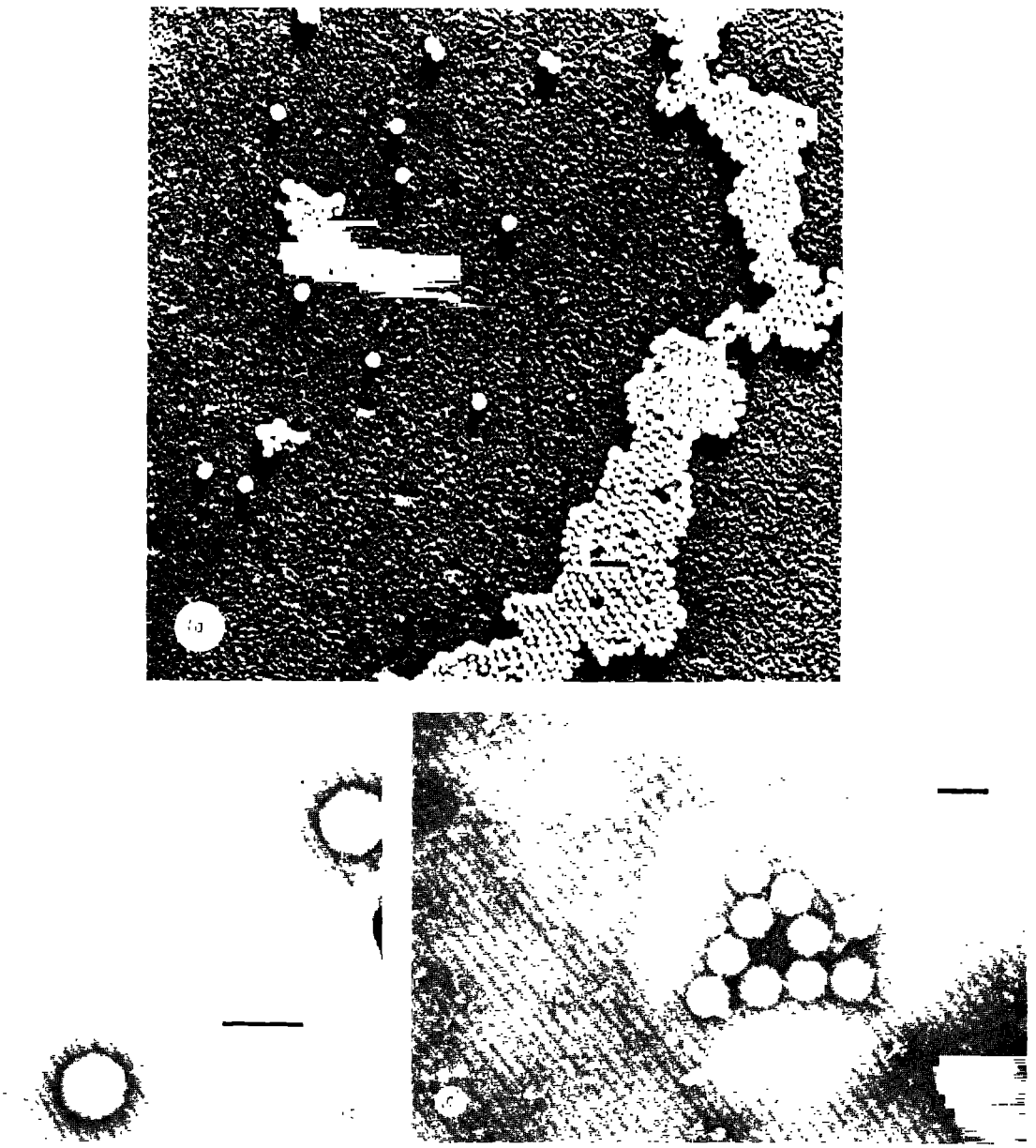

Figure 9-1. *Polio-, adeno-, and reoviruses under electronmicroscopy.* (*a*) Polioviruses under scanning electronmicroscopy. Other enteroviruses have a similar appearance. Scale bar = 0.1 micrometers. (Photo: World Health Organization, Geneva, Switzerland.) (*b*) Adenoviruses under transmission electronmicroscopy. Scale bar = 0.1 micrometers. (Photo: A. J. Zuckerman, London School of Hygiene and Tropical Medicine, London, U.K.) (*c*) Reoviruses under transmission electronmicroscopy. Scale bar = 0.1 micrometers. (Photo: A. J. Zuckerman, London School of Hygiene and Tropical Medicine, London, U.K.)

*Incubation period*

Minor illness when present occurs within 2–3 days of infection. Nervous system involvement possibly leading to paralytic poliomyelitis may occur between 5 and 35 days after infection—on average 17 days. The other enteroviruses are generally similar to poliovirus.

*Period of communicability*

Viruses have been found in the throat secretions and feces within 24 hours of infection, and contact cases have been observed within 3 days. Virus excretion, mainly in the feces, has been observed for as long as 17 weeks, and on average 7 weeks.

Most of the other enteroviruses are excreted for a shorter period than poliovirus. Coxsackie B viruses, for example, are excreted for 1 week or less. The adenoviruses, however, may persist in a latent form in the tonsils and adenoids.

### *Resistance*

Young children are the most susceptible age group, most adults having acquired resistance to poliovirus during earlier infections or by vaccination. Infection leads to the development of life-long immunity to the infecting type of virus, but the individual may still be vulnerable to other types.

The severity of the disease is markedly dependent on the age of the patient; in a nonresistant population, teenagers and young adults show the most severe symptoms. Certain other factors have been found to increase the severity of the disease—including tonsillectomy, pregnancy, recent inoculations, physical exertion, and trauma.

Infection by the other enteroviruses usually confers resistance in a similar manner, but vaccines are not generally available.

### *Epidemiology*

During this century the incidence of poliomyelitis has been observed to change from a constant background infection to epidemics of increasing severity. In temperate climates these occur in late summer and early autumn. In the tropics and subtropics the fluctuations are less marked, but the trend is the same. The reasons for this change are not fully understood but may be due to variations in hygiene or other factors affecting virus transmission and so leading to variations in the resistance within the community. In areas with poor hygiene children acquire immunity while very young, and the proportion of paralytic cases is therefore low and confined to this age group. Disruption of this pattern of infection may lead to a higher incidence of severe symptoms at a later period. Another possibility is that more virulent strains of virus have been introduced.

Many of the other enteroviruses act in a similar manner to poliovirus. Often serial waves of infection move through the community, fading away to a very low or undetectable level and being replaced by infection with another type of virus. Seasonal variations also occur.

## Control Measures

In considering the control of enterovirus infections, one must distinguish clearly between reducing infection and reducing disease. In the case of poliovirus, for instance, environmental measures may reduce transmission and thus the incidence of infection. However, these measures may increase the incidence of serious clinical disease by deferring the age of first infection to that at which disease is more likely to be severe. A survey of 10,000 households in 25 villages in Gujarat (India) found that the number of paralytic poliomyelitis cases per 1,000 households was related to household income. Among low-income households there were 12 cases per 1,000 houses; among middle-income households there were 18 cases per 1,000 houses; and among high-income households there were 24 cases per 1,000 houses (Jhala, Goel and Dave 1979). Contrary evidence is presented in a recent review showing that the real (as opposed to reported) annual incidence of paralytic poliomyelitis in Burma, Egypt, Ghana and the Philippines is between 233 and 3,800 per 1 million children age 0–4 years, and between 37 and 589 per 1 million of the total population (Sabin 1980). About 90 percent of paralytic episodes in these countries occurred during the first 3 years of life. Despite the fact that for various technical reasons these annual incidences are underestimates, they are similar to or considerably higher than the rates occurring in the USA in the immediate prevaccine era. These data cast doubt on the belief that improved living conditions, in the absence of vaccination, increase the incidence of poliomyelitis disease.

### *Individual*

Highly efficient vaccines are available for the three polioviruses. Both killed and live attenuated vaccines can be used. The live vaccine is probably preferable in developing countries because it is easily administered on a lump of sugar, whereas several injections are required for the killed vaccine. The live vaccines contain a mixture of attenuated (weakened) strains of virus that establish an infection which leads to resistance but, unlike many of the wild strains, does not infect the nervous system. These attenuated strains can also spread from person to person and so immunize a greater number of people but do not spread as efficiently as the wild virus. The wild viruses are suppressed but not eliminated from the community. It is therefore necessary to maintain the vaccination of young children to prevent the build up of a susceptible

group. Resistance appears 7–10 days after vaccination. The response is sufficiently rapid to be of great benefit during an epidemic. No vaccines are available for the other enteroviruses.

No specific drugs are available either for chemotherapy or prophylaxis. Good personal hygiene and the avoidance of contaminated food and water may reduce the risk of mild infection in early childhood but thereby increase the risk of severe infection later.

*Environmental*

Improvements in excreta disposal alone are unlikely to have a great impact. The highly infectious nature of viruses, the preponderance of young children among the cases, and the large proportion of symptomless infections indicate that the main route of infection will remain from person to person. The elimination of excreta as a source of infection may change the primary means of transmission from the fecal-oral to the oral-oral route. Improvements in both general hygiene and excreta disposal are likely to have an effect and may be responsible for the trend toward the epidemic type of poliovirus transmission. In these circumstances infection will probably be delayed rather than prevented, and the proportion of patients with severe symptoms may increase. This can be prevented in the case of poliomyelitis by the use of vaccines.

Polioviruses and other enteroviruses have been isolated from flies and cockroaches. For instance, in Texas (USA) polioviruses were isolated from 15 percent of flies, while coxsackieviruses were isolated from 45 percent, and flies experimentally fed polioviruses continued to excrete them for up to 2 weeks (Melnick and Dow 1953). It is clear from this and similar studies that insects can pick up viruses and may subsequently contaminate food, but it is unknown whether this mode of transmission is of any epidemiological significance (see chapter 37).

## Occurrence and Survival in the Environment

Viruses are not capable of multiplying outside of living cells; therefore, in the environment their numbers can only decrease. In favorable conditions, however, they can survive for months. Their survival is aided by neutral pH and the presence of particulate or organic matter, moisture and, in particular, low temperatures. Resistance to inactivation varies considerably among different types of virus and even among different strains of the same virus. Inactivation is a rate process, and the removal of infectivity therefore depends on both the efficiency of removal and the numbers initially present. In feces and sewage these may be higher than $10^6$ per gram and $10^6$ per liter, respectively.

The enteroviruses (chiefly polio, coxsackie, and echo), and to a lesser extent adenoviruses and reoviruses, have been the only excreted viruses to be extensively studied in the environment. This is partly because certain other important excreted viruses (particularly hepatitis A virus and rotavirus) cannot be routinely grown in cell culture at the present time. However, as laboratory skills improve, and as models for human excreted viruses are developed (for instance, reoviruses and simian rotaviruses may provide models for human rotaviruses), more data will be obtained on the environmental behavior of excreted viruses other than the enteroviruses. Preliminary evidence from a few studies indicates that there may be significant differences between rotavirus, hepatitis A virus, and the enteroviruses in their environmental characteristics (Farrah and others 1978: Wallis and Melnick 1967), although a recent study has shown that simian rotavirus has survival properties in fresh and saline waters similar to enteroviruses (Hurst and Gerba 1980).

Throughout the rest of this chapter, data are presented on the numbers of viruses that researchers have isolated from various environmental samples. It must be stressed that these numbers depend very considerably on the techniques used; in general, as techniques improve reported concentrations of enteroviruses from a particular source (for example, river water) increase. The very earliest studies reported only the proportion of samples from which viruses could be isolated—for instance, 62 percent of sewage effluent samples contained enteroviruses. Subsequently, quantitive techniques were developed that were based on observed cell death following inoculation with varying dilutions of sample, and these yielded a count of median tissue culture infective doses ($TCID_{50}$) per volume of sample. More recently, most laboratories have adopted a technique whereby a direct count is made of plaques formed by viruses on cell monolayers, or in cell suspensions, which yields a count of plaque-forming units (PFUs) per volume of sample. In both the TCID and PFU techniques, counts depend upon the choice of cell line because different viruses will replicate with varying readiness in different primate cells (Schmidt and others 1978).

When viruses are present in small numbers in large volumes of sample, a variety of different methods can be used to concentrate them. When viruses are bound

to each other in clumps, or are adsorbed to solid particles, efforts must be made to disaggregate them. Even adopting the most careful and sophisticated techniques, recovery of seeded enteroviruses from environmental samples is typically below 60 percent. To add to the complexity and uncertainty, results obtained are still too dependent upon the personal technique of the laboratory staff and the whole tradition and routine of the particular laboratory. Quantitative data on viruses in the environment should therefore be taken as indicative only. Throughout this chapter concentrations are given as viruses per volume or weight of sample although, strictly speaking, they should be as infective units or $TCID_{50}$ or PFUs per sample.

*In surface waters*

Enteroviruses can be isolated in low concentrations from almost all surface waters receiving human wastes. For instance, the Thames at London (UK) contains up to about 100 enteroviruses per liter, with a peak usually occurring in winter (WHO 1979; the River Sowe (UK) in December contained up to 620 enteroviruses per liter (Morris and Waite 1980); the Missouri and Mississippi rivers (USA) have yielded up to 0.1 and 0.4 enteroviruses per liter respectively; the Seine at Paris and the Moselle at Nancy (France) have contained up to 170 and 280 enteroviruses per liter, respectively (Berg and Metcalf 1978).

Survival in water is dependent primarily upon temperature and the degree of contamination. Studies listed in the appendixes of Feachem and others (1980) show that at temperatures less than 10°C survival times of between 24 and more than 272 days are reported, while at temperatures above 20°C the range is 4 to 135 days. In a study of enterovirus survival in the Rio Grande (New Mexico, USA), at 23–27°C, 90 percent inactivation occurred in 25 hours for poliovirus 1, 19 hours for poliovirus 3, and 7 hours for coxsackievirus A13. At river temperatures of 4–8°C, the time for 90 percent inactivation of poliovirus 1 was 46 hours (O'Brien and Newman 1977). Niemi (1976) studied the survival of coliphage T7 in samples of Finnish river water and found that, after 64 days at 3°C, a 99.5 percent reduction occurred, whereas after 64 days at 20°C a 99.98 percent reduction was recorded. Joyce and Weiser (1967) found that poliovirus did not survive for more than 63–84 days at 20–25°C, but survived for more than 91 days at 4°C when stored in samples of various farm pond waters. Hurst and Gerba (1980) studied the survival of poliovirus 1, echovirus 7, coxsackievirus B3 and simian rotavirus in polluted and nonpolluted fresh water at 20°C and found that a 3 log unit reduction occurred in 3 to >14 days. For each virus, survival times were similar in polluted and nonpolluted waters. The strong influence of temperature on enterovirus survival is illustrated by the survival of 34 percent of enterovirus during 7.1 days of travel under the ice of a frozen 317 kilometer section of the Tanana River (Alaska, USA) (Dahling and Safferman 1979).

A number of workers (for instance, Cubbage and others 1979, Katzenelson 1978, Young and Sharp 1977) have noted that the observed loss of infectivity of viruses in water may be due in part to genuine damage to the virus and in part to an artifact caused by many viruses aggregating and simulating a single infectious particle. This aggregation may involve the adsorption of viruses onto suspended particulate matter or it may involve the formation of virus clumps.

An important aspect of the behaviour and survival of viruses in natural waters is their tendency to become adsorbed to organic or inorganic suspended particles. Adsorption is enhanced at slightly acidic pH and in the presence of divalent cations and is deterred by the presence of soluble proteins (Schaub and Sagik 1975; Schaub, Sorber and Taylor 1974). Furthermore, viruses adsorbed to solids retain their infectivity both to tissue culture cells and mice (Moore, Sagik and Malina 1975; Schaub and Sagik 1975; Schaub, Sorber and Taylor 1974). Adsorption to solids may cause an accumulation of viruses in bottom sediments, from which they may subsequently be resuspended in the overlying waters. Wellings, Lewis and Mountain (1976) isolated more than 15 excreted viruses per 100 grams of mud nearly 1 kilometer dowstream from a primary effluent discharge site. No virus was isolated from the overlying river water at this site.

There is very little evidence that the transmission of enteroviruses during recreation in polluted surface waters is of any public health importance. The spread of adenovirus in swimming pools has been demonstrated or suspected in several investigations (for instance, Heinz and others 1976).

In summary, a few enteroviruses may survive for many months, although 90 percent reduction usually occurs within a few days, and 99 percent reduction within 1 month. Temperature is the single most determining factor and 99 percent reduction at 20°C may be expected within about 10 days. Survival is longer in heavily polluted or in very clean waters. Very little data are available on virus survival in surface waters in the tropics (Lund 1979), and more research is required. Addy and Otatume (1976) isolated enteroviruses from 28 percent of surface water samples in

the Accra area (Ghana), with highest isolation rates (80 percent) obtained from polluted street drains. However, because surface waters in the tropics are typically in the temperature range of 20–30°C, it is reasonable to expect that survival times will be considerably shorter than those generally reported from temperate areas.

*In groundwater*

Enteroviruses have been isolated from groundwater, especially in situations where the groundwater is shallow and sewage effluent or sludge has been applied to the overlying soil. Survival of enteroviruses in groundwater may be somewhat longer than in similar surface waters at the same location because in hot climates the groundwater is cooler than the surface water (in cold climates the opposite may be true) and groundwater is not exposed to sunlight.

Wellings and others (1975) demonstrated enteroviruses in shallow groundwater beneath a wastewater irrigation site in Florida (USA) and showed that the viruses could survive in groundwater for at least 28 days. Wellings also reported a tentative association between the pollution of groundwater by septic tank effluent and an outbreak of disease associated with echovirus 22/23 complex at a migrant labor camp in Florida (Wellings, Mountain and Lewis 1976). Vaughn and others (1978) reported low levels of virus contamination (up to 3 viruses per liter) in wastewater recharged groundwater on Long Island (New York, USA) at sites where recharge basins were located less than 10 meters from the aquifer. This contamination occurred despite the fact that all wastewaters had undergone either chlorination or tertiary treatment by sand filtration. Slade and Edworthy (1981) isolated up to $1.3 \times 10^4$ viruses per liter from groundwater in chalk beneath groundwater recharge lagoons receiving raw comminuted sewage, but failed to detect viruses from boreholes 120 meters and 400 meters downstream (in relation to groundwater flow) from the recharge area. A study of groundwater pollution in Israel isolated enteroviruses in 20 percent of 99 samples and in 12 samples enteroviruses were isolated in the absence of fecal coliforms and fecal streptococci (Marzouk, Goyal and Gerba 1979).

Little is known about virus survival in groundwater, but estimates may be made from the data on survival in surface waters reported above. Yeager and O'Brien (1977) reported that a 90 percent reduction of enteroviruses in groundwater occurred in 11 to 14 days. Information on the travel of enteroviruses through soils to pollute groundwater is contained in the sections below on survival in soil and on sewage treatment by septic tanks and land application. The literature on viruses in groundwater has been reviewed elsewhere (Keswick and Gerba 1980).

*In drinking water*

Attention has been paid recently to the occurrence and epidemiological significance of enteroviruses in water supply systems (Anon. 1978: Committee on Viruses in Drinking Water 1979; EPA 1978; Mahdy 1979; Melnick, Gerba and Wallis 1978; WHO 1979). Enteroviruses have been isolated in very low concentrations from some treated, chlorinated water supplies in France, India, Israel, Italy, Rumania, South Africa, USA and USSR (Committee on Viruses in Drinking Water 1979; Gamble 1979; Kott and others 1974; Melnick, Gerba and Wallis 1978; Rao, Lakhe and Waghmare 1978; WHO 1979). A considerable debate has developed over whether these very low virus concentrations in treated drinking water constitute a cause for concern on public health grounds. There is strong support, especially among environmental virologists in the USA, for the concept that, because viral infectious doses may be very low, small numbers of viruses in large volumes of drinking or recreational water are important. It is postulated that viruses in water may cause low-level transmission which remains undetected due to the large proportion of asymptomatic infections and the varied symptomatology (table 9-1) in those individuals experiencing frank disease (Berg 1967; Committee on Viruses in Drinking Water 1979; WHO 1979). Some of those who take this view urge the adoption of stringent virus quality standards, such as less than one infective unit per 40–100 liters in recreational water and less than one infective unit per 100–1,000 liters in drinking water (Mahdy 1979; Melnick, Gerba and Wallis 1978; Shuval 1975; WHO 1979). This school of thought has been influenced by the unfortunate view that, because it is technically possible to achieve a certain level of water purity, it is therefore desirable.[1]

1. For example, Melnick (1976) writes: "I suggest ... a maximum of one detectable virus unit per 10 gallons of recreational water and a maximum of one infectious virus unit per 100 gallons of drinking water. As our methods for detecting and monitoring water supplies have continued to improve, I would suggest that we can do better, and raise the standards to a maximum of one infectious virus per 1,000 gallons of drinking water." Similarly, the WHO Scientific Working Group on Human Viruses in Water, Wastewater and Soil concluded that "the presence of even a few enteric viruses in a large volume of drinking water should be prevented, since treatment measures exist to achieve this goal and detection techniques are becoming available which can provide the required level of monitoring" (WHO 1979).

This point of view is refuted by others (especially European workers and those with an epidemiological perspective) who point out that there is no evidence for the existence of low-level waterborne transmission and that, even if it did exist, it might make no significant contribution to the maintenance of the endemicity of enterovirus infections (Gamble 1979). The authors of this book support this second viewpoint. There is strong epidemiological and theoretical evidence that enterovirus transmission is primarily by the person-to-person route[2] and may indeed be oral-oral as well as fecal-oral. It is unlikely that very low concentrations of enteroviruses in treated drinking water make any epidemiologically significant contribution to transmission[3], and any decision on increased water quality standards, implying increased treatment costs, must await evidence of the benefits to be expected from such a policy. This view is especially pertinent in developing countries where there are severe shortages of both financial and technical resources.

Some studies on the survival of enteroviruses in treated drinking water are listed in the appendixes of Feachem and others (1980). One study (Lefler and Kott 1975) found that at 18–25°C 99.9 percent of polioviruses were inactivated in 91 days in tap water and in 112 days in distilled water. At 4–8°C, poliovirus was completely stable in tap water and distilled water for 231 days. Kott, Ben-Ari and Vinokur (1978) reported a 99.9 percent reduction of poliovirus after 80 days in tap water at 18–23°C.

A quite distinct problem is that of enteroviruses in untreated and polluted drinking water. Very few data exist on virological aspects of water supplies in developing countries (Lund 1979), but the bacteriological data indicate the probability of substantial viral pollution of many sources. A study in Ghana (Addy and Otatume 1976) isolated enteroviruses from 3 out of 8 water samples taken from 3 drinking water wells near Accra. Poliovirus 1 was isolated from one well and, since vaccination rates are extremely low, it is likely that this was a wild strain of poliovirus. More data of this type are urgently required.

*In seawater*

Many coastal communities discharge untreated or partially treated wastes into the sea. This is not only the case in developing countries but is common practice throughout the world; for instance untreated sewage is discharged in large quantities at Honolulu and Miami Beach (USA) (Ruiter and Fujioka 1978; Edmond, Schaiberger and Gerba 1978). The potential health hazards are those of infection of bathers and marine sportsmen and the contamination of shellfish. Recent advances in laboratory techniques have permitted the concentration of small numbers of viruses from large volumes of turbid seawater (Payment and others 1976) and have encouraged a number of investigations into viral pollution of the marine environment.

Edmond, Schaiberger and Gerba (1978) studied enterovirus contamination of seawater along the Florida coast (USA), an area of exceptional importance for marine recreation. Between Palm Beach and Virginia Key there are 10 ocean outfalls discharging approximately $6 \times 10^5$ cubic meters per day of raw and treated sewage. The Miami Beach outfall discharges $1.8 \times 10^5$ cubic meters per day of untreated sewage at a depth of 44 meters. The authors found enteroviruses at the surface above the outfall at concentrations between 21 and 42 infectious units per 400 liters. Fecal coliforms at this site were in the range $0.9–1.4 \times 10^4$ per 100 milliliters, and fecal streptococci were $0.5–4.9 \times 10^3$ per 100 milliliters. At Miami the outfall discharges $2 \times 10^5$ cubic meters per day of sewage treated by activated sludge and chlorination at a depth of only 5 meters. At this site between 0 and 3 enteroviruses per 400 liters were detected, with fecal coliforms always less than 3 per 100 milliliters and fecal streptococci less than 33 per 100 milliliters. The marked difference in fecal pollution caused by discharging untreated and treated effluent was clearly demonstrated.

Loh, Fujioka and Lau (1979) reported that the city of Honolulu (Hawaii, USA) discharges $2.5 \times 10^5$ cubic meters of raw sewage per day into the Mamala Bay at a point 3.2 kilometers from Ala Moana beach and 6.5 kilometers from Waikiki beach. Up to 420 enteroviruses per liter were isolated from the sewage, and enteroviruses were isolated from the bay water at distances of up to 3.2 kilometers from the discharge site. At some sampling locations distant from the sewage outfall, enteroviruses were isolated from waters which contained negligible numbers of fecal indicator bacteria.

2. In this connection, it is unlikely that the assertion of Berg (1978*a*) that "the source of most of the viruses that infect man through the oral route is wastewater" is true. There is certainly no evidence to support it, whereas there is ample evidence of the vigorous transmission of enteroviruses, particularly vaccine-derived polioviruses, among members of the same household and especially among very young children (see the subsection below on enteroviruses in feces and night soil).

3. Further evidence of this is provided by unpublished data from Britain which shows that enterovirus levels in rivers and reservoirs (and therefore presumably in drinking water) tend to peak in winter, whereas enterovirus infections, and levels in sewage , tend to peak in late summer.

Survival of enteroviruses in seawater is generally reported to be shorter than in freshwater. However, enteroviruses survive for longer in seawater than coliform bacteria and there have been several reports of enterovirus isolations from seawater containing very few or no coliforms (Berg and Metcalf 1978). Won and Ross (1973) reported a 99.9 percent reduction of echovirus 6 in aerated seawater after about 2 days at 22°C and after about 6–7 weeks at 3–5°C. The survival in seawater was unaffected by the addition of organic substances. Akin and others (1976) reported a 99.9 percent reduction in poliovirus 1 in 5–6 days at 24°C in the Gulf of Mexico. Fujioka, Lau and Loh (1978) found a 90 percent reduction in poliovirus at 25°C in 1 day in seawater collected close to the shore, and in 2–3 days in seawater collected farther out. Further experiments revealed the strong possibility of a specific virucidal activity displayed by unidentified marine microorganisms. In another study the same authors (Loh, Fujioka and Lau 1979) reported 90 percent reduction of poliovirus 1 in 2 days, and 99.9 percent reduction in 4 days, in both sewage-contaminated and sewage-free seawater at 24°C.

Considerable attention has focussed upon the study of enteroviruses in estuaries and the associated risk of viral disease transmission via contaminated shellfish. Metcalf, Wallis and Melnick (1974) reported studies of pollution of the Houston ship canal (temperature 10–33°C; salinity 0.1–1.1 percent; fecal coliforms 6.6–500 $\times$ $10^3$ per 100 milliliters) and Galveston Bay (temperature 9–30°C; salinity 1–2.4 percent; fecal coliforms <2–330 per 100 milliliters) in Texas (USA). Two activated sludge plants were discharging up to 1.7 $\times$ $10^9$ enteroviruses per day into the ship canal, and the dominant virus types identified were polioviruses 1 and 2 and echovirus 7. In the ship canal, 0.8 kilometers downstream from the nearest effluent discharge site, enterovirus concentrations were 0.05–0.9 per liter, whereas 6.4 kilometers further downstream concentrations were 0.08–0.7 per liter. Enteroviruses were also isolated from the bottom sediments of the ship canal at concentrations of up to 4 per 100 grams. In Galveston Bay, 33 kilometers from the discharge site, no enteroviruses could be detected in the water, but polioviruses 1 and 2 were isolated from oysters (concentrations up to 26 per 100 grams). Further examination of 89 poliovirus isolates from the ship canal and oysters indicated that 8 percent might be wild, virulent strains. Survival tests in the laboratory indicated that, in ship canal water at 22°C, poliovirus 1, coxsackievirus B5 and echovirus 7 were eliminated within 21 days. Further studies in Galveston Bay (Gerba and others 1979) showed that the concentration of enteroviruses was weakly but significantly correlated with rainfall, total coliform counts and salinity (accounting together for approximately 16 percent of the variance in the virus data). Enteroviruses were isolated from 43 percent of recreational water samples judged acceptable by total coliform standards (<1,000 per 100 milliliters), and from 35 percent of shellfish-harvesting waters judged acceptable by total coliform standards (<230 per 100 milliliters). Similar studies in the same area are reported by Goyal, Gerba and Melnick (1978).

Studies of pollution in the Firth of Forth estuary (Scotland) revealed adenoviruses, coxsackieviruses, polioviruses and echoviruses at two sites where median fecal coliform levels were 2.1 $\times$ $10^4$ per 100 milliliters and 4.2 $\times$ $10^3$ per 100 milliliters (Watson 1977). Studies in an estuarine environment in the USA (salinity 0.9–2.8 percent, temperature 6–24°C) isolated enteroviruses from 6 percent of water samples and 17 percent of bottom sediment samples (Vaughn and Metcalf 1975). Survival experiments in the same study showed a 99 percent reduction in coxsackievirus B3 in about 12 days at summer temperatures (18–21°C), whereas this degree of reduction took approximately 28 days during winter and spring (4–15°C).

Colwell and Kaper (1978) reported viral stability for 322 days at 4°C, whereas at 25°C viruses were rarely detected after 56 days. These results were unaffected by salinity changes in the range 1 to 3.4 percent. Roberts, Haggerty and Johnson (1976) found that poliovirus 2 suffered an 84–88 percent reduction after 20 days at 17°C when held in seawater, estuary water, river water or lake water. Hurst and Gerba (1980) recorded a 99.9 percent reduction of poliovirus 1, echovirus 7, coxsackievirus B3 and simian rotavirus in estuarine water (temperature 20°C, salinity 1.2–2.8 percent) in 2–3 days. The rate of inactivation was unrelated to salt concentration. The same virus types in clean and polluted freshwater at the same temperature were reduced by 99.9 percent in 3 to >14 days. The unimportance of salinity in determining virus survival has been shown also by Berry and Noton (1976), Matossian and Garabedian (1967) and Metcalf, Wallis and Melnick (1974).

Of particular relevance to recreational hazards are the findings of Baylor and others (1977), which show that viruses can be transferred from surf to the air and blown onto the beach. This is caused by viruses adsorbing to air bubbles as they rise through the water. When they burst at the surface, tiny droplets rich in viruses are ejected into the air and are carried on the wind. The concentration of viruses in these droplets may be at least 100 times greater than in the seawater

from which they came. This presents a potential health risk similar to that postulated for aerosol droplets produced by activated sludge plants, spray irrigation systems and flush toilets (see the section below on the occurrence and survival of enteroviruses in air).

A number of other studies (for instance, Gerba and others 1977; Hetrick 1978; Metcalf and Stiles 1968; Pietri and Breittmayer 1976; Shuval 1978; Vaughn and others 1979*a*) have investigated enteroviruses in marine and estuarine environments and have reached broadly similar conclusions. Survival in seawater is generally shorter than in freshwater, and a specific virucidal property of seawater (possibly of microbiological origin) has been postulated. The evidence for this has been reviewed by Kapuscinski and Mitchell (1980) and Katzenelson (1978). Sunlight may have some slight virucidal action on viruses suspended near the surface. Viruses are found associated with bottom sediments in a greater proportion of samples, and at higher concentrations, than in the overlying waters, and their survival is prolonged in this state (Gerba and others 1977; LaBelle and Gerba 1979; Smith, Gerba and Melnick 1978; Vaughn and Metcalf 1975). Sediment may serve as a reservoir of enteroviruses that may be resuspended into the overlying water by wind, currents, dredging or boats.

Temperature is the most determining factor in viral survival, with greatly increased inactivation rates in warmer waters (Berry and Noton 1976; Colwell and Kaper 1978). Few or no data are available on marine pollution near major tropical coastal towns (Lund 1979). However, these towns typically discharge considerable volumes of untreated or partially treated wastes into the sea, and so substantial viral pollution is to be expected. Survival times are probably shorter than those reported from temperate areas.

### *In feces and night soil*

The source of enteroviruses in the environment is the feces of infected individuals, which may contain $10^6$ or more viruses per gram. There is extensive information on the proportion of people, especially children, excreting enteroviruses at a given time. Little is known directly about the occurrence of viruses in night soil, although typical concentrations may be inferred from data on enteroviruses in feces (given here) and in sewage (given below). Enterovirus survival in feces and night soil may be estimated from data on survival in sludge (see below and the appendixes to Feachem and others 1980).

Under conditions of poverty and poor hygiene the incidence of enteroviral infections, and the prevalence of enterovirus excretion, are high. Otatume and Addy (1975) isolated enteroviruses from 44 percent of 386 fecal specimens collected from 45 healthy infants in Accra (Ghana). Virus isolation rates were not affected by seasons, were higher in urban (44 percent) than in rural (21 percent) areas, and were higher among infants from houses without flush toilets (50 percent) than among infants from houses with flush toilets (30 percent). Out of 138 typed virus isolates, 12 percent were poliovirus (presumed to be wild), 4 percent were coxsackieviruses, and the remainder were echoviruses. In Ibadan (Nigeria) three separate studies showed 39 percent of infants excreting enteroviruses or adenoviruses (Montefiore and others 1963), 49 percent of children under 3 years excreting enteroviruses other than polioviruses (Poliomyelitis Commission 1966), and 45 percent of children between 3 and 24 months excreting enteroviruses (Peradze, Montefiore and Coker 1968). In Yaounde (Cameroon), 39 percent of 524 children age 0–6 years were excreting enteroviruses, and 11 percent were excreting polioviruses (Boche, Millan and Le Noc 1973). Sabin and others (1960) found that 11 percent of children 1–5 years old in Toluca (Mexico) were excreting wild poliovirus, while 51 percent were excreting other viruses. Rao, Lakhe and Waghmare (1978) reported that 45 percent of children between 1 and 15 years old in Nagpur (India) were excreting enteroviruses. These data on the prevalence of enterovirus excretion among children in Africa, Asia, and Central America show a quite remarkable consistency.

Another gauge of the very high incidence of enterovirus infections among young children in developing countries is data on the proportion of unvaccinated children having antibodies to poliovirus. For instance, John and Jayabal (1972) found that 79 percent of 191 unvaccinated infants and children under 5 years old in Vellore (India) had antibodies to one or more types of poliovirus, thus indicating a history of infection. Sabin and others (1960) found that 100 percent of children in Toluca (Mexico) developed antibodies to one or more polioviruses before the age of 4 years, and that over 90 percent of 4 year olds had antibodies to all three polioviruses. Montefiore and others (1963) reported that 100 percent of children over 3 years in Ibadan (Nigeria) had immunity to poliovirus 1. In a subsequent study in Ibadan (Poliomyelitis Commission 1966), the prevalence of immunity to polioviruses 1, 2, and 3 in children between 24 and 36 months old was 68, 48, and 68 percent respectively. Studies in Kabul (Afghanistan) showed that over 90 percent of unvaccinated children had acquired immunity to polioviruses by the age of 5 and that peak

transmission occurred in the early summer (Šerý and others 1970; Thraenhart and others 1970).

By contrast, enterovirus isolation rates from healthly individuals in industrialized countries are much lower. Froeschle, Feorino and Gelfand (1966) isolated enteroviruses (excluding polioviruses, which they assumed to be vaccine derived) from 4.9 percent of healthy 1–5 year old children in six cities in the USA. Isolation rates peaked in late summer and early fall with a maximum of 12.4 percent positive in September. Infection rates were higher in males than in females and highest in 1 year olds, with rates decreasing with increasing age. Cooney, Hall and Fox (1972) studied over 14,000 fecal specimens in Seattle (USA) and found polioviruses (presumed to be vaccine derived) in 8.8 percent, adenoviruses in 2.2 percent, coxsackieviruses in 0.7 percent, and echoviruses in 0.6 percent. From 18,000 respiratory specimens, polioviruses were isolated in 0.8 percent, adenoviruses in 0.9 percent, coxsackieviruses in 0.1 percent, and echoviruses in 0.5 percent. Isolation rates were inversely related to age.

In summary, it is clear that enteroviruses spread vigorously by the fecal-oral route (and possibly also by the oral-oral route) in conditions of poverty and low personal and domestic hygiene. Thus the prevalence of enterovirus excretion is high (40–50 percent among infants and young children), as is the prevalence of antibodies to enteroviruses, which indicate past or current infection. Under conditions of relative affluence and optimal hygiene (as in the USA) wild enteroviruses, and vaccine-derived polioviruses, continue to circulate in the community and among members of the same family. However, the prevalence of enterovirus excretion is very much lower (around 5 percent in young children) than in the developing countries. In all countries studied, the prevalence of virus excretion is inversely related to age, so that the highest virus excretion rates are found among infants. As hygiene improves in the absence of vaccination programs, the age distribution of infections tends to shift upwards, and the amount of clinically serious disease may increase (see, for instance, Anon. 1971, Hillis 1979, Metselaar 1968).

### *In sewage*

Since enteroviruses are not normally excreted for prolonged periods by healthy individuals, their occurrence in sewage is subject to wide fluctuations. However, nearly all sewages contain enteroviruses, and the larger the contributing population the less variable is the concentration of viruses. Communities with poor hygiene and a high proportion of children will produce a sewage especially rich in enteroviruses, and reported concentrations of enteroviruses will continue to rise as laboratory techniques improve and as more studies are carried out in developing countries. Sewage in developing countries must be assumed to contain at least $10^5$ enteroviruses per liter, and sewage effluents produced by conventional treatment plants will also contain high concentrations of enteroviruses (see the section below on enterovirus inactivation by sewage treatment processes).

In an early study of this subject, Bloom and others (1959) investigated the enteroviruses in the sewage of Lansing and East Lansing (Michigan, USA) between 1955 and 1957. East Lansing sewage yielded enteroviruses in 14 percent of samples, compared with 7 percent in Lansing, which had large volumes of industrial wastes in its sewage. Peak isolations occurred during July through November. Thirty-three percent of samples of influent at the East Lansing activated sludge plant were positive for enteroviruses, while only 10 percent of samples of the final effluent were positive.

Haifa (Israel) sewage between 1972 and 1974 contained a monthly average of between $6 \times 10^3$ and $4.9 \times 10^5$ viruses per liter. The highest value recorded was $1 \times 10^6$ viruses per liter (Buras 1976). Fattal and Nishmi (1977) reported a predominance of polioviruses amongst enteroviruses isolated from the sewage of six Israeli towns and found that 13 percent of isolated polioviruses were wild strains rather than attenuated vaccine strains. By contrast, Katzenelson and Kedmi (1979) reported detecting poliovirus in only about 50 percent of 25 samples of raw sewage and sewage effluents in Jerusalem, Tel Aviv and elsewhere in Israel. The proportion of polioviruses to all enteroviruses was low.

In Seattle (USA) Heyward and others (1979) isolated up to $1.3 \times 10^3$ viruses per liter from combined sewer-stormwater overflows. In Ottawa (Canada) Sattar and Westwood (1977) detected pathogenic viruses in 79 percent of sewage samples. Of 72 isolates identified, 56 (78 percent) were reoviruses; the remaining 16 were enteroviruses. Of the 16 enteroviruses, 1 was coxsackie, 10 were vaccine strains of poliovirus, and 5 were wild polioviruses. The authors point out that the presence of these wild strains in sewage, at a time when immunity against poliovirus is declining due to a fall-off in vaccination, is a cause for concern. Fujioka and Loh (1978) isolated enteroviruses from 100 percent of raw sewage samples investigated in Hawaii (USA) at concentrations between 27 and $1.9 \times 10^4$ per liter. In the same study, 26 poliovirus isolates were assayed for virulence and 3

(all from chlorinated effluent) were found to be wild.

Rao, Lakhe and Waghmare (1978) reported virus concentrations of up to 11,500 per liter in Indian raw sewage. There was a pronounced diurnal and seasonal variation in virus load; maximum concentrations occurred between 8 and 10 a.m. and during the rainy season. Nearly 80 percent of viruses isolated were polioviruses and 60–80 percent of these were wild strains. The authors noted that about 60 percent of recorded cases of paralytic poliomyelitis cases in India are reported during the rainy season.

Virus survival in sewage has been investigated in several studies (see the appendixes of Feachem and others 1980). The results generally indicate longer survivals than in river water, with survival times of over 231 days at cool temperatures ($<10$°C) and up to 110 days at warmer temperatures (20°C). Lefler and Kott (1975) showed 99.9 percent reduction of poliovirus in sewage, after 42 days at 18–25°C, and after 231 days at 4–8°C. Kott, Ben-Ari and Vinokur (1978) reported the complete disappearance of enteroviruses in lagooned trickling filter effluent within 73 days, and the disappearance of poliovirus in oxidation pond effluent at 18–23°C within 110 days (99.9 percent reduction in about 70 days). Rao and others (1977) found up to 1,250 enteroviruses per liter in Bombay sewage and report that, when stored at 8°C for 2 days, a 22 to 40 percent loss occurred.

Prolonged survival of enteroviruses in sewage may be due in part to the protective effects of adsorption to solids. Wellings, Lewis and Mountain (1976) found that between 16 and 100 percent of viruses in raw sewage and sewage effluent, at two treatment plants in Florida (USA), were solids associated. Gerba, Stagg and Abadie (1978) reported that 3 to 49 percent of viruses were solids associated in treatment plant effluents near Houston (Texas, USA).

In a unique study Ruiter and Fujioka (1978) investigated the sewage produced by two communities in Honolulu (Hawaii, USA). Kuhio Park Terrace had a total population of 2,745, of whom 46 percent were children under 14 years. The density of settlement was 376 persons per hectare, 73 percent of households had an income of under US$5,000 per year, and 23 percent of heads of household were unskilled laborers. Nuuanu had a total population of 2,302, of whom 10 percent were children under 10 years. The density of settlement was only 19 persons per hectare, 4 percent of households had an income of under US$5,000 per year, and 4 percent of household heads were unskilled laborers. Kuhio Park Terrace produced a sewage with an average of 345 enteroviruses per liter (maximum 820 per liter), while Nuuanu produced an average 93 enteroviruses per liter (maximum 218 per liter). This difference was attributed to the differences in socioeconomic status and age structure between the two communities. Enteroviruses in sewage reflect the levels of infection and vaccination within the contributing population and may be used as an aid to epidemiological surveillance (see, for instance, Zdražílek, Šrámová and Hoffmanová 1977).

Little information is available on the concentration of enteroviruses in tropical sewage. It is to be expected that poor communities, living in conditions of inadequate hygiene, will produce a sewage with $10^7$ or more infectious virus units per liter (Lund 1979), although, of course, most such communities produce no sewage at all because they are not connected to a sewerage system. However, even fairly affluent communities in developing countries will almost certainly produce a sewage with a greater concentration of pathogenic viruses than in Europe and North America, because the incidence of viral infections is higher, water use is lower, and a greater proportion of the population is under 15 years old (see the previous section on viruses in feces).

### *In sludge*

The sludges of sewage works are rich in enteroviruses because a high proportion of viruses in sewage are, or become, solids associated and are therefore concentrated into both primary and secondary sludges (Lund 1973, 1976; Lund and Ronne 1973; Wellings, Lewis and Mountain 1976). It is probable that most of the difference between the virus concentration of the influent and the effluent of a sewage treatment plant is accounted for by the viruses in the sludge. The few studies on virus survival in sludge are listed in the appendixes to Feachem and others (1980).

Subrahmanyan (1977) studied the survival of several types of enteroviruses added to sewage sludge at a concentration of $10^7$ per liter and kept at 22°C. Survival times ranged from a minimum of 2 weeks (coxsackievirus A9) to a maximum of over 12 weeks (coxsackievirus B5 and echovirus 9). Polioviruses survived from 8 to 12 weeks. Damgaard-Larsen and others (1977) inoculated digested and dewatered sludge with coxsackievirus B3 at a concentration of $10^6$ per gram. The sludge was applied outdoors in Denmark to sandy and clay soils during December to May, when rainfall totalled 300 millimeters and temperatures ranged from $-12$ to 26°C. Virus inactivation took place at a rate of about 1 log unit per month, and it took 23 weeks before viruses could not be

detected. Viruses remained bound to the sludge and did not travel downward through the soil. Nielsen and Lydholm (1980) reported the survival of naturally occurring coxsackievirus B5 for 4 months (March–July) in digested sludge applied to land in Denmark.

Sattar and Westwood (1979) studied the viruses present in raw sludge (5 percent solids), anaerobically digested sludge (20 days at 35°C), and lagoon-dried sludge (minimum of 6 months drying time) from the largest sewage treatment plant in Ottawa (Canada). Excreted viruses were isolated from 84 percent of raw sludge samples, 53 percent of digested sludge samples, and 39 percent of dried sludge samples. Viruses were isolated from sludge that had been drying for over 8 months. Most virus isolates were reoviruses, the remainder being enteroviruses.

Hurst and others (1978) isolated viruses from various activated sludge samples at a sewage treatment plant at Houston (Texas, USA) in concentrations of around 30 viruses per liter of sludge. After sludge thickening, aerobic digestion and centrifugation the concentration of viruses rose to 231 per liter. This sludge was then applied to land, where the virus concentration was monitored over two separate periods. During the first 7 day period (during September with no rain), the solids content of the sludge rose from 6.9 to 18.4 percent, and 97 percent of the viruses were inactivated. During the second 7 day period (during September with rain on day 6), the solids content of the sludge stayed almost constant at 13–14 percent, and 99.5 percent of the viruses were inactivated. A sample of sludge which had been on the field for 3 months had a solids content of 59 percent and no demonstrable virus. Thus in a Texas summer a 2 log reduction per week was recorded, compared with a unit log reduction per month during a Danish winter (Damgaard-Larsen and others 1977).

*In soil*

Increased interest in the health aspects of the agricultural use of sewage and sludge has generated several studies on virus survival in soil. However, little information is yet available. More is known about virus travel and adsorption in soils, and this subject is reviewed below in the section dealing with land treatment.

A review by Gerba, Wallis and Melnick (1975*a*), and the studies listed in the appendixes to Feachem and others (1980), indicate that survival times of over 175 days are possible. Hurst, Gerba and Lance (1979) found that enterovirus survival in soil was prolonged by low temperatures but was unaffected by whether the soil was wetted with distilled water or with various concentrations of sewage effluent. Virus inactivation occurred much more rapidly under nonsterile aerobic conditions than under sterile aerobic conditions or under sterile and nonsterile anaerobic conditions. Duboise, Moore and Sagik (1976) found that polioviruses held for 84 days in loamy sand were reduced by less than 90 percent at 4°C but by 99.999 percent at 20°C. In studies into the disposal of septic tank effluent it was found that poliovirus 1 adsorbed to sandy soils was reduced by 97.5 percent after 28 days at 20°C and by less than 50 percent after 56 days at 7°C (Small Scale Waste Management Project 1978).

Lefler and Kott (1974*a*) studied the survival of poliovirus 1 in sand kept in the dark at room temperature (18–22°C). With the sand saturated in distilled water, no poliovirus was detected after 112 days, and 99 percent inactivation was achieved in about 63 days. With tap water or oxidation pond effluent, complete inactivation took 105 days, while 99 percent reduction took 42 days. Coliphage f2 survived for longer than poliovirus 1. When the saturated sand was kept at 4–8°C, 20 percent of poliovirus was still active after 175 days. On dried sand at 4–8°C, 96 percent inactivation occurred in 21 days and virus was still detectable (at 0.02 percent of the original concentration) after 77 days.

Tierney, Sullivan and Larkin (1977) inoculated poliovirus 1 into samples of activated sludge and secondary effluent to produce a viral concentration of $2.5 \times 10^8$ viruses per liter. The fluids were sprayed over soil plots so as to flood them to a depth of 25 millimeters. Runoff water from the plots contained $10^6$ viruses per liter on the day of flooding but this fell to 0 by day 6. In winter ($-14$ to 27°C) viruses applied in effluent survived in soil for between 89 and 96 days, whereas viruses applied in sludge survived between 96 and 123 days. In early summer (19°C to 34°C), viruses were not detected for more than 11 days after flooding with either effluent or sludge.

Yeager and O'Brien (1979*a*) elucidated several aspects of virus survival in soil. They found that poliovirus survival was heavily temperature dependent, with virus survival in saturated soil being up to 12 days at 37°C, up to 92 days at 22°C, and up to 180 days at 4°C. Viruses survived longer in soils saturated with septic tank liquor (90 percent reduction in 8–21 days at 22°C) than in soils saturated with river or groundwater (90 percent reduction in 5–7 days at 22°C). Viruses survived for longer in sandy loam (90 percent reduction in 6–21 days at 22°C) than in sand (90 percent reduction in 4–8 days at 22°C). Soil drying

was found to be highly virucidal, irrespective of soil type, and speed of soil drying depended on temperature. Soil moistures of below 2.9 percent appeared to be especially virucidal. In an accompanying study (Yeager and O'Brien 1979*b*) the same authors investigated the nature of virus inactivation in soil. They concluded that loss of infectivity is due to irreversible damage to the virus particles. They speculate that two general mechanisms may underly the inactivation of picornaviruses in the environment: under temperate, saturated conditions RNA degradation may occur, whereas in dried soil (and also perhaps in aerosols and under a variety of hostile circumstances such as heat, irradiation and desiccation) the virions may dissociate into intact RNA and isoelectrically altered capsids (see also O'Brien and Dacus 1978).

Virus survival in soil depends upon many factors including the type of soil, its pH, the sterility of the soil, and the type of liquid in which the viruses are applied. However, temperature and moisture appear to be the dominant factors. While very long virus survival times (over 5 months) are possible at cool temperatures ($<10°C$), at warmer temperatures ($>25°C$) viruses are likely to be eliminated within 2 weeks. Soil drying is also highly virucidal, and the evidence suggests that, in warm climates, intermittent agricultural application of sewage, night soil or sludge, with drying periods of 3–5 days between applications, would result in little or no build-up of viable pathogenic viruses in the soil. This contention is further supported by the studies of Sadovski and others (1978)—reported in the next subsection, concerning viruses on crops.

*On crops*

The interest in virus survival in soil has been accompanied by interest in virus occurrence and survival on vegetables fertilized or irrigated with sludge or sewage.

Konowalchuk and Speirs (1975*a*) studied virus survival on vegetables stored at 4°C. In a humid atmosphere, coxsackievirus, poliovirus, echovirus, reovirus, and adenovirus inoculated in a water droplet onto lettuce, celery, green peppers, tomatoes, radish, and carrots were undetectable after 4–5 days. When inoculated in dilute feces, 4–5 percent were detectable after 5 days; when inoculated in feces, 7–12 percent were detectable after the same time. Additional studies on virus survival in vegetable infusions indicated that the vegetables contained no antiviral agents. In a later study Konowalchuk and Speirs (1975*b*) investigated the survival of various viruses on strawberries, cherries, and peaches at 4°C and investigated the effect of inoculating the viruses in water or in dilute feces and of storing the fruit in humid or dry atmospheres. Viruses were inactivated far more rapidly under dry than under humid conditions, and virus survival was prolonged by inoculation in feces rather than in water. Viruses survived longer on cherries and peaches than on strawberries, and coxsackieviruses and echoviruses survived for longer than polioviruses and reoviruses. Under dry conditions survival of viruses was between 40 and $<1$ percent after 1–2 days, and viruses were undetectable after 4–6 days. The short survival times are notable in view of the cool temperature (4°C), and the authors consider that an antiviral substance produced by the fruit is active. The authors also consider that the longer survival of viruses inoculated in dilute feces is due to delayed desiccation as compared with viruses inoculated in water. Subsequently, the same authors (Konowalchuk and Speirs 1977) reported that the concentration of poliovirus 1 and coxsackievirus B5 was reduced by about 99 percent after 5 days on grape bunches hung indoors at 22°C.

Kott and Fishelson (1974) investigated the effect of effluent chlorination and sunlight on the survival of seeded poliovirus 1 on tomatoes and parsley. The maximum recoveries of polioviruses from vegetables 6 hours after application in waste stabilization pond effluent were: 2.2 percent when applied in unchlorinated effluent with exposure to sunlight, 1.6 percent when applied in chlorinated effluent with exposure to sunlight, 12.7 percent when applied in unchlorinated effluent and kept in darkness, and 8.5 percent when applied in chlorinated effluent and kept in darkness. Poliovirus applied outdoors did not survive for more than 1 day on tomatoes or 2 days on parsley at 15–31°C. Poliovirus survival was considerably prolonged when viruses were applied in phosphate-buffered saline rather than effluent.

Larkin, Tierney and Sullivan (1976) planted lettuces and radishes and, 8–10 days later, sprayed them with secondary effluent or sludge seeded with $2.5 \times 10^8$ polioviruses per liter. These experiments were conducted in Ohio (USA) in the summer of 1973 and 1974 when there was extensive direct sunlight, periodic rain, air temperatures of 19–34°C, and soil surface temperatures rising to 45°C. On the days immediately after spraying, large amounts of virus (up to $2.9 \times 10^4$ per 100 grams) could be recovered from the vegetables. Two weeks after spraying no more than 100 viruses per 100 grams could be detected, and small numbers (10 per 100 grams) persisted for at least 36 days. In similar

studies, Tierney, Sullivan and Larkin (1977) were still able to isolate 60 polioviruses per 100 grams of lettuce 23 days after spraying. (Lettuces and radishes in this climate are normally harvested 3 to 4 weeks after planting.)

Sadovski and others (1978) studied the survival of polioviruses inoculated into waste stabilization pond effluents and applied by drip irrigation to cucumber plots on two farms in Israel. At one site (air temperature 13–30°C, soil temperature at noon 22–30°C, sunlight 9.5 hours per day, relative humidity 27–55 percent), a single irrigation was performed with inoculated effluent containing $9 \times 10^7$ polioviruses per liter. Viruses were still detectable in the irrigation system, at a concentration of $>10^4$ per liter, 8 days after the flow of inoculated effluent. The soil contamination immediately after irrigation with inoculated effluent was $10^4$ viruses per 100 grams of soil (dry weight) and persisted at a level of $>10^3$ per 100 grams for at least 8 days. Cucumbers grown in exposed soil were contaminated by $2.2 \times 10^3$ viruses per 100 grams immediately following irrigation, and this contamination fell to 30 per 100 grams on day 8. However, when the soil and drip lines were covered with plastic sheets no viruses could be isolated from the cucumbers after a few hours had elapsed after inoculated irrigation. At the second site (air temperature 23–28°C, soil temperature at noon 40–43°C, sunlight 11.8 hours per day, relative humidity 62–70 percent), three irrigations were performed with inoculated effluent containing $2.2 \times 10^{12}$ polioviruses per liter. After the third inoculated irrigation the soil contained $9.1 \times 10^3$ viruses per 100 grams (dry weight), and this contamination fell to 47 per 100 grams after 10 days and to 0 after 15 days. Unlike at the first site, where irrigation with uninoculated effluent had continued throughout the study, in this case irrigation terminated 5 days after the third inoculated irrigation, and consequently the soil moisture content fell from 15 to 3 percent. Virus contamination of the cucumbers grown in exposed soil rose to 0.13 per 100 grams, but was undetectable 6 days after the last inoculated irrigation. In covered soil no viral contamination of the vegetables could be detected. At both sites, virus survival in the soil was unaffected by whether plastic sheets were lain over the soil and the drip lines. Earlier studies at the first site by the same workers (Sadovski, Fattal and Goldberg 1978) showed that no viruses could be isolated from cucumber or eggplants drip-irrigated with uninoculated waste stabilization pond effluent containing $10^3$ enteroviruses per liter.

These studies, and others listed in the appendixes of Feachem and others (1980)[4], indicate highly variable survival times for enteroviruses on vegetables and fruit. Type of virus and type of crop are clearly important, but the dominant factors are temperature, sunlight and humidity (which will control the degree of warming), and radiation and desiccation experienced by the viruses. Survival times of up to 2 months are possible, but at day temperatures above 25°C (and especially in dry climates) one may anticipate negligible survival of enteroviruses on crops for more than 2 weeks. Indeed, it may be that almost complete elimination will occur in under 5 days and that the longer survival times reported by some investigators are only achieved by the untypically high concentration of seeded enterovirus in the applied effluent [for instance Larkin, Tierney and Sullivan (1976) employed $2.5 \times 10^8$ polioviruses per liter, and Sadovski and others (1978) had $2.2 \times 10^{12}$ per liter]. It has been clearly demonstrated by Israeli workers that drip irrigation, particularly when combined with soil covered with plastic sheets, is a method of effluent application that minimizes the risks of crop contamination by enteroviruses.

### *In fish and shellfish*

The primary hazard associated with estuarine and marine discharge of fecal wastes may not be risks to bathers and water sportsmen but to those who eat the fish and shellfish that are harvested in polluted waters. The greatest risks of viral infection are associated with the ingestion of contaminated molluscs (such as oysters, mussels, cockles, and clams) and crustacea (such as crabs, lobsters, shrimps, and prawns) in a raw or partially cooked state. Most attention has focussed upon oysters because they are commonly eaten raw and their method of filter feeding (common to all bivalve molluscs) concentrates pathogenic organisms from the water into their tissues (Anon. 1976; Gerba and Goyal 1978; Hughes, Merson and Gangarosa 1977; Metcalf 1978; Wood 1979). Attention has also been paid to improved laboratory techniques for isolating enteroviruses from shellfish (Gerba and Goyal 1978; Metcalf 1978; Vaughn and others 1979*b*).

Mitchell and others (1966) placed 600 eastern oysters (*Crassostrea virginica*) in seawater at 20°C containing $10^6$ polioviruses per liter and found that viruses rapidly accumulated in the oyster tissue such that after 1 hour the virus concentration in the oyster was 27 times higher than in the surrounding water.

4. See also Engley (1956) and Berg (1978*b*) for reviews of literature on virus survival in food (as opposed to on crops).

When the seawater contained $3 \times 10^5$ polioviruses per liter, viruses accumulated less rapidly but were always concentrated in the oyster by at least 10-fold after 3 hours. When the oysters were rinsed and placed in sterilized seawater, over 95 percent and 99.9 percent of viruses were eliminated after 8 and 24 hours respectively. Viruses were sometimes undetectable after 48 hours and sometimes detectable in very small numbers (2 per gram) for up to 96 hours.

Hoff and Becker (1969) studied the accumulation of poliovirus by the Olympia oyster, the Pacific oyster, and the Manila clam and found that these species concentrated the virus to a level between 10 and 180 times higher than in the surrounding waters. When the contaminated shellfish were held in disinfected seawater (6–16°C), poliovirus concentrations in the meat were reduced by at least 99.9 percent after 96 hours. Hedstrom and Lycke (1964) found that poliovirus survived for 3.5 days in seawater but for well over 6 days in oysters in contaminated seawater. Oysters did not cleanse themselves of poliovirus within 22 hours when transferred to uninfected water or to water containing up to 1.7 milligrams per liter of free chlorine.

DiGirolamo, Liston and Matches (1975) placed oysters in seawater (salinity 2.8 percent, temperature 13°C) containing $1.9 \times 10^7$ polioviruses per liter and found that after 2 days the oysters had accumulated about $10^4$ polioviruses per gram of meat. Most viruses were concentrated in the digestive organs and feces. When placed in stationary sterilized seawater (salinity 2.8 percent, temperature 13°C), contaminated oysters lost between 79 and 84 percent of polioviruses in 5 days. When placed in flowing sterile seawater, oysters lost over 99 percent of accumulated poliovirus after 3 days. Gerba and Goyal (1978) reviewed 17 other studies on the accumulation and depuration of excreted viruses by shellfish.

The isolation of enteroviruses from oysters living in lightly contaminated waters has been frequently reported. Metcalf and Stiles (1965) isolated coxsackie B4 and echo 9 viruses from oysters in estuary waters at distances of up to 4 miles from the nearest sewage outlet. Vaughn and Metcalf (1975) found that 7.6 percent of oyster samples contained enteroviruses in waters from which only 5.6 percent of samples were positive for viruses. Vaughn and others (1979*a*) isolated up to 30 viruses per 100 grams of flesh from clams in Great South Bay (New York, USA), the waters from which contained no more than 2 viruses per liter.

Goyal, Gerba and Melnick (1979) studied the presence of enteroviruses in oysters and oyster-harvesting waters in Galveston Bay (Texas, USA). Of a total of 44 water samples, 26 yielded viruses in concentrations of up to 0.4 per liter, whereas of 40 pools of 10 to 12 oysters each, viruses were isolated from 14 pools at concentrations of up to 224 per 100 grams. On five occasions viruses were found in oysters but not in the overlying waters. Gerba and Goyal (1978) reviewed 17 reported isolations of viruses from shellfish.

It is generally agreed that no human enterovirus multiplication takes place in shellfish (Chang and others 1971) and that the dangers lie in the uptake, concentration, and survival of viruses in shellfish tissue. Uptake, depuration, and survival in oysters has been found to be temperature dependent. Below a given temperature, a particular species of shellfish will cease to filter. The European flat oyster (*Ostrea edulis*) appears to filter at temperatures down to 5°C, whereas the eastern oyster (*Crassostrea virginica*) will not filter below 7°C. The edible mussel (*Mytilus edulis*) filters at temperatures down to 2°C, but the hard clam (*Mercenaria mercenaria*) ceases active filtration at about 12°C (Metcalf and Stiles 1968; Wood 1979). As the temperature falls, the rate of filter feeding declines and so does the rate of accumulation of viruses. Below the critical temperatures mentioned above, virus accumulation should cease. The same is true of depuration, and so a contaminated shellfish will cleanse itself more slowly as temperature falls and will cease to cleanse itself at all below a critical temperature.

Survival of viruses in stored shellfish is very much prolonged by low temperatures. Vaughn and Metcalf (1975) reported that coxsackievirus B3 in oysters survived for up to 42 days at 1–11°C, but for only 22 days at 14–21°C. Metcalf and Stiles (1965) found that viruses in oysters stored at 5°C remained relatively stable for at least 28 days. DiGirolamo, Liston and Matches (1970) found that 10 percent of polioviruses survived for 84 days in oysters after freezing at −36°C and storage at −17.5°C. DiGirolamo and Daley (1973) froze crabs at −20°C and found that 17–35 percent of seeded coliphage T4 survived after 30 days. Other studies of viruses in refrigerated shellfish have shown that survival times of up to 120 days are possible (Gerba and Goyal 1978).

These and many other studies (reviewed in Gerba and Goyal 1978) show that viruses in water are readily accumulated by shellfish. In edible bivalve molluscs (clams, mussels, oysters) the viruses are concentrated mainly in the digestive system and may be present in concentrations over 100-fold higher than in the surrounding waters. This is because these shellfish are filter feeders, and one oyster may filter as much as 1,500

liters of seawater per day in its quest for food. Crustacea that inhabit polluted waters or that feed on contaminated molluscs may also accumulate enteroviruses, although less work has been done on this. DiGirolamo and others (1972*a*) showed that crabs kept in contaminated seawater for 2 days at 10°C, or allowed to feed on contaminated mussels for 12 hours, accumulated over $10^3$ polioviruses per gram of meat.

Depuration or cleansing of viruses from shellfish is a mechanical process induced by the filter feeding of the mollusc in clean water. Maximum depuration occurs when feeding activity is greatest. Thus, cleansing is more rapid at warmer temperatures, at optimal salinities, and in flowing water. Depuration of commercially harvested oysters by placing them in clean water is practiced. Chlorination of the water to maintain its purity has been advocated, but this is antagonistic to the oyster, inhibits feeding, and thus delays depuration. Preliminary experiments have been conducted on virus removal from contaminated seawater prior to aquaculture by adsorbing viruses to magnetite and removing them in a magnetic field (Bitton and others 1977).

When contaminated shellfish reach the market, the risks are obviously greatest if they are eaten raw. However, a residual risk remains even with cooked shellfish. Studies have shown that a proportion of polioviruses in oysters survived stewing (after 8 minutes, 10 percent survived), frying (after 10 minutes, 13 percent survived), baking (20 minutes, 13 percent survived), steaming (30 minutes, 7 percent survived), and irradiation (4 kilogray,[5] 7–13 percent survived) and that up to 20 percent of coliphage T4 in crabs survived boiling (DiGirolamo, Liston and Matches 1970, 1972; DiGirolamo and others 1972*b*).

Nearly all the documented disease outbreaks associated with excreted virus contamination of shellfish are outbreaks of hepatitis A (see chapter 10) or viral gastroenteritis (see chapter 11). These outbreaks have been reviewed by Gerba and Goyal (1978) and Levin (1978). However, as reported above, most studies on viruses in shellfish have focussed on the enteroviruses because it is for these viruses that well-developed laboratory isolation methods exist. Before the development of isolation techniques for hepatitis A virus and rotavirus, it must be assumed that these viruses behave in shellfish similarly to the enteroviruses.

5. The SI unit of radiation dose is the Gray (Gy), which is equal to 100 rads and is the equivalent of 1 joule per kilogram.

*In the air*

Airborne droplets of water and wastewater may contain enteroviruses, and these viruses may cause infection when inhaled. Droplets containing viruses may be formed by the flushing of a toilet, by spray irrigation, or by any occasion in which bubbles are rising through contaminated waters and bursting at the surface (such as activated sludge plants, waves and surf, or the passage of boats). As a bubble rises through water, viruses become adsorbed to its surface. The bubble bursting at the surface ejects a tiny jet of water that breaks into many droplets, and these droplets contain most of the viruses that were adsorbed to the bubble. Thus the droplets contain a much higher concentration of viruses than the water from which they came.

Baylor, Peters and Baylor (1977) bubbled air through a column of liquid containing coliphages T2 and T4 and produced droplets that contained a concentration of phage 50 times that in the column. Baylor and others (1977) seeded the breaking surf with coliphages T2 and T4 at beaches near New York. Droplets were formed in the surf that contained a concentration of phages 100 to 250 times higher than the seawater, and these droplets were carried by the wind for at least 30 meters.

The aerosolized excreted viruses most encountered by people in developed countries are those produced by the flush toilets in their houses. Gerba, Wallis and Melnick (1975*b*) seeded $10^8$ polioviruses into a toilet bowl and found that flushing ejected at least $2.8 \times 10^3$ infectious units to the level of the seat. Further experiments with seeded coliphage MS2 showed that these organisms remain airborne long enough to settle out in large numbers on surfaces throughout the bathroom, and presumably also to be inhaled by people in the bathroom. In an unventilated bathroom, 94 percent of recovered coliphage had settled out within 2 hours of the flush, and most of the remainder had settled within 4 hours. Small numbers of viruses could apparently remain airborne for much longer.

Fannin and others (1977) studied airborne viruses produced by a trickling filter plant and an activated sludge plant in Michigan (USA). The naturally occurring level of animal viruses in sewage at the trickling filter and activated sludge units was 100 per liter, while the coliphage concentration was $5 \times 10^5$ per liter. No animal viruses were recovered from air samples at the plants, but coliphage was recovered at concentrations up to 0.5 per cubic meter of air. Airborne coliphage recovery was correlated with relative humidity (higher humidity associated with

higher recovery) but was not correlated with wind speed or ambient temperature (see also Cochran and Fannin 1976 and Fannin and others 1976). Earlier laboratory studies by de Jong and Winkler (1968) had shown that the inactivation of poliovirus 1 during spraying was greatest at low humidities.

Sorber, Schaub and Bausum (1974) developed a theoretical model of the transmission of viruses in aerosols produced by spray irrigation with effluents containing various enterovirus concentrations. The model indicates that a healthy young male working at the wetted perimeter when strong effluent (6,000 viruses per liter) is being sprayed may inhale as many as 240 viruses in 10 minutes; whereas if he is working 200 meters from the wetted perimeter when weak effluent (10 viruses per liter) is being sprayed, he may inhale only 0.0006 viruses in 10 minutes. These findings depend upon assumptions made about climatic conditions. The authors conclude that spray irrigation with chlorinated effluents from conventional treatment plants poses considerable risk of virus inhalation and that better virus removal systems need to be applied prior to spray irrigation.

Moore, Sagik and Sorber (1979) were able to isolate small numbers of coliphages (up to 1.5 per cubic meter of air) and enteroviruses (up to $1.7 \times 10^{-2}$ per cubic meter of air) from large volumes of air sampled 50 meters downwind of the wet-line edge of a wastewater spray irrigation site in California (USA). Teltsch and Katzenelson (1978) isolated echoviruses from 4 out of 12 air samples collected 40 meters downwind of wastewater sprinklers in Israel. Bausum, Schaub and Kenyon (1978) studied a spray-irrigated golf course in Arizona (USA) and isolated seeded coliphage f2 from aerosol droplets 563 meters downwind of sprinklers delivering secondary effluent and 137 meters downwind of sprinklers delivering chlorinated effluent.

The possibility of virus transmission via aerosol droplets will undoubtedly be the subject of a considerable amount of research over the next few years. Attention will focus upon risks to workers at sewage treatment facilities and at agricultural sites employing spray irrigation with wastewater. A study from Israel (Katzenelson, Buium and Shuval 1976) showed that the populations of kibbutzim that practiced spray irrigation with waste stabilization pond effluent had a higher incidence of hepatitis A infection than kibbutzim in which no form of wastewater irrigation was used. However, it is most unlikely that transmission by aerosol droplets plays any major part in the maintenance of endemic enteroviral infections in poor communities where basic hygienic facilities are lacking.

## Inactivation by Sewage Treatment Processes

The realization that raw sewage is rich in pathogenic viruses, and recent advances in laboratory techniques (for instance, Lydholm and Nielsen 1980), have given rise to many investigations into the effectiveness of various treatment processes in reducing viral concentrations in sewage effluents. These studies have almost exclusively examined the removal of enteroviruses and coliphages, and these two groups of viruses do not always behave in a similar way (nor may they be good models for rotavirus or hepatitis A virus). Several reviews have been published (for instance Berg 1973; Sproul 1976; WHO 1979).

### *By primary and secondary sedimentation*

Primary sedimentation tanks, with retention times of 2–6 hours, allow a proportion of the viruses in the sewage to adsorb onto solids and settle. Many viruses will already be adsorbed to settleable solids in the influent. Removals reported in the literature, listed in the appendixes of Feachem and others (1980), suggest between 0 and 83 percent removal from influent to effluent.

Rao and others (1977) recorded a 24 to 33 percent removal of enteroviruses by primary settling tanks in the wet season in Bombay. At other times of the year removal was between 41 and 83 percent with a 2-hour retention time. Rao, Lakhe and Waghmare (1978) reported a 50 percent reduction of viruses in a pilot plant settling tank at Nagpur (India). Sherman and others (1975) and Naparstek and others (1976) studied removal of seeded coliphage f2 in treatment plants in Maryland (USA) and found 35–47 percent average removals in primary sedimentation tanks and 30 percent removal during secondary sedimentation. One report suggests that factors other than settlement may be operative in removing viruses from sedimentation tank effluent (Clarke and others 1961).

Similar performance may be expected from secondary sedimentation tanks, except that these are often designed with higher overflow rates. The sludge removed from sedimentation tanks will normally contain a 10–100 times higher concentration of enteroviruses than the raw sewage.

### *By storage*

Storage is an effective method of virus inactivation, especially at temperatures above 20°C. In any storage vessel, some sedimentation will also be taking place

that will remove a proportion of viruses to the sludge layer. Expected removal rates in stored sewage may be derived from the data given above on the survival of enteroviruses in sewage (see also the appendixes of Feachem and others 1980), although little is known about survival under tropical climatic conditions.

*By septic tanks*

Removal of enteroviruses by septic tanks has been very little studied, and not at all in developing countries. A septic tank is simply a settling chamber (or chambers) with a mean retention time of 3 days or less. In poorly designed tanks, or those requiring desludging, there is very considerable carryover of solids into the effluent. Viruses will be removed both by inactivation in the anaerobic liquor and by adsorption to solids that settle to the sludge layer. Some studies of enterovirus removal have been conducted, and estimates may also be derived from information on survival in sewage and on removal by primary sedimentation (see the appendixes to Feachem and others 1980). A series of laboratory experiments showed that a 99 percent reduction of poliovirus 1 in septic tank effluent took 14 days at 20°C and 43 days at 7°C (Small Scale Waste Management Project 1978). Therefore, if all influent was held for 3 days at 20°C (because of short circuiting, it never is), a 64 percent virus reduction might be expected. In practice, enterovirus reductions of 50 percent and under are to be expected. Septic tanks usually serve small populations (5–200 people), and so influent and effluent virus concentrations will fluctuate dramatically.

The ultimate fate of viruses entering a septic tank depends on the disposal of the effluent and the sludge. Effluents are normally discharged to drainfields, where viruses may be retained and inactivated in the soil. Cliver, Green and Bouma (1975) reported that septic tank effluent (containing $10^9$ seeded polioviruses per liter) was rendered virus free after travelling 0.4 meters through sand, with an application rate of 0.05 cubic meters per square meter per day at 20°C. Higher application rates or lower temperatures greatly reduced virus removal. This and other studies relevant to enterovirus removal from septic tank effluent have been recently reviewed in detail (Small Scale Waste Management Project 1978). More information on virus removal by sand and soil is given in the subsections below on filtration and land treatment.

Septic tank sludge will be rich in accumulated enteroviruses and requires treatment by digestion, drying or composting (see the subsections below on sludge treatment). The inactivation of enteroviruses, both in sludge within the septic tank and in the drainfield, will be considerably enhanced by warm temperatures.

*By trickling filters*

The basic mechanism for virus removal by trickling filter plants is adsorption onto the biological slime; retention times are too brief for other processes to be significant. However, reported removal rates are low, and this suggests that there is poor contact between viruses and slime surface, that adsorbed viruses are subsequently eluted by the flow of sewage passing through the filter, or both.

Sherman and others (1975) found that 9 percent and 19 percent of seeded coliphage f2 were removed by the trickling filter beds in two treatment plants. When primary sedimentation trickling filters and secondary sedimentation were considered together, coliphage removals were 55 percent and 64 percent. Buras (1976) studied the performance of the Haifa (Israel) trickling filter plant over a two year period. Average influent biochemical oxygen demand by the standard test ($BOD_5$) was 500 milligrams per liter, while average effluent $BOD_5$ was 70 milligrams per liter. The monthly average enterovirus concentrations in the influent varied between $6 \times 10^3$ per liter and $4.9 \times 10^5$ per liter, with a 2-year mean of the monthly means of $1.3 \times 10^5$ per liter. The monthly average concentrations in the effluent varied between $3 \times 10^3$ per liter and $4.5 \times 10^5$ per liter, with a 2-year mean of the monthly means of $9.6 \times 10^4$ per liter. An overall removal efficiency of only 26 percent is derived. Kott, Ben-Ari and Vinokur (1978) isolated between $2.4 \times 10^3$ and $1.2 \times 10^4$ enteroviruses per liter of trickling filter plant effluent at Haifa.

Clarke and Chang (1975) studied the performance of bench-scale, rotary-tube trickling filters. At medium filtration rates poliovirus 1, echovirus 12, and coxsackievirus A9 were reduced by 85, 83, and 94 percent, respectively. At higher filtration rates removals were 59, 63, and 81 percent, respectively. The authors failed to disassociate viruses from the biological slime in the filters and concluded that either the slime-virus complex is very stable or that the virus is somehow inactivated by adsorption to the slime. Fecal coliform and fecal streptococci removal rates closely paralleled those for enteroviruses and lead the authors to suggest that these bacteria may be used as indexes of viral removal by trickling filters. Gerba, Stagg and Abadie (1978) investigated the association with solids of enteroviruses in the effluent of a trickling

filter plant in Houston (Texas, USA). Between 9 and 196 enteroviruses per liter were contained in the effluent, and between 3 and 20 percent of these were adsorbed onto solids. This is a much lower solids-associated proportion than that reported by the same authors for activated sludge effluent (49 to 100 percent) and supports the contention that the poor virus removal efficiency of trickling filters is due to the system's providing insufficient opportunity for virus adsorption to solids or slime.

Few data are reported on the removal of enteroviruses by trickling filters in developing countries. Nupen (1970) reported that the trickling filter plant (together with primary and secondary sedimentation) at Windhoek (Namibia) reduced an influent concentration of $2 \times 10^4$ viruses per liter by 82 percent. In a subsequent report (Nupen, Bateman and McKenny 1974) it was stated that the outflow from the primary sedimentation tanks at Windhoek contained $7 \times 10^4$ viruses per liter and that, following trickling filtration and secondary sedimentation, this was reduced by 70 percent in winter and by 95 percent in summer.

Removal rates reported in the literature listed in the appendixes of Feachem and others (1980) vary between 0 and 95 percent. It is not always clear from the literature whether removal in the trickling filter alone, or across the whole treatment plant, are being recorded. Predictably, removal achieved in laboratory models (for instance Clarke and Chang 1975) is far higher than that achieved in practice (for instance, Buras 1976), and removal is reduced at higher loading rates. A typical removal rate for a trickling filter unit alone might be 5 to 20 percent, whereas a complete trickling filter plant (with no tertiary processes) could be expected to remove 25–60 percent of enteroviruses. Many of the viruses removed from the sewage will be concentrated into the primary and secondary sludges.

*By activated sludge*

The most significant variables in the removal of enteroviruses from activated sludge effluent are temperature, retention time (Heyward and others 1977; Malina and others 1975), the degree of adsorption of viruses onto activated sludge flocs (which may vary considerably between different virus types—Farrah and others 1978; Gerba and others 1980), and the efficiency of removal of suspended solids from the final effluent. Studies on virus removal by activated sludge are listed in the appendixes of Feachem and others (1980).

Since retention times in activated sludge plants are short (typically 6–12 hours), it is to be expected that most removal of virus is by adsorption to flocs that are subsequently removed by sedimentation. Glass and O'Brien (1980) calculated that the inactivation rate of enteroviruses in activated sludge mixed liquor at 25°C was about 12 percent per hour. Therefore, in an activated sludge tank with a mean retention of 9 hours, only 68 percent virus removal would be obtained by inactivation even if all liquor were retained for the mean retention time.

Moore and others (1974) studied a contact stabilization plant (contact time of 20–30 minutes followed by a 4-hour stabilization period) near Austin (Texas, USA). Incoming enterovirus concentrations were 250–1,500 per liter. Between 80 and 90 percent of enteroviruses became solids associated in the mixed liquor, and overall removal varied from 80 to 90 percent. In subsequent laboratory studies it was found that 99 percent of poliovirus in mixed liquor became solids associated after 1 hour's aeration. The same authors (Malina and others 1974) also reported laboratory model studies on seeded poliovirus removal by activated sludge and contact stabilization processes. Poliovirus removals by the activated sludge model were 92–99.9 percent and were not especially sensitive to changes in aeration time (range of 5–15 hours) or to mixed liquor suspended solids concentration (range of 1940–2710 milligrams per liter). Poliovirus removal was also not affected by whether pure oxygen or compressed air was used. Contact stabilization (contact time of 16–32 minutes followed by 2.1 hours stabilization period) removed 84–99.8 percent of poliovirus. Sludges, from both the activated sludge and contact stabilization models, contained between 70 and 5,800 enteroviruses per gram.

Balluz, Jones and Butler (1977) studied a laboratory-scale activated sludge plant that received raw settled sewage from Guildford (UK) with a mean $BOD_5$ of 270 milligrams per liter and produced an effluent with a mean $BOD_5$ of 11 milligrams per liter. The temperature was 15°C. An average poliovirus removal of 99.8 percent was recorded, with 85 percent of virus associated with the solids fraction of the mixed liquor. The authors stress that the efficiency of the plant in removing viruses may be closely related to the ability to remove suspended solids and that the subsequent treatment of the virus-rich sludge is of the utmost importance. In similar experiments with coliphage f2, the same authors (Balluz, Butler and Jones 1978) found a removal of only 68 percent and that a mere 16 percent of phage was associated with the solids fraction. It was concluded that coliphage is an

unsuitable indicator of enterovirus behavior in sewage treatment processes (see also Butler and Balluz 1979).

These findings on coliphage in activated sludge are important in interpreting the results of studies in which coliphage has been seeded into treatment plants to study virus removal. Naparstek and others (1976) recorded the removal of seeded coliphage f2 at an activated sludge plant in Maryland (USA). On average, only 11 percent of coliphage was removed by the aeration tanks and secondary sedimentation units, and the removal across the whole plant (which included chlorination) was only 80 percent. Safferman and Morris (1976) studied the coliphage removal ability of a sophisticated pilot plant that incorporated high-rate activated sludge, clarification, nitrification, denitrification, aeration, and filtration. Average flow was 200 cubic meters per day, and the final effluent had a $BOD_5$ of 2 milligrams per liter. Removal of coliphage by the high-rate activated sludge unit was between 90 and 99 percent, whereas removal across the whole plant averaged 99.97 percent.

Gerba, Stagg and Abadie (1978) found between 0.1 and 7 enteroviruses per liter in the effluents from two activated sludge plants in Houston (Texas, USA). Between 49 and 100 percent of viruses in the effluent were adsorbed onto solids. Fujioka and Loh (1978) investigated a treatment plant in Hawaii (USA) that employed settling and activated sludge. Influent contained 27–19,000 viruses per liter, while effluent contained 7–5,222 per liter. Rao and others (1977) studied virus removal at the Dadar sewage treatment plant (Bombay, India) where about 19,000 cubic meters of sewage per day are treated by activated sludge prior to marine discharge. Effluent $BOD_5$ over a 2-year period averaged 6 milligrams per liter (98.5 percent reduction), and effluent suspended solids averaged 20 milligrams per liter (97.2 percent reduction). Raw sewage contained 250–1,250 enteroviruses per liter, and final effluent contained 5–60 per liter. Removal rates were between 90 and 99 percent.

Both laboratory and field experience indicate that activated sludge systems are not particularly effective in removing enteroviruses but are more effective than trickling filters (see above and Heyward and others 1977). Enterovirus removal in activated sludge treatment works is in the range 0 to 99 percent, although better results (up to 99.9 percent) have been achieved in laboratory and pilot-scale models. It is reasonable to assume that a well-run activated sludge plant may reduce the enterovirus concentration by 50–95 percent, but that a poorly operated plant will achieve negligible removal. Many of the viruses removed from the sewage will be concentrated into the primary and secondary sludges.

*By oxidation ditch*

Practically no information is available on enterovirus removal by oxidation ditches (see the appendixes of Feachem and others 1980). The process is essentially similar to activated sludge, but the longer hydraulic retention times (1–3 days), and the higher proportion of sludge recycling giving a solids retention time of 10–30 days, are features that should produce improved virus removal. This is supported by laboratory studies in the USSR indicating the elimination of seeded enteroviruses following 2 day's aeration (Goncharuk and others 1970) and by pilot-plant studies in India showing 97–99.7 percent reduction of naturally occurring enteroviruses (Rao and others 1973). However, full-scale ditches will achieve considerably lower removal rates, and poorly operating plants will most likely remove a negligible proportion of enteroviruses.

*By waste stabilization ponds*

Very few systematically compiled data exist on the virus removal properties of well-constructed waste stabilization pond systems in warm climates. Removal rates reported (see the appendixes of Feachem and others 1980), vary widely, which is partly due to poor pond design, poor experimental procedures and short-circuiting of sewage flow across the ponds (Malherbe and Strickland-Cholmley 1967).

Rao, Lakhe and Waghmare (1978) reported that even very poorly designed stabilization ponds in India achieved virus removal rates similar to those of activated sludge plants. A single pond with 3–10 days retention removed 89.9–96.2 percent of viruses; a single pond with a 2.7 days retention time removed 94.8–97.3 percent, and 4 ponds in series with a 17.2 days retention time removed 88–98.9 percent of viruses.

There is ample evidence of reduced survival of enteroviruses in stabilization ponds at warm summer temperatures when compared with the same ponds at cooler winter temperatures (Funderberg and others 1978; Kott, Ben-Ari and Betzer 1978; Lefler and Kott 1975; Slanetz and others 1970). Viruses adsorbed to settleable solids will fall to the sludge layer at the base of the facultative pond where they may survive for extended periods (Funderberg and others 1978). Other biological and physical factors—such as a virucidal increase in pH to 9 or above caused in part by blooms

of algae (Funderberg and others 1978)—also may determine virus survival.

Lund (1979) estimated that virus inactivation in heavily polluted water might proceed at approximately 1 log unit in 5 days at 32°C and 1 log unit in 1 day at 35°C. Funderberg and others (1978) reported poliovirus removal in model outdoor ponds near Austin (Texas, USA). During the summer over 99 percent of added virus was lost within 5 days, whereas this degree of removal took 15 and 25 days in spring and winter, respectively.

These data suggest that well-designed stabilization ponds in the tropics (with minimal short-circuiting, water temperature above 25°C, and overall retention time of 30 days or more) should achieve very high levels of virus removal (at least a 4 log reduction). Confirmation of this must await further experimentation on well-designed waste stabilization ponds in developing countries.

*By aerated lagoons*

An aerated lagoon on its own may be expected to have a virus removal rate similar to, or a little better than, an oxidation ditch. If the effluent is treated in maturation ponds, removal rates as in waste stabilization ponds are expected. No specific data are available, but warm temperatures will certainly increase enterovirus inactivation rates. The sludge drawn off from secondary sedimentation tanks or settling ponds will be rich in enteroviruses.

*By tertiary treatment*

Some tertiary, or advanced physicochemical, treatment processes are effective in removing viruses. However, they add cost to sewage treatment and in some cases are too technically and mechanically sophisticated to be appropriate in developing countries.

LAGOONING. Secondary effluents may be further treated in maturation lagoons. Enterovirus removal rates and processes are the same as in waste stabilization ponds, except that little or no sedimentation takes place. High rates of virus removal can be achieved if several lagoons are employed and short-circuiting is avoided.

Kott, Ben-Ari and Betzer (1978) investigated the use of lagoons for the tertiary treatment of trickling filter effluent at Haifa (Israel). In winter (temperatures down to 8°C), initial enterovirus concentrations of $1.1 \times 10^4$ per liter were reduced to zero in 47–73 days. In summer (18–20°C), with initial concentrations of 2,000 per liter, inactivation was complete within 11–35 days. Nupen (1970) and Nupen, Bateman and McKenny (1974) reported a 95 percent reduction in enteroviruses in a chain of 9 maturation lagoons (total retention time 14 days) receiving the trickling filter plant effluent at Windhoek (Namibia). Lagoon effluent contained up to 25 enteroviruses per liter in summer and up to 842 per liter in winter.

COAGULATION. Coagulation is one of the more effective chemical processes for removing viruses from wastewater. Alum [$Al_2(SO_4)_3$], lime [$Ca(OH)_2$], iron salts, and polyelectrolytes have all been used. Wolf and others (1974) reported a greater than 99.6 percent removal of seeded coliphage f2 and poliovirus 1 in a laboratory model coagulation-sedimentation system employing alum. Lime is probably the most effective coagulant, since the high pH values produced are strongly virucidal (particularly above pH 11—see, for example, Nupen, Bateman and McKenny 1974). For maximum efficiency, coagulation should be followed by slow sand filtration (Berg 1973; Berg, Dean and Dahling 1968; Derbyshire and Brown 1979; Grabow, Middendorf and Basson 1978; Nupen 1970; Shelton and Drewry 1973; Sproul 1976). A comprehensive review of virus removal by coagulation and pH adjustments has been recently published (Sproul 1980).

FILTRATION. Sand filters can remove a high proportion of viruses from secondary effluents, but reported performances are erratic. Higher removal rates are achieved at lower filtration rates. Removal of viruses is also enhanced by low or high pH and by the presence of cations (Jenkins and others 1980) and very much enhanced by coagulation prior to filtration (Berg 1973).

Sproul (1976) reported 99.7 percent removal from activated sludge effluent at a filtration rate of 0.04 cubic meters per square meter per day and 100 percent removal at 0.007 cubic meters per square meter per day. Safferman and Morris (1976) reported very poor removal of coliphage (0 to 48 percent) by dual and multimedia filters without precoagulation. Berg, Dean and Dahling (1968) recorded an 82–99.8 percent removal of viruses in lime coagulated effluent at a filtration rate of 131 cubic meters per square meter per day.

Very significantly from the viewpoint of developing country operating problems, Vaughn and others (1978) stated that the treatment plant at Holbrook (New York, USA), which features extended aeration,

denitrification, and gravity sand filtration, was experiencing "operating difficulties" and they isolated up to 283 enteroviruses per liter from the final effluent. Assuming a raw influent concentration of about 1,000 per liter, a removal rate of only 72 percent was achieved in filtered tertiary effluent in an industrialized country. Similarly, Rao, Lakhe and Waghmare (1978) reported that a sewage reclamation plant at a factory in Bombay incorporated extended aeration, alum coagulation, rapid sand filtration, and deionization but achieved a virus removal of only 81–99 percent. These are powerful illustrations both of the operating difficulties frequently experienced with advanced wastewater treatment plants even in developed countries and of the inapplicability of much virus removal data obtained in laboratory or pilot-scale plants to full-scale operating treatment plants.

DISINFECTION. Enterovirus removal from secondary or tertiary effluents by disinfection has been the subject of numerous investigations in recent years. The most widely used disinfection technique for sewage effluents is chlorination, and there is considerable evidence that viruses are less readily destroyed by effluent chlorination than enteric bacteria (Berg and Metcalf 1978; Snead and others 1980), although, unlike some bacteria, viruses cannot regrow in the effluent subsequently.

As with the bactericidal effects of chlorine in water treatment, free chlorine (especially in the form of hypochlorous acid at low pH and particularly at warm temperatures) is a far more potent virucide than combined chlorine (monochloramine, dichloramine, and other compounds), which is formed in the presence of ammonia and organic matter (Olivieri, Donovan and Kawata 1971). Chlorine added to most sewage plant effluents is rapidly converted to combined chlorine, and this, together with the protective effect of virus association with solid particles, may result in very poor virus removal.

In clean water at pH 7–8 1–2 milligrams per liter of free chlorine maintained for 1–2 hours will be more than sufficient for complete virus inactivation. Englebrecht and others (1978) showed that 6 different enteroviruses in water were all inactivated by 99 percent in under 5 minutes when 0.5 milligrams per liter of free chlorine were applied at 5°C and pH 7–8. In a secondary sewage effluent ($BOD_5$ of 45 milligrams per liter), however, poliovirus was reduced by 50 and 90 percent in 6 hours after applying 5 and 11 milligrams per liter of chlorine, respectively (Shuval and others 1966). Similarly, Berg and Metcalf (1978) reported that, at 22–24°C, 11–23 milligrams per liter of combined chlorine added to primary effluent inactivated only between 1 and 2 log units of enterovirus in 15 minutes, whereas in the same experiments fecal coliform reductions ranged from 3 to more than 5 log units, and total coliform reductions ranged from 5 to more than 7 log units.

Different species of human excreted virus have different sensitivities to inactivation by chlorine. Reoviruses are among the most sensitive, and polioviruses are among the most resistant (Drulak, Wallbank and Lebtag 1979; Englebrecht and others 1978; Shuval and others 1966; Sproul 1976).

Boardman and Sproul (1977) studied the inactivation of coliphage T7 by chlorine when the phage was adsorbed to particles of clay, aluminium oxide, or calcium carbonate in water. It was concluded that adsorption of virus to the surface of inorganic particles offered no protection against inactivation by chlorine, but that encapsulation by a particle may afford protection. This conclusion has been confirmed by studies by Hejkal and others (1979) into the inactivation by chlorine of poliovirus in fecal homogenates. They found that the virus that was closely associated with, or occluded within, small fecal particulates was protected from chlorine inactivation. A combined chlorine residual of 6.6 milligrams per liter (at pH 8 and 22°C) achieved a 50 percent inactivation of solids-associated virus in 15 minutes, whereas only 1.4 milligrams per liter of combined chlorine were sufficient to obtain the same reduction of free virus in the same time. However, these differences were small compared with differences in inactivation due to dissolved organics that determined whether any free chlorine, as opposed to combined chlorine, was present. Stagg and others (1978) studied three treatment plants in Houston (Texas, USA) and found that between 2 and 21 percent of coliphages in plant effluent prior to chlorination were solids associated. Passage through chlorine contact chambers inactivated freely suspended phages to a greater extent than solids-associated phages, and increased the proportion of solids-associated phages in the final effluent to between 6 and over 99 percent. Only about 15 percent of the solids-associated viruses were embedded; the remainder were adsorbed.

It is clear from the above that the efficacy of effluent chlorination in virus removal depends considerably upon the quality of the effluent prior to chlorination. The better the quality of the effluent, the higher the virus inactivation attained by a given chlorination system; tertiary treatment (for instance, by filtration) is therefore often recommended to reduce further suspended solids and dissolved organics prior to

chlorination (Dryden, Chen and Selna 1979; Kirkpatrick and Presecan 1978).

Several reports indicate that well-run chlorination units, receiving high quality effluents, can produce a virus-free final effluent. Kott, Ben-Ari and Betzer (1978) investigated the effect on enteroviruses of chlorinating a trickling filter effluent at Haifa (Israel). At a chlorine dose of 20 milligrams per liter and a contact time of 2 hours, enterovirus concentrations were reduced from up to 5,900 per liter to zero (see also Kott, Ben-Ari and Vinokur 1978; Lindeman and Kott 1971).

The possibility of a virus-free chlorinated effluent is also illustrated by data from some of the advanced wastewater reclamation plants. Culp (1974*a*, 1974*b*) reports complete virus inactivation at a Lake Tahoe (USA) sewage treatment plant by carefully controlled chlorination. Grabow and Isaäcson (1978) failed to isolate any enteroviruses from 144 10-liter samples of water produced by the advanced wastewater reclamation plants at Windhoek (Namibia) and Pretoria (South Africa). These plants incorporated a train of advanced processes and included break-point chlorination sufficient to produce 0.2–0.6 milligrams per liter of free residual chlorine after 2–3 hours of contact time (Nupen 1970; Nupen, Bateman and McKenny 1974).

However, chlorination in no way guarantees a virus-free effluent. Sherman and others (1975) found that the chlorination of trickling filter plant effluents from two treatment plants in Maryland (USA) reduced seeded coliphage by 60 percent. Overall reductions of phage across the two plants were 82 percent and 86 percent. Fujioka and Loh (1978) reported isolating 25–34 and 2–750 enteroviruses per liter from the chlorinated effluent of two treatment plants in Hawaii (USA). Influent concentrations were 5–268 and 27–19,000 enteroviruses per liter, respectively. Wellings and others (1975) found on average 0.13 enteroviruses per liter (53 percent of samples positive) in the chlorinated effluent from a package treatment plant receiving sewage containing 161 viruses per liter from a mobile home park. Wellings, Lewis and Mountain (1974, 1976) isolated up to 12 and 98 enteroviruses per liter, in two studies on the chlorinated effluent from an activated sludge plant in St. Petersburg (Florida, USA). Metcalf, Wallis and Melnick (1974) isolated up to 4 enteroviruses per liter from a chlorinated effluent (1.2–1.9 milligrams per liter of residual chlorine after 10 minutes of contact) at an activated sludge plant near Houston (Texas, USA). Vaughn and others (1978) isolated up to 26 and 98 viruses per liter from the chlorinated secondary effluents from two sewage treatment plants on Long Island (New York, USA). Bausum, Schaub and Kenyon (1978) found that chlorination of a trickling filter effluent in Arizona (USA) reduced the concentration of seeded coliphage by only 95 percent, as compared with a bacterial reduction of 99.97 percent. Kott and others (1974) studied chlorinated waste stabilization pond effluents in Israel (8 milligrams per liter of applied chlorine for 1 hour at 20°C) and found an average enterovirus reduction of only about 10 percent, whereas the total coliform reductions were 2 log to >6 log units. The chlorinated pond effluents contained between 300 and 1,000 enteroviruses per liter. Other experiments found that, at pH 6.0 with a 2-hour contact time, seeded poliovirus 1 in stabilization pond effluent was reduced by 86 percent with 20 milligrams per liter of applied chlorine, by 87 percent with 40 milligrams per liter of chlorine, and by 100 percent with 60 milligrams per liter of chlorine.

The chlorination of inadequately treated sewage, in the hope of thereby removing much of the microbial hazard, is disturbingly widespread despite clear evidence that it is generally ineffective and represents bad engineering practice. The authors of this book have observed this practice on several occasions, and have frequently heard it recommended in developing countries when concern is being expressed about the discharge of highly polluted effluents from improperly designed or malfunctioning sewage treatment plants. An interesting case study of this problem is the investigations by Sattar and Westwood (1978) in Ottawa (Canada), a city of 500,000 people that discharged 90 percent of its sewage into the Ottawa River after the wastes received only primary treatment (sedimentation) and chlorination. Two treatment plants were studied that received a raw sewage with a $BOD_5$ of 79–98 milligrams per liter and that produced a chlorinated primary effluent with a $BOD_5$ of 44–48 milligrams per liter. Raw sewage samples were 80 percent positive for enteroviruses, with an average concentration of 1,000 per liter. Samples of sedimentation tank effluent were 72 percent positive for enteroviruses and also contained 1,000 viruses per liter. Samples of final chlorinated effluent were 56 percent positive for enteroviruses and contained 27 viruses per liter. The Ottawa River is used for recreation and provides the raw water source for about 600,000 people.

Various other wastewater disinfection systems have been studied with respect to their ability to inactivate viruses. Ozone is a very potent virucide, and its activity is less disturbed by organic pollution than is the case for chlorine (De Michele 1974; Dryden, Chen and Selna 1979; Evison 1978; Katzenelson and

Biedermann 1976; Munger, Heyward and Swartz 1977; Pavoni and Tittlebaum 1974; Rakness and Hegg 1979; Sproul 1976). Bromine chloride and paracetic acid have been evaluated and are also little affected by organic matter in the effluent (Hajenian and Butler 1980). Chlorine dioxide also effectively inactivates enteroviruses in water at a rate similar to hypochlorous acid. Unlike hypochlorous acid, chlorine dioxide is more potent at higher pH values (Cronier, Scarpino and Zink 1978). Radiation at a level of 3–5 kilogray[6] inactivates approximately 90 percent of viruses in sewage effluent, whereas only 1–1.5 kilogray are required to achieve the same inactivation of viruses in distilled water (Metcalf 1977; Sullivan and others 1971). Photodynamic oxidation has also been evaluated for the disinfection of wastewaters (Gerba, Wallis and Melnick 1977*a*, 1977*b*). However, all these techniques are currently at the experimental stage; in any case, they may involve a level of technical sophistication and cost that would make them inappropriate in many situations in developing countries.

LAND TREATMENT. Land treatment by soil filtration or groundwater recharge can be highly effective in removing viruses from primary or secondary sewage effluents, but results reported in the literature vary widely.

Lance, Gerba and Melnick (1976) showed, in laboratory studies, that poliovirus in secondary effluent was almost completely removed by filtration through loamy sand after flowing to a depth of 1.6 meters and was reduced by 99 percent after 0.4 meters of flow. These results were obtained at filtration rates of both 0.55 cubic meters per square meter per day and 0.15 cubic meters per square meter per day. Flooding the soil with deionized water (to simulate a rainstorm) caused some downward movement of the viruses, but this was greatly reduced when $CaCl_2$ was added to the deionized water. Drying of the soil between effluent application and a simulated rainstorm considerably reduced desorption of viruses; 5 days drying prevented subsequent desorption completely. The authors concluded that viruses would move through 2.5 meters of calcareous sand only if heavy rains fell within a day following the cessation of sewage application. In follow-up studies (Gerba and Lance 1978; Lance and Gerba 1980; Lance, Rice and Gilbert 1980), similar adsorption and elution results were obtained in loamy sand whether polioviruses were suspended in primary or secondary effluent. In one series of experiments, 99 percent of poliovirus in primary effluent was removed in the top 0.2 meters of loamy sand, and no poliovirus was detected below 0.8 meters, when the flow rate was approximately 0.2 cubic meters per square meter per day. In other experiments it was shown that poliovirus adsorption by loamy sand is reduced when the flow rate is increased above some critical value, whereas flow rate changes above and below this value do not affect adsorption. The critical value for loamy sand was about 1 cubic meter per square meter per day; below this flow rate poliovirus penetration was less than 1.6 meters, whereas above it viruses penetrated the entire 2.5-meter column.

Duboise, Moore and Sagik (1976) reported that poliovirus and coliphage adsorbed to cores of loamy sand were eluted less by intermittent application of effluent or water than by continuous application. In addition, the chemical quality of the effluent improved more by soil filtration when the effluent was applied intermittently than when it was applied continuously (see also Duboise and others 1974).

Bitton, Masterton and Gifford (1976) found that under experimental conditions coliphage T2 and poliovirus were adsorbed more readily when suspended in tap water than in secondary effluent, and that effluent is a more potent eluent than tap water for washing adsorbed viruses out of soil columns. Lefler and Kott (1976) also found higher elution of poliovirus and coliphage from sand with effluent as compared with tap water. However, Duboise, Moore and Sagik (1976) reported that flooding cores of loamy sand with dechlorinated effluent eluted fewer polioviruses than when distilled water was used.

Landry and others (1979) found that 72–100 percent of polioviruses, coxsackieviruses, and echoviruses were retained when tertiary sewage effluent was passed through 1.25-meter natural cores of gravelly sand at a rate of 20 cubic meters per square meter per day. Flooding the cores with artificial rain water released 0–67 percent of the adsorbed viruses, whereas flooding with sewage effluent released 0–14 percent. A total of eight laboratory and field strains of enteroviruses were used in these experiments. Some differences in adsorption among the strains were recorded (with echovirus 1 showing the greatest soil affinity), and the proportion of adsorbed viruses being eluted by rainwater or sewage differed markedly (with echovirus 1 being the least mobilized and a wild strain of poliovirus 3 being the most readily eluted). The authors concluded that soil adsorption-elution behavior is strain dependent and warn against the application of laboratory data from experiments using only a single strain of poliovirus 1. Gerba and others (1980), Goyal and Gerba

6. See footnote 5, above.

(1979), and Goyal and Melnick (1978) also reported wide intertypic and intratypic variation in adsorptive behavior among enteroviruses in various soils.

Burge and Enkiri (1978*a*) reported substantial differences in the ability of five soils to adsorb coliphage viruses. More acidic soils had higher adsorption rates, and one loamy sand adsorbed no viruses (see also Burge and Enkiri 1978*b*; Vilker and Burge 1980). Similarly, Lefler and Kott (1974*b*) found that many coliphage f2 and poliovirus 1 were able to pass through a 0.2-meter sand column (at an application rate of 1.7 cubic meters per square meter per day) and that only a high concentration of bivalent cations ($Ca^{++}$ and $Mg^{++}$) prevented this. Funderberg and others (1979), Goyal and Gerba (1979), and Moore and others (1979) have also reported wide variation in virus adsorption behavior dependent on soil properties.

Studies by Scheuerman and others (1979) showed that some organic soils have poor virus adsorption potential due to the presence of water-soluble humic substances (humic and fulvic acids), which may compete with viruses for adsorption sites on soil particles or may react with certain surface groups on virus particles that are functionally important in adsorption to soil.

Wellings, Lewis and Mountain (1974) studied an effluent spray irrigation site near St. Petersburg (Florida, USA). The effluent from the activated sludge plant contained up to 240 enteroviruses per liter, and the final chlorinated effluent contained up to 98 per liter. The effluent was applied to a sandy soil at between 0.007 and 0.04 cubic meters per square meter per day. Water collected in drains 1.5 meters under the soil contained polioviruses, echoviruses, and reoviruses (2 out of 9 samples positive). Enteroviruses were also isolated from wells 3 and 6 meters deep at the site following heavy rain.

Wellings and others (1975) studied the discharge of effluent from a 154-unit mobile home park into a cypress dome in the wetlands of Florida (USA). The population of the park varied between 310 and 337 during the study and produced a raw sewage with an average of 161 enteroviruses per liter (range between 0 and more than 700 per liter). Polioviruses 1, 2, and 3 accounted for 40 percent of isolates identified, with coxsackieviruses (B3 and B4) making up 43 percent, and echoviruses (7, 11, and 14) the remaining 17 percent. Chlorinated effluent from a package treatment plant yielded polioviruses and coxsackieviruses in 8 out of 15 samples taken. The effluent was discharged into the cypress dome, and groundwater quality was monitored in 18 3-meter-deep wells constructed in and around the dome. Enteroviruses were isolated from 3 out of 48 well-water samples, and horizontal movement of viruses through the saturated soils of at least 7 meters was demonstrated.

Gilbert and others (1976*a*, 1976*b*) studied virus removal from activated sludge effluent (up to 75 viruses per liter) applied to loamy sand (infiltration rate of 0.27 cubic meters per square meter per day) at a 7-year-old wastewater renovation plant near Phoenix (Arizona, USA). No viruses were detected in the observation wells, indicating at least a 99.99 percent removal after flow through 3–9 meters of soil. In contrast, Schaub and Sorber (1977) reported very low viral removal by soil filtration (infiltration rate 0.07 cubic meters per square meter per day) of primary effluent at a 30-year-old treatment plant in Massachusetts (USA). The soil was unconsolidated silty sand and gravel. Seeded coliphage f2 viruses were reduced by an average of 53 percent after 18 meters of percolation, and seeded f2 and indigenous enteroviruses were sporadically detected in the groundwater at horizontal distances of 180 meters from the application zone.

It is clear from these and other studies that adsorption to soil particles, rather than viral death, is the dominant removal mechanism. Adsorption is increased by low infiltration rates (say $<0.1$ cubic meters per square meter per day), by low pH, and by the presence of divalent cations ($Ca^{++}$ and $Mg^{++}$). Adsorption is reduced in the presence of soluble organic matter which competes for adsorption sites on the soil particles. Adsorption and elution behavior depend very much upon the characteristics of the soil and the particular strain of virus. A great deal of further experimentation will be required before the optimal soil structures—and their relationships to infiltration rates, application schedules, and virus removal performance—are fully understood. It is already apparent, however, that by passing even raw sewage through less than 1 meter of a suitable soil it is possible to reduce the virus concentration by as much as, or more than, that normally achieved by wastewater chlorination (Gerba 1979). Because of higher temperatures there, the effectiveness of land application in virus removal in the developing countries is likely to be greater than that generally reported from temperate areas. It must be remembered, however, that without efficient management, operation, and maintenance land application systems will become insanitary bogs.

The above discussion has dealt exclusively with soil filtration and groundwater recharge as methods of land treatment for wastewaters. The other major type of land treatment technology is the grass plot or overland run-off method. In these systems a significant

proportion of the effluent may run over the surface of the soil and not flow through it, and it may be expected that virus removal will be poor compared with the soil filtration data reported above. Experiments by Schaub and others (1980) confirmed this, showing a removal of only 30–60 percent of seeded f2 bacteriophage and only 76–88 percent of indigenous excreted virus during treatment on 36 meter long grass plots with a 3 percent slope.

OTHER PROCESSES. A variety of other treatment processes are associated with advanced wastewater treatment plants and with water reclamation or renovation projects. Some of these processes have been assessed for virus removal capability. Carbon adsorption and nitrification do not seem particularly effective, whereas denitrification was reported to remove 97 percent of coliphage (Berg 1973; Gerba and others 1974; Safferman and Morris 1976; Sproul 1976). Complete water reclamation plants, incorporating a train of advanced processes, are generally reported to achieve total virus removal when operating perfectly. The Lake Tahoe (Nevada, USA) reclamation plant has occasionally let through viruses beyond the carbon adsorption stage, but they were eliminated by chlorination (Berg 1973). Similarly, the Windhoek (Namibia) reclamation plant is reported to have achieved a virus-free effluent, despite an influent virus concentration of up to $2 \times 10^4$ per liter (Grabow and Isaäcson 1978; Nupen 1970; Stander and Clayton 1977).

## Inactivation by Night soil and Sludge Treatment Processes

Raw night soil contains all the viruses being excreted by the contributing population. Sewage works sludges are rich in viruses because a high proportion of viruses in sewage are, or become, solids-associated and are therefore concentrated into both primary and secondary sludges (Lund 1973; Lund 1976; Lund and Ronne 1973; Wellings, Lewis and Mountain 1976). Interest in viruses in sludges has been stimulated by the fact that a large proportion of sewage sludge is applied to the land as a method of disposal and soil enrichment.

Information on this subject is restricted to laboratory studies and a few field studies conducted in North America and Europe. Little is known about viruses in sludge in developing countries or about sludge treatment under tropical climatic conditions. Even less is known about the virological aspects of night soil and night soil treatment.

### *By pit latrines*

Little information is available, but it is probable that enteroviruses survive for several weeks in pit latrines (see the sections above on occurrence and survival in feces and night soil and in sludge; see also the appendixes to Feachem and others 1980). In warm climates, the pit contents should be free of enteroviruses if they are left for at least 3 months before digging out.

A pit latrine may act as a source of viral groundwater pollution depending on the type of soil, groundwater levels, and the proximity of local wells (see the sections above on occurrence and survival in groundwater and on virus removal by land treatment).

Francis, Brown and Ainslie (1953) isolated polioviruses (16 out of 220 samples positive) and coxsackieviruses (10 out of 63 samples positive) from pit latrines in poor areas of four towns in southern Texas (USA). Pit latrines with polioviruses were not associated with known cases of poliomyelitis, an epidemic of which was taking place at the time (March–July of 1948), but were associated with the isolation of polioviruses from flies in the vicinity.

### *By anaerobic digestion*

Although some form of anaerobic digestion is used to treat sludge from most larger sewage treatment plants, very little information is available on the virus removal performance of full-scale digesters. (Some laboratory studies are discussed below and are listed in the appendixes of Feachem and others 1980).

Ward and Ashley (1976) investigated the inactivation rate of poliovirus in digested sludge and found that it was greater than 1 log unit per day at 28°C and about 1 log unit per 5 days at 4°C. They concluded that anaerobically digested sludge contains a specific virucidal agent; in a subsequent study (Ward and Ashley 1977*a*) they identified this agent as ammonia (see also Fenters and others 1979). Ammonia is not virucidal in its charged state, but free ammonia, which is formed at pH values of 8 and above, is highly virucidal to enteroviruses but much less so to reoviruses. Ward and Ashley concluded that ammonia acts as a potent enteroviricide in raw and digested sludges with high pH values. At pH 9.5 and 21°C, greater than 3 log unit and 5 log reductions in poliovirus concentrations were obtained in 72 hours in raw and digested sludges, respectively. A later study

confirmed that reovirus 3 was unaffected by the presence of ammonia (Ward and Ashley 1977*c*).

Eisenhardt, Lund and Nissen (1977) studied the inactivation of coxsackievirus B3 in a laboratory-scale anaerobic sludge digester at pH 7. At 32°C a 5 log reduction in virus concentration occurred in about 14 days, whereas at 35°C the same reduction took only 4 days. Inactivation was slightly faster when the virus was held in pasteurized sludge. Bertucci and others (1977) ran a laboratory anaerobic digester (pH 7.2–7.4) at 35°C and compared inactivation rates of various enteroviruses. Inactivation rates varied from 75 percent per day for echovirus 11 to 97 percent per day for coxsackievirus A9.

Sanders and her coworkers (1979) pointed out that most previous laboratory studies investigated the inactivation of free viruses inoculated into sludge immediately prior to digestion. However, to simulate more exactly real operating conditions it is necessary to allow the viruses to become associated with solids prior to commencing digestion. Sanders therefore investigated the inactivation by anaerobic digestion of solids-associated poliovirus and found that survival was enhanced by solids incorporation. The inactivation rates at 34 and 37°C were 84 to 99 percent per day, respectively, for the first 24 hours. After that time inactivation slowed considerably to between 30 and 60 percent per day. At 50°C the inactivation rate was high at more than 7 log units per day.

Berg and Metcalf (1978) reported the destruction of between 76 and 96 percent of viruses by mesophilic digestion (35°C for 20 days) and between 98.9 and >99.9 percent by thermophilic digestion (50°C for 20 days). Enterovirus concentrations in raw sludge were $4 \times 10^3$ to $>1 \times 10^5$ per liter, 300 to 4,100 per liter in mesophilically digested sludge, and 0 to 170 per liter in thermophilically digested sludge. In these experiments, samples of digested sludge were taken shortly after the addition of fresh sludge to the digesters, and so theoretical retention times would not have applied to all aliquots of digested sludge.

Wellings, Lewis and Mountain (1976) isolated enteroviruses and reoviruses, at concentrations of up to 34 per liter, from sludge from an anaerobic digester in Florida (retention time >60 days at 34°C) to which no raw sludge had been added for the previous 7 days. Sattar and Westwood (1979) found excreted viruses in 53 percent of samples of digested sludge (20 days at 35°C) and in 39 percent of dried sludge samples (>6 months' drying time) at a large sewage treatment plant in Ottawa (Canada). Hurst and others (1978) isolated viruses, at concentrations of up to 231 per liter, from sludge that had been thickened, aerobically digested, and centrifuged at a Houston (Texas, USA) treatment plant.

Investigators who have looked for viruses in digested sludge have generally found them in considerable numbers (Berg and Metcalf 1978; Grigoryeva, Korchak and Bey 1969; Hurst and others 1978; Sattar and Westwood 1979; Wellings, Lewis and Mountain 1976). Some laboratory studies have reported an inactivation rate of around 1 log unit per day at 30–35°C (for instance, Eisenhardt, Lund and Nissen 1977; Fenters and others 1979; Ward and Ashley 1976). At this rate of inactivation, typical anaerobic digestion at 35°C for 35 days should produce a virus-free sludge with a wide margin of safety. However, Sanders and others (1979) have shown that inactivation rates of solids-associated viruses after the first day of digestion may be very much slower (around 1 log unit every 2–7 days). In addition, most digesters are operated by continuous, or regular, addition and removal of sludge. Therefore, some sludge has a retention time of very much less than the design value and will contain significant concentrations of viruses after digestion. It is probable that only batch digestion at 35°C, for 35 days, or digestion at temperatures of around 50°C, will produce a virus-free sludge. More field data are required, on the actual virus removal performance of operating plants of these types, to confirm this assumption.

### *By drying*

Both raw and digested sludges are normally dewatered prior to disposal, and the most common technique is spreading on outdoor sludge drying beds. Very little information is available on virus removal by sludge drying (see below and the appendixes of Feachem and others 1980), and no studies have been reported from developing countries. However, the data on enterovirus survival in sludge are also relevant (see the section above on the occurrence and survival of enteroviruses in sludge and the appendixes of Feachem and others 1980).

Ward and Ashley (1977b) investigated the inactivation of viruses in sewage sludge that occurs during dewatering by evaporation. Sludge, with a solids content of 5 percent and a pH of 6, was inoculated with $2.7 \times 10^7$ viruses per milliliter and air dried at 21°C in 1 centimeter thick layers over 4 days. As evaporation proceeded, poliovirus 1 was inactivated at a low but constant rate until, at a solids concentration of 65 percent, approximately 75 percent inactivation had occurred. At this stage inactivation

increased rapidly so that, during concentration up to 83 percent solids, a further 99.9 percent inactivation occurred. Further concentration up to 91 percent solids produced little more inactivation. A similar result was obtained with coxsackievirus B1 and reovirus 3. In a subsequent study (Ward and Ashley 1978*b*), it was found that sludge drying increased the heat required to inactivate enteroviruses and reovirus. Sattar and Westwood (1979) isolated viruses from anaerobically digested sludge that had been drying for 8 months in Canada. Wellings, Lewis and Mountain (1976) isolated 10 enteroviruses per 100 grams of sludge that had been on sludge drying beds for two weeks during February in Florida (USA).

These studies, and the reports on virus survival in sludge indicate that during cool, wet weather enteroviruses may survive in drying sludge for several months. Data on rapid virus inactivation at solids concentrations between 65 and 83 percent may be irrelevant, since under temperate conditions sludges may achieve a solids content of only about 25 percent after about 2 months on a drying bed. Even in Texas (USA) in the summer, a sludge that had been applied to land for 3 months had dried to only 59 percent solids (Hurst and others 1978). However, a comparison between virus inactivation rates in sludge during Danish winters (1 log unit per month), and during Texan summers (2 log units per week), clearly indicates that enterovirus inactivation is far more rapid in hot climates under bright sunshine. A good virus removal performance may therefore be obtained by sludge drying beds in many developing countries, and field studies are required to confirm this possibility.

*By heating*

Under certain circumstances enteroviruses can be remarkably resistant to heating. For instance, Larkin and Fassolitis (1979) reported that infectious ribonucleic acid (RNA) liberated from poliovirus 1 and coxsackievirus B2 could withstand 65 minutes at 70°C. In general, however, heat is a potent virucide, and the heating of sludges, or their digestion at elevated temperatures, is an effective method of virus inactivation.

Ward, Ashley and Moseley (1976) studied the effect of raw and anaerobically digested sludge on the heat inactivation of poliovirus. Raw sludge was found to be very protective of poliovirus inactivation whereas digested sludge was not, and subsequent studies (Ward and Ashley 1977*a*) determined that this difference was due to the virucidal activity of uncharged ammonia present at the high pH levels found in anaerobically digested sludge. At 43°C after 200 minutes, poliovirus concentration was reduced by over 3 log units in digested sludge, but it was almost stable in raw sludge. At 51°C, the poliovirus concentration was reduced by over 5 log units in under 5 minutes in digested sludge and by over 4 log units after 50 minutes in raw sludge. In a second study (Ward and Ashley 1977*c*), the heat inactivation of reovirus 3 in sludge was investigated. Reovirus was found to be quite heat resistant compared with poliovirus but was not protected against heat inactivation by sludge. At 50°C for 20 minutes, reovirus concentrations were reduced by 4 log units in digested sludge and by 2 log units in raw sludge. At 60°C after 20 minutes, the reductions were 5 log units in digested sludge and 4 log units in raw sludge. A virucidal agent against reoviruses was discovered in the sludge that had greatly increased activity at pH values above 8. Unlike the case of the enteroviruses (Ward and Ashley 1977*a*), this agent was not ammonia. A follow-up study (Ward and Ashley 1978*a*) determined that ionic detergents found in sewage sludges reduce the heat required to inactivate reoviruses (cationic detergents being more active than anionic detergents, and nonionic detergents having no activity). In contrast, some detergents were found to protect poliovirus against heat inactivation. (More information on the virucidal activity of detergents, and the effects of differing pH values, is given in Ward and Ashley 1979). In a subsequent study (Ward and Ashley 1978*b*), a reduction in moisture content was found to reduce significantly the rates of heat inactivation of both enteroviruses and reovirus. Poliovirus in raw sludge at 51°C was reduced by over 5 log units in 5 minutes when the sludge had 5 percent solids but by less than 2 log units after 100 minutes when the sludge had an 80 percent solids content. Reovirus in raw sludge at 51°C was reduced by 4 log units in 50 minutes when the sludge had 5 percent solids and by less than 2 log units after 50 minutes when the sludge had a solids content of 80 percent.

When compared with the reality of sludge treatment processes, however, these are trivial differences and distinctions. Figure 9-2 presents data on the survival of different types of enteroviruses under different conditions for various time-temperature combinations. A conservative upper bound is drawn, above which the combinations of time and temperature should guarantee enterovirus elimination. From this figure it is postulated that holding sludge at 30°C for 3 months, at 40°C for 2 weeks, at 50°C for 1 day, or at 60°C for 2 hours, will inactivate all enteroviruses, reoviruses, and adenoviruses.

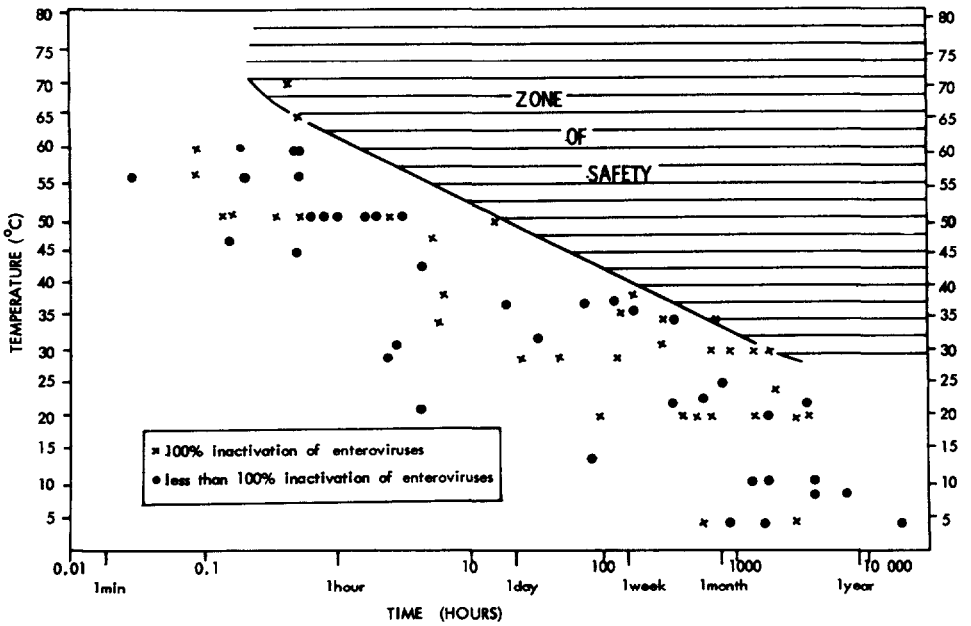


Figure 9-2. *The influence of time and temperature on enteroviruses.* The data probably also apply to adenoviruses and reoviruses. The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

*By composting*

Aerobic thermophilic composting is an effective method of inactivating viruses in sludge if all parts of the pile or mass are heated to 50°C or above for sufficient time (see figure 9-2 and the appendixes of Feachem and others 1980).

Krige (1964) reported that seeded poliovirus 1 was eliminated from a sludge, grass, and refuse mixture composted at 38–58°C for 7 days. Wiley and Westerberg (1969) determined that the thermal death points for poliovirus 1 were 60°C for 5 minutes or 55°C for 30 minutes. When poliovirus 1 was added to a forced-air sludge-composting unit operating at 60–76°C, it could not be detected after 1 hour.

Kawata, Cramer and Burge (1977) reported the inactivation of seeded bacteriophage f2 in a sludge and wood chips mixture composted at a plant in Maryland (USA). The mixture was formed into windrows, which were turned regularly (up to once per day depending upon the temperature within the mass) for 2 weeks and were then made into large piles for 4 weeks of curing. When raw sludge was composted, the temperatures rose to 50–70°C within 3 days and remained there except for short periods following rainstorms. When digested sludge was composted, the temperature rose gradually to 40–60°C after 10–14 days, and during cold wet winter weather the temperatures rose only to the 20–30°C range. Complete inactivation of the seeded bacteriophage (originally present at a concentration of $10^6$ per gram) took about 50 days in composting raw sludge and up to 70 days in composting digested sludge. Naturally occurring enteric viruses were isolated throughout the windrow phase of the composting but were never isolated from the curing piles. All these experiments were conducted during the cold and wet months of October–March. Experiments were later conducted into the inactivation of seeded coliphage f2 by a forced air composting system (21 days of aerated composting followed by 30 days of curing) at the same site (Burge, Cramer and Epstein 1978). Temperatures rose to 50°C and above within the first five days, and coliphage destruction deep in the pile was complete within 13 days. However, at the edge of the pile, very small proportions (around 0.001 percent) of virus survived after 21 days. The inactivation rate in the pile was approximately 1 log unit per 2 days. Pile temperatures in the forced air system were unaffected by ambient temperature or rainfall.

Much more research is required on virus removal from various types of composting system using night soil, sludge, refuse, woodchips, and other materials. Pending this work, virus inactivation may be tentatively predicted from figure 9-2. Even where the time–temperature characteristics in the pile are well within the safety zone in figure 9-2, virus survival may still be occurring at the edges of the pile, which are

usually much cooler, especially during rain. Complete elimination of enteroviruses is therefore dependent upon pile management techniques such as turning, lagging, or forced aeration.

*By other sludge treatment processes*

Any sludge treatment process that involves temperatures of 50°C or above should yield a virus-free product if the process is well controlled and carried out for sufficiently long to ensure that all parts of the mass are heated. This latter point is particularly important when continuous, rather than batch, processes are being used. Examples include pasteurization (70–80°C), anaerobic or aerobic thermophilic digestion (46–55°C), wet oxidation (180–220°C), incineration (over 650°C), and pyrolysis, as well as heating and composting (discussed above).

Sludge disinfection by irradiation with high-energy electrons is attracting increasing interest. (Osborn and Hattingh 1978). The few data available on virus inactivation in sludge by irradiation indicate a rather poor removal of 75–90 percent after the application of 3–5 kilograys.[7] (Lessel and Suess 1978; Sullivan and others 1971; Ward 1977). Sludge protects poliovirus from irradiation, but little or no extra protection is afforded by increasing solids content above about 1 percent (Ward 1977). Superchlorination or chlorine oxidation (the application of 700–4,000 milligrams per liter of chlorine under pressure) may inactivate most viruses in sludge but has been objected to because it proliferates chlorinated organic compounds in the environment (Kamlet 1979).

Most of these technologies for sludge disinfection are still in the research and development stage, and many of them will prove to be too costly and too technically complex to be appropriate in most situations in developing countries.

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7. See footnote 5, above.

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# 10

# Hepatitis A Virus and Infectious Hepatitis

MUCH OF THE WORK on poliovirus and other enteroviruses in the environment described in chapter 9 was inspired by an interest in other excreted viruses that cause major public health problems but cannot be routinely isolated at the present time. Foremost among these other excreted viruses is hepatitis A virus, which is a common and important cause of disease throughout the world.

The study of the hepatitis viruses is a rapidly moving field of research—hepatitis A virus was not identified in fecal extracts until 1973, although the disease it causes has been recorded by civil and military historians since the fifth century BC. This chapter is brief, because little is known at the time of writing about hepatitis A virus in the environment, and tentative, because the rate of scientific progress is rapid, and much new information will come to light in the next few years. A recent and comprehensive account of the hepatitis viruses is given by Zuckerman and Howard (1979).

## Description of Pathogen and Disease

Two distinct forms of viral hepatitis have been recognized: hepatitis A (also known as infectious hepatitis, epidemic hepatitis, or epidemic jaundice) and hepatitis B (also known as serum hepatitis). They differ in etiology and in some epidemiological, immunological, clinical, and pathological characteristics. From the environmental viewpoint, the primary distinction between them is that hepatitis A is transmitted by the fecal-oral route, whereas hepatitis B is normally transmitted by infected blood or tissue fluid (for instance, during blood transfusion, injection, immunization, tattooing, and acupuncture) but may also rarely be transmitted by saliva, semen, breastmilk, other body fluids, and feces if contaminated with blood. Because of these differences in mode of transmission, the approaches to prevention and control of hepatitis A and B are very different. Recently a third form of viral hepatitis has been described: non-A:non-B hepatitis. This new form of hepatitis is now known to be the most common type of post-transfusion hepatitis in some areas. Non-A:non-B hepatitis may prove to be divisible into more than one form. As far as is known, only hepatitis A is primarily an excreted infection, and therefore it alone will be discussed in this chapter.

### *Identification*

The clinical picture of viral hepatitis varies in its presentation from inapparent or subclinical infection, to slight malaise, mild gastrointestinal symptoms and the anicteric (without jaundice) form of the disease, to acute icteric illness, severe prolonged jaundice, and chronic liver disease. The anicteric form is characterized by malaise, anorexia, various gastrointestinal disturbances, an enlarged and tender liver, and perhaps a fever. In acute icteric infections these symptoms may be more pronounced and may be followed after 5–10 days by dark urine, clay-colored stools, and jaundice, which persists commonly for 1–2 weeks. Typically, the disease is especially mild in children, for whom the ratio of anicteric to icteric illness may be 10 or more to 1. Convalescence usually is prolonged. In general, severity increases with age, but complete recovery without sequelae or recurrences is the rule. Many mild cases without jaundice, especially in children, are recognizable only by liver-function or serum-enzyme tests.

### *Occurrence*

Viral hepatitis type A occurs endemically in all parts of the world, with frequent reports of minor and major outbreaks. The exact incidence is difficult to estimate because of the high proportion of subclinical infections and infections without jaundice, differences in surveillance, and differing patterns of disease. The degree

of underreporting is very high; even in developed countries with compulsory notification, it is doubtful whether more than 50 percent of cases of jaundice are actually reported.

Because serological tests for hepatitis A antibody are now available, it has become possible to study the incidence and distribution of hepatitis A infections in different populations and in various geographical areas. A recent survey of sera for hepatitis A antibody by immune adherence hemagglutination from samples of healthy adults, mostly volunteer blood donors, from seven geographical populations has shown that the age-standardized prevalence of hepatitis A antibody was 29 percent in Switzerland, 45 percent in the USA, 76 percent in Senegal, 81 percent in Belgium, 89 percent in Taiwan, 95 percent in Israel, and 97 percent in Yugoslavia (Szmuness and others 1977). This survey confirmed that infections with hepatitis A virus are widespread throughout the world.

*Infectious agent*

Small cubic particles measuring 27 nanometers have been seen by electron microscopy in infective feces of human subjects (see figure 10-1). The virus contains single-stranded RNA and has the biochemical and biophysical properties of a picornavirus.

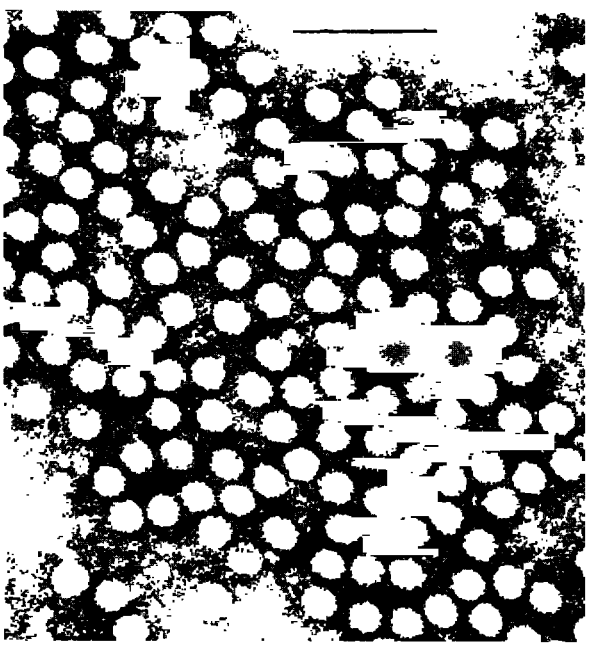

Figure 10-1. *Hepatitis A viruses under transmission electronmicroscopy.* Scale bar = 0.1 micrometers. (Photo: A. J. Zuckerman and A. Thornton, London School of Hygiene and Tropical Medicine, London, UK)

*Reservoirs*

Man is almost certainly the important reservoir for human hepatitis A infection. However, cases have been reported that suggest transmission to man from chimpanzees, gorillas, Celebes apes, gibbons, and woolly monkeys. Antibodies to hepatitis A virus are found in nonhuman primates, and susceptible chimpanzees and marmosets are readily infected in the laboratory.

*Transmission*

Hepatitis A virus is spread by the fecal-oral route, most commonly by person-to-person contact, and infection occurs readily in conditions of poor sanitation and overcrowding. Common-source outbreaks are most frequently initiated by fecal contamination of water and food, but waterborne transmission is not a major factor in the maintenance of this infection. Ingestion of shellfish cultivated in polluted water is associated with a high risk of acquiring hepatitis A.

*Incubation period*

The incubation period of hepatitis A is between 3 and 5 weeks, with a mean of 28 days.

*Period of communicability*

Hepatitis A virus is shed in the stools primarily during the period from 2 weeks before, to 2 weeks after, the onset of symptoms; in other words, for up to 4 weeks commencing 1–3 weeks after infection. Persistent carriage or excretion of hepatitis A virus in humans does not occur.

*Resistance*

Susceptibility is general. Low clinical incidence in infants and preschool children suggests that mild and anicteric infections are common. The degree and duration of homologous immunity after attack are unknown but presumed to be long lasting.

*Epidemiology*

All age groups are susceptible to hepatitis. Until recent years the highest incidence in the civilian population was observed in children of school age, but

in a number of countries, including Sweden and the USA, as many as 60–70 percent of notified cases now occur in adults. This shift in age incidence in the developed countries is reminiscent of the changing pattern of poliomyelitis (see chapter 9) and may reflect reduced transmission, caused by improved socioeconomic conditions, which defers infection to an older age group when the clinical consequences are usually more severe.

In conditions of poor hygiene, it is probable that transmission of hepatitis A virus occurs among children by direct fecal-oral routes. This leads to a high incidence of subclinical or very mild infections that confer substantial immunity. Moritsugu and others (1979) found that 92 percent of children under 9 years old in Colombo (Sri Lanka) had evidence of antibodies to hepatitis A virus. In Costa Rica (Villarejos and others 1976), age-specific antibody prevalences reached 80 percent by the age of 9 years. In Liberia, 90 percent of children over 5 years old in both urban and rural areas had hepatitis A antibodies (Willcox and others 1980). Similar results have been obtained from Kenya (Wankya and others 1979) and from Fiji, Tuvalu (formerly Ellice Islands), Niue, Cook Islands, and Western Samoa (Gust, Lehmann and Dimitrakakis 1979).

In developed countries, improved hygiene reduces the incidence of hepatitis A infection in the young, but many people become infected at some time in life. Gust, Lewis and Lehmann (1978) examined the sera (collected in 1954–55) of 959 people (mainly of low socioeconomic status) in Melbourne (Australia) and found that the prevalence of those having hepatitis A antibodies as 38 percent in the 6 to 10 year olds, 56 percent in the 20 year olds, and over 97 percent in those over 40 years old. The sera were collected prior to mass poliomyelitis vaccination campaigns, and it was found that the age-related acquisition of poliovirus antibodies was extremely similar to hepatitis A antibodies. This strengthens the possibility that the epidemiology and transmission of polio and hepatitis A infections may be similar and that they share a common response to improved hygiene. A repeat survey in Melbourne in 1975 found antibody prevalences of 23 percent in 6 to 10 year olds, 45 percent in 20 year olds, and 66 to 97 percent in those over 40 years old (Gust and others 1978). The decrease in antibody prevalence presumably reflects an improvement in economic and environmental conditions between the mid-1950s and the mid-1970s.

Villarejos and others (1976) found low prevalence of hepatitis A antibody among people of high socioeconomic status in the Philadelphia area (Pennsylvania, USA). Antibody was not detected in any individual under 20 years old, and the highest age-specific prevalence for hepatitis A antibody was 59 percent in the 40–49 age group. Antibody prevalences were higher among institutionalized individuals in Philadelphia. Szmuness and others (1976) studied 947 randomly selected individuals in New York City (USA) and found an overall prevalence of hepatitis A antibody of 45 percent. Lower social classes had significantly higher prevalences (72–80 percent) than higher social classes (18–30 percent). Jews had a significantly lower prevalence (7 percent) than other whites (39 percent), but sex and homosexuality did not affect prevalence. Antibody prevalence was closely related to age in all groups; for instance, among Chinese the prevalence rose to over 90 percent by the age of 40 years. For both blacks and whites, antibody prevalence was 2.5 to 4 times lower among individuals with postgraduate education than among those without.

These various studies show clearly that transmission of hepatitis A virus is more common, and thus the prevalence of antibodies among children is higher, among people of lower socioeconomic and educational status. This relationship can be detected both between and within countries. These and other aspects of hepatitis A epidemiology, as revealed by serological surveys, have been reviewed by Dienstag and others (1978). Recent investigations of hepatitis A outbreaks in day care centers in the USA have highlighted the major role of children who are not toilet trained (those under 2 years old) in spreading the infection (Hadler 1980; Vernon 1980).

In temperate zones the characteristic seasonal trend has been for a marked rise in incidence in the autumn and early winter months, with a progressive fall to a minimum in midsummer.

## Control Measures

The spread of infection is reduced by simple hygienic measures and the sanitary disposal of excreta. Normal human immunoglobulin may prevent or attenuate a clinical illness but may not always prevent the infection. The use of normal immunoglobulin is of value in the control of outbreaks of infection in specific circumstances, such as in institutions and nursery schools. Following the recent development of a tissue culture technique for hepatitis A virus, hopes of developing a specific vaccine are high. The epidemiology of hepatitis A infection, and approaches to its control, would be altered considerably by the widespread use of such a vaccine.

## Occurrence and Survival in the Environment

There is no direct evidence of the behavior or occurrence of hepatitis A virus in the environment because the virus has never been isolated from an environmental sample. Such isolations must await the development of sensitive and specific serological techniques for detecting small amounts of hepatitis A antigen or routine tissue culture techniques. The recent successful propagation of hepatitis A virus in marmoset liver, fetal rhesus monkey kidney, and other cells gives hope that tissue culture techniques will become available soon.

The available evidence on hepatitis A virus in the environment is indirect. The knowledge that hepatitis A incidence is high in some communities leads to the assumption that hepatitis A virus may be present where fecal pollution is present, and especially where high concentrations of enteroviruses are present (see chapter 9). However, far too little is known about the prevalence of hepatitis A virus excretion, or the numbers of virus particles excreted, to predict at what concentration hepatitis A virus might be found in, for instance, sewage. Some evidence is provided by outbreaks of hepatitis A infection that can be traced by epidemiological analysis to a particular source, such as contaminated water or food or shellfish.

Many outbreaks of hepatitis have been linked to polluted drinking water. An explosive outbreak occurred in New Delhi in December–January 1955–56 (Viswanathan 1957). About 30,000 cases were reported (presumably many cases were subclinical or went unreported) when sewage contaminated the city water supply after heavy rain. This outbreak produced evidence that this presumed hepatitis A virus-like strain is more resistant to chlorination than some enteric bacteria, and this is predictable from the data on the effect of chlorination on enteroviruses (see chapter 9). A more recent report from India (Newaskar, Vidwans and Vachha 1978) also suggests a link between sewage contamination of water supplies and an outbreak of hepatitis in Maharashtra.

Numerous small outbreaks attributed to water pollution are reported from North America and Europe (see Craun 1978). These outbreaks typically are associated with contamination of the water distribution system by cross-connection or back-siphonage or with the use of small untreated rural water supplies that are contaminated by sewage discharges or leaks. Neefe and Stokes (1945) reported an outbreak of 350 cases over 13 weeks at a summer camp in the USA. The outbreak was attributed to seepage from cesspools traveling a horizontal distance of 23–55 meters through fissured red shale and limestone to pollute a well 67 meters deep. Similarly, 24 cases of hepatitis A occurred among 180 residents of holiday cottages near Pilsen (Czechoslovakia) when their well was contaminated by seeping sewage (Vilim and others 1977). However, there is no evidence that water conforming to conventional bacteriological criteria has ever caused hepatitis A, and the disease occurs under conditions similar to those which lead to other waterborne, fecal-oral outbreaks of disease (Grabow 1976).

Known waterborne cases of hepatitis A in the USA range between 35 and 120 year, which is only about 0.4 percent of total reported cases. Recent studies have sought to associate hepatitis incidence in the USA with raw water quality, water treatment practices, or old distribution systems. However, many of these physical parameters are associated with socioeconomic status in the area, and it is well established that hepatitis A incidence is higher in poor and deprived communities. To clarify the situation, Batik and others (1980) compared hepatitis incidence with water supply factors among a sample of nearly 3 million people who experienced 11,633 reported cases of hepatitis A during 1965–77. The comparisons were controlled for age distribution, educational levels, population density, and poverty. None of the water supply source or treatment variables were significantly correlated with hepatitis A incidence.

Another source of circumstantial evidence of hepatitis A virus behavior in the environment are the many documented accounts of hepatitis outbreaks associated with contaminated shellfish (Gerba and Goyal 1978; Hughes, Merson and Gangarosa 1977; Levin 1978). As many as 8.6 percent of reported hepatitis A cases in the USA are associated with shellfish consumption (Levin 1978), usually with consumption of raw oysters and raw or steamed clams. Koff and others (1967) interviewed 270 adult hepatitis patients in Boston (Massachusetts, USA) hospitals and concluded that ingestion of raw or steamed shellfish was as common a source of infection as contact with jaundiced persons, even during a nonepidemic period.

An outbreak of hepatitis A (278 cases) that occurred in Louisiana during October and November 1973 was investigated and shown to be related to simultaneous outbreaks in Texas and Georgia (USA). The outbreak was attributed to the consumption of contaminated oysters harvested from approved growing areas on the east Louisiana coast. The oysters were probably polluted when contaminated river water was discharged into the area during floods earlier in the year.

Oyster fishing was prohibited while the counts of coliform bacteria were in excess of 70 per 100 milliliters. Oysters harvested some 4 weeks after the coliform counts fell below this limit led to the disease outbreak. The evidence suggests that the oysters concentrated the hepatitis virus and retained it for a period of 1.5–2 months. A more severe outbreak was probably avoided because oysters in heavily contaminated areas were killed by changes in salinity (Mackowiak, Caraway and Portnoy 1976; Portnoy and others 1975).

On the basis of the epidemiological evidence, there is good reason to believe that shellfish accumulate and retain hepatitis A virus from polluted waters in the same way that they accumulate and retain poliovirus (chapter 9). There is also good epidemiological evidence that cooking of shellfish does not necessarily inactivate all hepatitis A virus, just as it does not necessarily inactivate all poliovirus. Recent confirmation of this was provided by Peterson and others (1978), who reported that hepatitis A virus injected into oysters and treated at 60°C for 19 minutes could still induce acute disease and seroconversion when fed to fasted marmosets.

## Inactivation by Sewage Treatment Processes

The lack of isolation techniques for hepatitis A virus has prevented any direct studies on the inactivation of the virus by sewage treatment. Indirect evidence on the reaction of hepatitis A virus to chlorination is provided by the work of Neefe and others (1947), who contaminated water with feces known to contain hepatitis A virus, subjected the water to various treatment regimes, and then asked volunteers to drink it. The results indicated that coagulation and filtration reduced but did not eliminate hepatitis A virus but that coagulation, filtration, and chlorination (to provide 0.4 milligrams per liter of free residual chlorine after 30 minutes) eliminated the virus. However, direct chlorination of the contaminated water (to provide a total chlorine residual after 30 minutes of 1 milligram per liter) did not prevent infection in the volunteers, whereas heavier chlorine doses applied to the contaminated water (15 milligrams per liter of total chlorine after 30 minutes) rendered the water noninfective to all volunteers. These results suggest that hepatitis A virus, like the enteroviruses (see chapter 9), is insensitive to combined chlorine and may only be inactivated in sewage disinfection by the application of heavy chlorine doses to highly purified effluents.

It is to be expected that isolation and enumeration techniques for hepatitis A virus will become available over the next few years and will be followed by a surge of investigations into the inactivation of the virus by sewage treatment processes. In the meantime, it is reasonable to assume that hepatitis A virus behaves in a way similar to the enteroviruses (chapter 9). This assumption will become more plausible if it is confirmed that hepatitis A virus is an enterovirus.

## Inactivation by Night Soil and Sludge Treatment Processes

As with sewage treatment processes, no direct evidence is available on the inactivation of hepatitis A virus by night soil or sludge treatment processes. A study by Raška and others (1966) provided indirect evidence of hepatitis A virus survival in cesspool sludge applied to farm land. During the winter months of December and January (1962–63), cesspool sewage was spread on farmland near a small stream used as the source of water for a dairy near Jablonec (Czechoslovakia). On March 11–13 a thaw combined with rain led to the contamination of the stream. The organic content of the water was sufficient to overwhelm the chlorination of the supply, and contaminated water was used by the dairy. The water treatment process included chlorination followed by sand filtration. An epidemic of hepatitis A spread by dairy products occurred during April–June. There were 424 cases, with the highest incidence in the 15–20 age group. Relatively few contact cases occurred possibly due to the mass administration of gamma globulin, particularly to children. The average incubation period was 45 days. There was no increase in other enteric infections, and the authors attribute this either to a lack of the disease organisms in the sewage or to the marginal chlorination having been sufficient to inactivate bacterial pathogens. It is evident from the information supplied that under cold weather conditions hepatitis A virus survived at least 5 to 6 weeks in sewage spread on farmland.

There is some evidence that hepatitis A virus may be more resistant to heat than the enteroviruses discussed in chapter 9. Krugman, Giles and Hammond (1970) found that hepatitis A virus was rendered noninfectious and nonimmunogenic by treatment at 98°C for 1 minute. Deinhardt (1976) reported that some hepatitis A virus survived treatment at 60°C for 1 hour. However, ability to withstand fairly high temperatures ($>60$°C) for short times ($<1$ hour) may not be

indicative of the ability of the virus to withstand cooler temperatures for longer periods (see figure 9-2).

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# 11

# Rotavirus and Viral Gastroenteritis

THE AWARENESS THAT VIRUSES play a major role in the causation of diarrheal disease has been one of the outstanding medical advances of recent time. Previously, surveys of diarrheal disease had failed to determine the causative agent in up to two-thirds of all cases—no known pathogen could be isolated from the stools of these patients. Since 1972, however, several different viruses have been identified in stools by electronmicroscopy and have been shown to be associated with diarrheal disease throughout the world. Rotaviruses have received the most attention and have been accepted as a major cause of childhood gastroenteritis.

## Description of Pathogens and Diseases

A growing number of different viruses are now associated with diarrheal disease (see table 9-1). Rotaviruses appear to be the most important of these and are therefore given greatest attention in this chapter. Several reviews of the rotaviruses have been published (Flewett and Woode 1978; Holmes 1979; McNulty 1978; Steinhoff 1980; Yolken and Kapikan 1979).

### *Identification*

A number of different viruses may cause gastroenteritis, and the disease may vary accordingly. In rotavirus gastroenteritis, the onset is generally quite sudden, and vomiting may be the presenting symptom or may accompany diarrhea at the start. Vomiting is often the dominant feature rather than diarrhea. Fever is present in many cases. Dehydration frequently occurs, but may be more severe in combined infections with pathogenic *Escherichia. coli.* There is no consistent pattern of association between rotavirus and pathogenic *E. coli.* In hospitalized children the fever and vomiting usually resolve in the first 5 days, and recovery occurs within about a week. Mortality rates are low in hospitalized children in developed countries but may be considerable among untreated children in developing countries. Dehydration and shock are the most likely terminal processes, and oral rehydration is as effective in treating viral diarrhea as it is in treating bacterial diarrhea (Nalin and others 1979). Rotavirus gastroenteritis may occur as a single case, or one episode of an epidemic outbreak. There is also a form of continuing infection in some newborn nurseries in which a high proportion of the infants are asymptomatic.

### *Occurrence*

Rotavirus appears to be almost universally distributed in human populations around the world. It has been found in the stools of children with diarrhea from Japan and New Zealand in the East, to Canada and Argentina in the West. It has been identified in tropical as well as temperate climates, although it may not be quite such an important etiological agent for diarrhea in tropical countries as in temperate regions (pathogenic *E. coli* may be more dominant in the tropics; see chapter 13).

### *Infectious agents*

Acute nonbacterial gastroenteritis has long been recognized as a clinical entity. There was epidemiological evidence of outbreaks of infectious diarrheas in which neither bacterial nor parasitic organisms could be found. Volunteer experiments demonstrated that diarrhea could be transmitted by oral administration of bacteria-free fecal filtrates. A particle of sub-bacterial size, presumably a virus, seemed likely. The development of techniques to culture enteroviruses and adenoviruses from stool samples failed to identify any organisms that occurred predominantly in patients with diarrhea. The application of electronmicroscopy

to diarrheal stool samples was the decisive advance. The technique was enhanced by ultracentrifugation, antiserum clumping of particles (immuno-electron-microscopy, IEM), and negative staining. These methods have their limitations because, unless particles occur in concentration of greater than $10^6$ per gram of feces, they may not be detectable. More recent techniques for the identification of rotavirus particles from stools include infected cell immunofluorescence, counter-immunoelectrophoresis, radioimmune assay, and enzyme-linked immunosorbent assay (ELISA), and the methodology is still rapidly improving.

Any form of examination of the feces may give a distorted indication of the pathophysiology of gastroenteritis, a condition in which the primary lesion is in the small bowel. The large bowel is distal to the site of infection and, even in a child, contains some $10^{13}$ bacteria and also many fungi, mycoplasmas, and protozoa. All of these frequently have their own viral infections and may shed particles into the feces. Bacteriophages, unless they have tails, may be very difficult to distinguish from small spherical human viruses. The colon and its flora alter the effluent from the small bowel in a number of ways before it presents as fecal material.

ROTAVIRUSES. These viruses from the stools of children are morphologically identical to those found in the stools of some calves, piglets, foals, lambs, mice, and young monkeys with acute diarrhea. The human virus has been transmitted to a number of these animals. The virus particles are spherical, 70 nanometers in diameter, and made up of double-stranded RNA in two distinct capsid layers that on electron-microscopy give the appearance of a wheel, hence the name rotavirus (figure 11-1*a*). Rotaviruses are now classified as a genus of the family Reoviridae (see table 9-1). There are at least two, and possibly four, serotypes of human rotavirus.

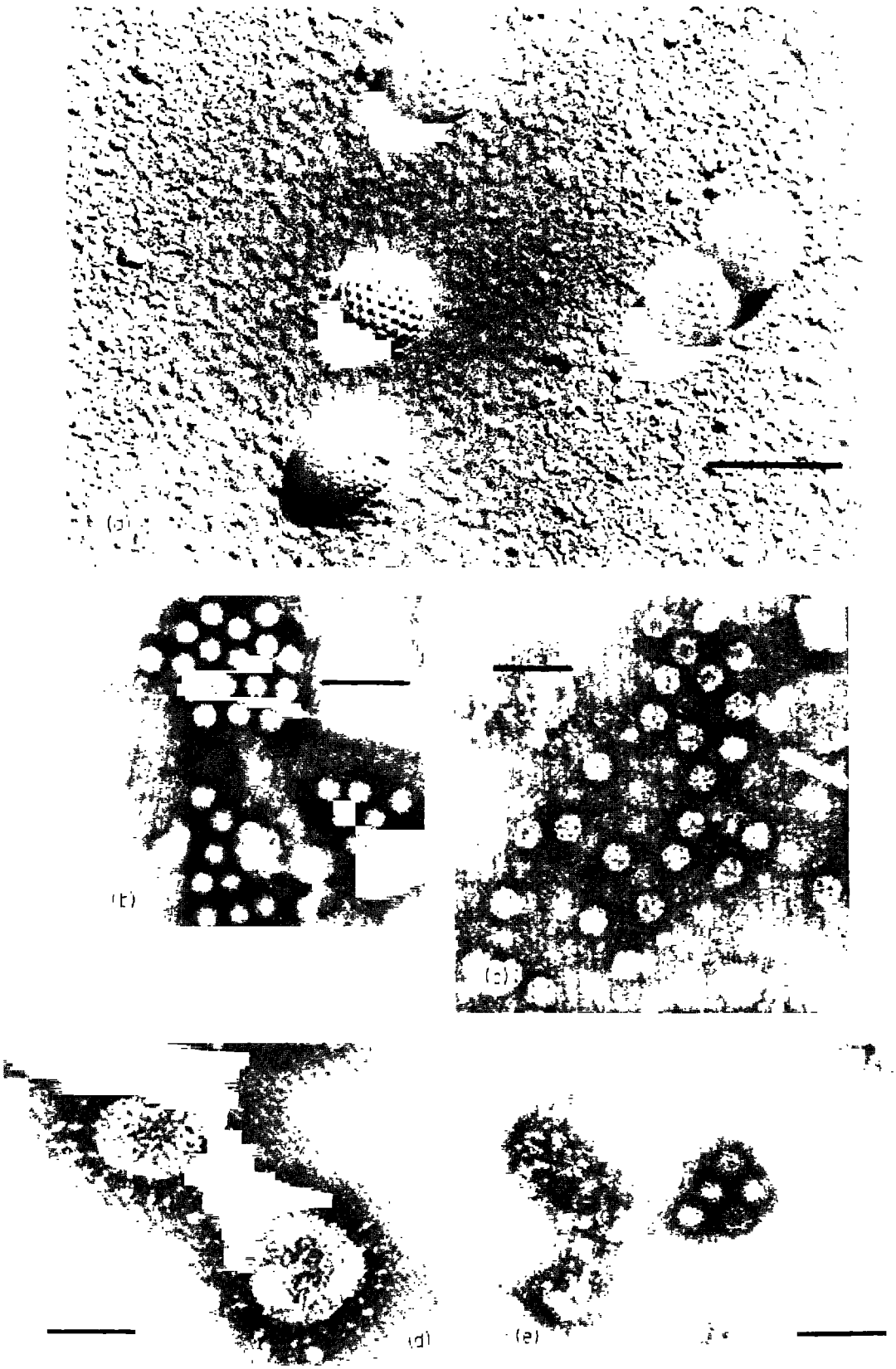
ADENOVIRUSES. These viruses are found in small numbers in feces of some patients with diarrhea, but occasionally in great numbers (see, for instance, Richmond and others 1979). The evidence that adenoviruses can cause diarrhea is circumstantial, and there is no equivalent animal model. However, their occasional presence in large numbers indicates that they must have replicated in and destroyed cells.

ASTROVIRUSES. These are particles of 28 nanometers in diameter that are roughly spherical with surface indentations that result in the appearance of a 5- or 6-pointed star (figure 11-1*b*). They have been found in the feces of normal children and of those with gastro-enteritis (see, for instance, Kurtz, Lee and Pickering 1977). There is still no firm evidence that they are pathogenic.

CALICIVIRUSES. These are picornaviruses of approximately 35 nanometers in diameter (figure 11-1*c*) and were previously known as fecal viruses in such diverse species as sealions, pigs, and cats. Calicivirus has now been associated with several outbreaks of gastro-enteritis and has also been found in the stools of asymptomatic individuals (Schaffer 1979; Studdert 1978).

CORONAVIRUSES. These are well-known agents of acute gastroenteritis in piglets and calves. They vary in shape and size but have a distinctive appearance in electron micrographs (figure 11-1*d*). Recently they have been described in feces from young adults in Britain and from children in Canada and Australia. They have been isolated in tissue culture. They are not proven human enteric pathogens, although they are a major cause of the common cold (Clarke, Caul and Egglestone 1979; McIntosh 1979; Robb and Bond 1979).

Figure 11-1. *Agents of viral gastroenteritis.* (*a*) *Rotaviruses* under scanning electronmicroscopy. Scale bar = 0.1 micrometers. (Photo: J. Cohen, Station de Recherches de Virologie et d'Immunologie, Institute National de la Recherche Agronomique, Thiverval, France.) (*b*) *Astroviruses* under transmission electronmicroscopy, showing the characteristic 5- or 6-pointed star appearance. Scale bar = 0.1 micrometers. (Photo: C. R. Madeley, Royal Victoria Infirmary, Newcastle-upon-Tyne, UK.) (*c*) *Caliciviruses* under transmission electronmicroscopy, showing the characteristic surface hollows. Scale bar = 0.1 micrometers. (Photo: A. J. Zuckerman, London School of Hygiene and Tropical Medicine, London, UK.) (*d*) *Coronaviruses* under transmission electronmicroscopy. The virus particles vary in shape and size and possess a distinctive array of widely spaced surface projections, approximately 20 nanometers long, which give the characteristic "corona" appearance. Scale bar = 0.1 micrometers. (Photo: E. O. Caul, Public Health Laboratory Service, Bristol, UK.) (*e*) *Norwalk agent particles* under transmission electronmicroscopy. Scale bar = 0.1 micrometers. (Photo: E. O. Caul, Public Health Laboratory Service, Bristol, UK)


(a)
(b)
(c)
(d)
(e)

ENTEROVIRUSES. Most studies have indicated that the occurrence of these viruses is no more common in the stools of children with diarrhea than in those from control groups. However, in one outbreak of gastroenteritis, believed to be due to a failure of water supply purification, coxsackie- and echoviruses were found as frequently as *Shigella sonnei* in the stools of those with diarrhea (Green and others 1968).

MEASLES VIRUS. In temperate climates measles is now an exanthem which affects children primarily with respiratory symptoms and a systemic upset. In the past, however, diarrhea was associated with measles and 28 percent of children with measles in a London clinic in 1904 had diarrhea (Balme 1904). In tropical countries where measles is frequently more severe, not only is the rash very prominent, but there is evidence of invasion of the bowel epithelium. The giant cells associated with measles have been seen in the mucosa of biopsy specimens, and the excretion of giant cells in the feces may be prolonged. Diarrhea is frequently associated with measles in the tropics. A study in Guatemala showed that half of the children under 5 years old with measles had acute diarrhea (Scrimshaw and others 1966).

NORWALK AGENT AND OTHER SMALL ROUND VIRUSES. Norwalk agent is a small round virus particle of 27 nanometers in diameter (figure 11-1*e*) that caused an outbreak of nonbacterial gastroenteritis in 50 percent of students and staff of an elementary school in Norwalk, Ohio (USA) in 1968. There was a 32 percent attack rate among family contacts. Rectal swab filtrates produced disease in volunteers, and subsequently the particle was visualized by IEM. Other morphologically similar viruses (Montgomery County agent and Hawaii agent) have been isolated from different epidemics of diarrhea (Dolin 1979). Some of these show a cross-immunity, but several distinct serotypes have been demonstrated. These agents produce a mild, self-limited gastroenteritis that lasts 24–48 hours and affects older children and adults more often than the rotavirus.

Particles of similar size have been associated with small epidemics of winter vomiting disease in Britain: the W and Ditchling agents. These particles differ antigenically from the Norwalk agent and a high proportion of adults appear to have antibody against them. Some patients continued to excrete the particles for over 2 months after the illness. Smaller spherical particles of 25–26 nanometers were recently found in the feces of a high proportion of patients suffering from food poisoning in Britain after eating seafood cocktails containing cockles (Appleton and Pereira 1977). IEM suggests this agent (cockle virus) is antigenically similar to W agent, but different from Norwalk agent.

There is still considerable confusion in identifying viruses in feces, especially when variations in methods, possible artifacts, and the presence of bacteriophages are considered. It is far from proven that all the viruses discussed above cause gastroenteritis, and all have been isolated from healthy persons as well as from those with gastroenteritis. On some occasions, more than one virus has been present. However, it appears likely that, just as the upper respiratory tract reacts to a range of viruses by producing the symptoms of the common cold, the alimentary tract will react to a range of viruses by developing gastroenteritis. It is only the rotaviruses that are unquestionably a major cause of gastroenteritis worldwide, and it is these which are discussed in the rest of this chapter.

### *Reservoirs*

Man is probably the only important reservoir for human rotavirus infection.

### *Transmission*

It is probable that transmission modes are similar to those of the enteroviruses and hepatitis A virus; that is, fecal-oral and usually person-to-person but sometimes via contaminated water, food, or shellfish. Airborne respiratory transmission remains an additional possibility.

### *Incubation period*

Studies that include information about more than one case of rotavirus diarrhea within a family or closed community indicate that the incubation period is between 24 and 72 hours.

### *Period of communicability*

This is very uncertain because many details about the route and mode of spread are unknown. Moreover, rotavirus diarrhea apparently disappears from a community for months at a time in hot weather; also, the organism cannot be found in stools unless it is present in high concentrations ($>10^6$ per gram). If transmission is dependent on the ingestion of a large number of virus particles, communicability from a patient will be at its maximum on about the third to fourth day of the disease, coinciding with the period of maximum virus shedding ($10^{11}$ per gram or more) and

would be unlikely after the eighth day—although excretion of rotavirus can continue for more than 20 days. Asymptomatic infection and excretion certainly occur, but persistent carriage has not been demonstrated.

*Resistance*

Facts are limited by ignorance about the epidemiology and pathophysiology of rotavirus infection. Newborn babies are apparently susceptible to the infection, particularly in the nursery situation, but only a proportion of them develop clinical symptoms. The low pathogenicity at this age may possibly be due to passively acquired maternal immunoglobulins. Although the majority of older children and adults have antibody to rotavirus, adults can be infected—as is shown by rising antibody titers and sometimes by clinical infections. The existence of at least two rotavirus serotypes that are not cross-protective may partly explain repeat attacks.

*Epidemiology*

Rotavirus gastroenteritis is primarily a disease of children, especially those between 6 months and 3 years old. Rotavirus infection can spread very rapidly among neonates in nurseries, but many of these infections are asymptomatic (Jesudoss and others 1979). Rotavirus infection has been recorded in adults, often in association with infection of their children (for instance. Wenman and others 1979; Zissis and others 1976). In all age groups asymptomatic infection is fairly common, but persistent carriage is not demonstrated.

Seroepidemiological surveys show that neonates have a high prevalence of rotavirus antibodies (presumably of maternal origin) that falls over the first 6 months of life. Antibody prevalence then rises again until, by about 3 years, 80–90 percent have rotavirus antibodies, and this high prevalence is maintained thoughout adult life. For instance, a survey of 266 children in Vellore (India) showed that the antibody prevalence was 75 percent among neonates, 30 percent among 5–6 month old infants, and 87 percent among 3 year olds (Jesudoss and others 1978). Similar results are found in affluent communities, indicating that rotavirus transmits successfully even in conditions of good hygiene, pure water, and full sewerage. This suggests direct person-to-person, fecal-oral, or respiratory routes of transmission, particularly within family groups. Parent-to-child, child-to-parent, and sibling-to-sibling spread are all likely (see, for instance, Wyn-Jones, Lillington and Alzaka 1978).

By 18 months of age 85 percent of children in the area of Washington, DC (USA) have acquired antibodies to both Type 1 and Type 2 rotaviruses, and the high antibody prevalence is maintained throughout life (Yolken and others 1978*a*). In contrast, in the same area, only about 10 percent of 3 year old children have Norwalk agent antibodies, and this prevalence rises to only around 50 percent later in life (Kapikian and others 1978). The same picture of rapid acquisition of rotavirus antibodies by nearly all children, contrasted with gradual acquistion of Norwalk agent antibodies (to a maximum prevalence of only 33 percent), was found in Bangladesh (Kapikian and others 1978; Sack and others 1980).

In temperate countries there is a striking seasonal variation, with most cases occurring in the coldest months of the year, whereas in tropical climates (and poorer communities) there appears to be much less seasonal variation. At a children's hospital in Washington, DC (USA) during 1974–78, rotavirus accounted for 39 percent of inpatient diarrhea and 22 percent of outpatient diarrhea. The equivalent figures in January were 71 percent and 62 percent, whereas during June and July they were 4.4 percent and 4.8 percent for inpatients and outpatients, respectively (Brandt and others 1979). A comparative study in Dallas (Texas, USA) and San Jose (Costa Rica) showed that in both settings rotavirus accounted for 50–60 percent of acute nonbacterial pediatric gastroenteritis episodes occurring from December through February. This is the cool period in Dallas and the dry season in San Jose. During the rest of the year the virus was not recovered from any Dallas patients but was found in 30–40 percent of Costa Rican patients in every month except May (Hieber and others 1978).

In developed and temperate countries (such as Australia, Britain, Japan, and the USA), about half of all diarrhea in children that requires hospitalization is caused by rotavirus infection. During the summer, 0–20 percent of cases are rotavirus associated, and in winter this figure rises to 70–80 percent. Studies in developing and tropical countries have indicated that rotavirus accounts for a somewhat lower proportion of hospitalized childhood diarrhea cases—maybe 25–50 percent.

Schnagl, Holmes and Mackay-Scollay (1978) studied 537 episodes of diarrhea among 473 hospitalized children under 6 years old in Western Australia. Among aboriginal children the percentages of 387 diarrheal stools from which known pathogens could be isolated were: parasites 17 percent, rotavirus 16 percent, *Salmonella* or *Shigella* 13 percent, pathogenic *E. coli* 9 percent, adenovirus 3 percent, and astrovirus 2

percent. Among nonaboriginal children the percentages of 150 diarrheal stools from which known pathogens could be isolated were: rotavirus 25 percent, parasites 11 percent, *Salmonella* or *Shigella* 10 percent, pathogenic *E. coli* 10 percent, adenovirus 4 percent, and astrovirus 3 percent. The data suggest the possibility of a winter peak of rotavirus gastroenteritis among nonaboriginals and a summer peak among aboriginals. Rotavirus was detected in the stools of only 2 out of 170 children without diarrhea.

Black and others (1979) investigated 4,498 diarrhea cases reporting to Matlab hospital (Bangladesh) and were able to identify a pathogen in the stools of 85 percent (see table 13-1). Rotavirus was associated with 23 percent of all reported diarrhea cases and with 40 percent of diarrhea cases under 5 years old.

During January–June 1976, Echeverria and others (1978) studied 82 hospitalized infants and children with diarrhea in Manila (Philippines). A viral etiology was indicated in 17 percent of cases, enterotoxigenic *E. coli* in 11 percent, *Salmonella* or *Shigella* in 7 percent, *Vibrio cholerae* in 4 percent, *Giardia lamblia* in 5 percent, and *Entamoeba histolytica* in 2 percent. Ten percent of children had evidence of infection with more than one enteric pathogen. Only 1 out of 49 healthy children had rotavirus particles in their stools. Echeverria and others (1977) found evidence of rotavirus infection in 56 percent (42/75) of children (3 days to 4 years old) with diarrhea seen at hospitals in Taipei (Taiwan) during the summer.

Studies of 293 hospitalized children under 5 years old with diarrhea in Caracas (Venezuela) showed a rotavirus etiology in 41 percent of cases (Viera de Torres, Mazzali de Ilja and Esparza 1978). Only 3 out of 66 healthy children were excreting rotavirus. Espejo and others (1978) studied 242 children under 5 years old with acute diarrhea in two hospitals in Mexico City (Mexico) and found rotavirus excretion in 25 percent. Although the peak of all diarrhea cases in Mexico occurred in June–September, the peak of rotavirus diarrhea occurred in October. The highest age-specific proportions of diarrhea cases with rotavirus excretion were in the 4–10 month age group. Of the 60 children who excreted rotavirus, 22 also excreted *Salmonella*, *Shigella*, or potentially pathogenic serotypes of *E. coli*. Rotavirus infection was less common among breastfed infants with diarrhea (10 percent) than among nonbreastfed infants with diarrhea (27 percent).

Little information is yet available on rotavirus gastroenteritis in Africa. Brookfield and others (1979) detected rotavirus in the stools of 31 percent of 123 hospitalized children under 4 years with diarrhea in Dar es Salaam (Tanzania). Mutanda (1980*a*) studied infants and children with diarrhea at 3 hospitals in Kenya. Rotavirus accounted for 41 percent of inpatients in Nairobi, for 14 percent of inpatients and 17 percent of outpatients in Mombasa, and for 29 percent of inpatients and 11 percent of outpatients in Kisumu (see also Mutanda 1980*b*; Mutanda, Cruickshank and Itotia 1979). Hansen and others (1978) found that 6 percent of adult inpatients with diarrhea in Nairobi had serological evidence of rotavirus infection, whereas 26 percent had *Shigella* and 18 percent had enterotoxigenic *E. coli*.

Some studies have found that rotavirus infection is associated with diarrhea of more than average severity; if this is the case, the proportion of hospitalized diarrhea cases due to rotavirus may be greater than the proportion of all diarrhea cases. Two community studies support this hypothesis. Spencer and others (1980) isolated rotavirus from only 7 percent (5 of 74) of nonhospitalized children under 4 years old with diarrhea in a coastal area of El Salvador. Similarly, rotavirus accounted for only 14 percent (26 of 183) of diarrheal episodes, from which no bacterial or protozoal pathogen could be isolated, among 0–3 year old nonhospitalized children in a highland village in Guatemala (Wyatt and others 1979). If all diarrhea among these children is considered, the proportion due to rotavirus was approximately 7 percent. The incidence of rotavirus diarrhea was estimated at only 1.1 episode per child during the first 3 years of life.

## Control Measures

The spread of infection may be reduced by improved personal and domestic hygiene and by the sanitary disposal of excreta, but this is uncertain. The very high prevalence of antibodies to rotavirus in children over 2 years old in affluent communities indicates that rotavirus transmits successfully even in conditions of near optimum hygiene, water supply, and sanitary facilities.

Infections by rotavirus in breast-fed infants are less likely and less severe than in bottle-fed infants. Breast milk has been shown to contain specific antibodies to rotavirus (Yolken and others 1978*a*), but it now appears that other unidentified properties of breast milk are responsible for its protective effect (Totterdell, Chrystie and Banatvala 1980).

The development of rotavirus vaccines is a distinct possibility within the next few years, but delivering them to the most vulnerable individuals (children aged 5–24 months) will be a difficult task in most developing countries.

## Occurrence and Survival in the Environment

There is no direct evidence on the behavior or occurrence of human rotavirus in the environment because the virus cannot be routinely isolated from environmental samples. Tissue culture methods have been developed (Wyatt and others 1980), but rotavirus does not grow readily in cell culture, and demonstration of cytopathic effects is difficult. Investigations into rotaviruses in the environment must await the development of improved tissue culture techniques or sensitive immunological antigen-detecting techniques.

The available evidence on human rotavirus in the environment is indirect. The knowledge that rotavirus may be excreted in large numbers ($10^{11}$ per gram) by infected individuals, and that incidence of infection appears to be very high in some communities, leads to the assumption that human rotavirus may be present where fecal pollution is present, especially where high concentrations of enteroviruses are found (see chapter 9). However, far too little is known about the prevalence of rotavirus excretion, or about the numbers of viruses excreted by asymptomatic excreters, to predict at what concentration human rotaviruses might be found in, for instance, sewage. Some evidence is provided by outbreaks of gastroenteritis believed to be of viral etiology that have been traced by epidemiological analysis to a particular source, such as contaminated water or shellfish.

Many outbreaks of gastroenteritis have been linked to polluted water, and many of these have had an undetermined etiology and could be of viral origin. Craun (1978) reported that during 1975 and 1976 an etiological agent could not be identified in 75 percent of waterborne gastroenteritis outbreaks in the USA. From observations of the symptoms, it is more than probable that some of these outbreaks were due to rotaviruses or other diarrhea-causing viruses. However, there is no evidence that water conforming to conventional bacteriological criteria has ever caused rotavirus infection, and waterborne gastroenteritis occurs in circumstances similar to those which may lead to other outbreaks of waterborne, fecal-oral gastroenteritis (see, for instance, Morens and others 1979).

Other sources of circumstantial evidence of rotavirus behavior in the environment are the documented accounts of gastroenteritis associated with the ingestion of contaminated shellfish. The so-called cockle virus was detected during outbreaks of gastroenteritis in the UK affecting 797 people who had a common history of eating seafood cocktails containing cockles grown in waters polluted by sewage (Appleton and Pereira 1977).

Dismukes and others (1969) reported 33 cases of gastroenteritis of unknown etiology, and 4 cases of hepatitis, occurring among 128 persons attending a picnic at which raw clams were eaten. Ironically, at an annual convention of a shellfish sanitation association held at New Haven (Connecticut, USA) in November 1968, 19 persons ate raw clams and 17 of them developed acute gastroenteritis of unknown etiology (Ratzan and others 1969). There was subsequently a 37 percent secondary attack rate among family contacts of the 17.

The largest outbreak of viral gastroenteritis so far reported occurred in Australia during June and July 1978 (Murphy and others 1979). At least 2,000 cases were reported throughout the country; cases had a common history of eating rock oysters harvested from polluted estuaries near Sydney. The causative organism was shown to be Norwalk agent. As a result of this outbreak, the New South Wales state government has required that all oysters harvested from the incriminated areas be depurated for at least 2 days in disinfected water, and a panel of volunteers has been set up to test-consume samples of oysters prior to marketing. The data reviewed in chapter 9 on the elimination of enteroviruses from oysters in sterilized water suggest that a 2-day depuration time is inadequate to remove the risk of viral contamination with reliability.

Little is known about the survival of human rotavirus in the environment, and it is reasonable to assume, for the time being, that its environmental behavior is similar to that of the enteroviruses (chapter 9). Simian rotaviruses and reoviruses may provide a closer model for human rotavirus in the environment than the enteroviruses, and recently data on simian rotaviruses in water have been reported. Hurst and Gerba (1980) compared the survival of poliovirus 1, echovirus 7, coxsackievirus B3, and simian rotavirus in clean and polluted freshwaters and in estuarine waters of various salinities (1.2–2.8 percent) at 20°C. All viruses survived for a very similar time, undergoing a 3 log reduction in concentration in 6 to over 14 days in freshwaters and in 2 to 3 days in estuarine waters. This preliminary experiment suggests that simian rotavirus in fresh and saline water exhibits death rates well within the range reported for enteroviruses. Rotavirus is very stable under some conditions. The virus is stable in the pH range 2–9.8 and survives for at least 7 months, but not 4 years, at 18–20°C. Rotavirus in feces remained infectious and virulent for calves after

5 years storage at 4°C. Preliminary studies indicate that rotavirus may resist a temperature of 60°C, but not 63°C, for 30 minutes (G. N. Woode, personal communication).

## Inactivation by Sewage Treatment Processes

The lack of adequate detection techniques has prevented any direct studies on the inactivation of human rotavirus by sewage treatment. However, there is some indication that rotaviruses may be less inactivated in treatment systems than polioviruses.

Farrah and others (1978) compared the ability of poliovirus, human rotavirus and simian rotavirus to adsorb to aluminum hydroxide flocs and activated sludge flocs. Aluminum hydroxide flocs reduced the concentration of poliovirus in tap water by 3 log units but only reduced the concentration of simian rotavirus by 1 log unit or less and did not noticeably reduce the number of rotavirus particles present in a dilute stool suspension. Activated sludge flocs reduced the concentration of added poliovirus by 0.7 to 1.8 log units but reduced simian rotavirus numbers by 0.5 log units or less. This suggests that the adsorptive characteristics of poliovirus and rotavirus are different and that lesser removals of rotavirus occur during water coagulation or activated sludge treatment than have been reported for polioviruses (see chapter 9).

Goyal and Gerba (1979) compared the proportion of 27 different excreted viruses that were adsorbed to a sandy loam soil when shaken in water for 30 minutes. Between 91 and 99.99 percent of all viruses adsorbed to the soil, except for echovirus 1 (55 percent), echovirus 12 (78 percent), echovirus 29 (14 percent), and simian rotavirus (52 percent). However, in another series of adsorption experiments using nine different soils, simian rotavirus tended to adsorb more than all other viruses studied except poliovirus 1, echovirus 7, and bacteriophage T4. A considerable amount of additional experimentation will be required before it is clear whether rotaviruses are less readily adsorbed than enteroviruses or merely less readily adsorbed than poliovirus 1.

The next few years will undoubtedly see many investigations into the removal of rotavirus from sewage and water. Pending the development of adequate concentration and detection techniques for human rotaviruses, studies may be done using seeded simian rotaviruses and reoviruses, which may provide suitable models for human rotavirus.

## Inactivation by Night Soil and Sludge Treatment Processes

As with sewage treatment processes, no direct evidence is available on the inactivation of human rotavirus by night soil or sludge treatment. As noted above, reoviruses may provide a suitable model for environmental studies on human rotaviruses, and several differences in the environmental characteristics of enteroviruses and reovirus are known to exist. For instance, a series of studies by Ward and Ashley (1976, 1977*a*, 1977*b*, 1977*c*, 1978) have shown that reovirus is more heat resistant than poliovirus but that, unlike poliovirus, it is not protected against heat inactivation by being in sludge. These studies also showed that ammonia, although it is highly virucidal to enteroviruses at pH above 8, does not affect reoviruses, and that some detergents sensitize reovirus to heat inactivation while they protect poliovirus.

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# SECTION II

# *Excreted Bacteria*

# 12

# *Campylobacter* and *Campylobacter* Enteritis

THREE CHAPTERS of this book describe recently recognized causes of diarrhea that are now believed to be of major importance throughout the world. These are the rotaviruses and other viruses (chapter 11); the various pathogenic forms of *Escherichia coli* (chapter 13); and some bacteria of the genus *Campylobacter*, which are described in this chapter. Knowledge of *Campylobacter* as a cause of diarrhea in man is recent and limited. The first isolations of the organism from stools of patients with diarrhea came in 1971 in Australia (Cooper and Slee 1971) and in 1972 in Belgium (Dekeyser and others 1972). It was only during 1977 that the scale of the problem became clear in Europe; information on *Campylobacter* enteritis in the USA is even more recent. Very little is yet known about this infection in the developing countries. Several comprehensive reviews have been published since 1977 (Butzler 1978; Butzler and Skirrow 1979; Karmali and Fleming 1979; Skirrow 1977; Smibert 1978).

## Description of Pathogen and Disease

The delayed recognition of the important role of *Campylobacter* as a cause of diarrhea is due to the problems of isolating these bacteria. As strict aerobes growing under low oxygen levels, they will not grow under the aerobic or anaerobic growth conditions used in most laboratories; furthermore, unless selective growth systems are used they are overgrown by other bacteria present in feces. Now that these difficulties have been overcome, it seems that campylobacters are the single most common bacterial cause of diarrhea in several countries (table 12-1). However, the mechanism by which campylobacters cause diarrhea remains obscure. Although they are commonly isolated from fecal specimens, their significance as pathogens is not always clear.

### *Identification*

*Campylobacter* enteritis (also called campylobacteriosis) is an enteric infection caused by *Campylobacter fetus* subspecies *jejuni*. The consequences of the infection vary from asymptomatic excretion or mild symptoms to severe disease. In some affected patients the diarrhea is profuse and watery and is often accompanied by strong abdominal pain, headache, and fever. Dysenteric stools containing blood and mucus are fairly common, especially in children. Vomiting is uncommon. Illness usually persists for a few hours to a few days, but in some patients it may continue for weeks. Rehydration and electrolyte replacement are sometimes required. Antibiotic therapy is usually recommended in severe cases, although its efficacy is not proven. Complications include an abdominal pain of such intensity that acute peritonitis is diagnosed and surgery often undertaken. Reactive arthritis has been reported as a complication of *Campylobacter* enteritis in 2 percent (8 of 340) of cases in Finland (Kosunen and others 1980).

### *Occurrence*

The exact distribution and importance of *Campylobacter* enteritis in various geographical regions are not yet known. It is very probable, however, that *C. fetus* ssp. *jejuni* is a major cause of diarrhea throughout the world (table 12-1).

### *Infectious agent*

Campylobacters are microaerophilic, Gram-negative, motile, slender (0.2–0.4 micrometers in width), curved or spiral bacteria (figure 12-1). They are oxidase positive and do not attack sugars. The genus is divisible into two groups on the basis of the catalase reaction and nitrate reduction test. The organisms

Table 12-1. *Prevalence of excretion of* Campylobacter *and other enteric pathogens by individuals with and without diarrhea in twelve countries*

| *Country* | *Age group* | *Number of persons with (+) and without (−) diarrhea* | *Prevalence of* Campylobacter *excretion (percent)* | *Prevalence of excretion of other bacterial or protozoal pathogens (percent)* | *Source* |
|---|---|---|---|---|---|
| Australia | All ages | +224 | 5.8 (5.4)[a] | ND[b] | Steele and McDermott (1978) |
| | All ages | −530 | 0 | ND | |
| Bangladesh | All ages | +204 | 12.0 | ND | Blaser and others (1980*a*) |
| | All ages | +97[b] | 5.2 | ND | |
| | 1–4 years | +80 | 8.6 | ND | |
| | 1–4 years | +34[b] | 5.9 | ND | |
| | 1–5 years | −141 | 18.0 | ND | |
| Belgium | Children | +3200 | 5.8 | ND | Butzler (1978) |
| | Children | −6500 | 1.7 | ND | |
| | Adults | +600 | 2.3 | ND | |
| | Adults | −700 | 0.7 | ND | |
| Brazil | 0–12 years | +217 | 6.4 | ND | Ricciardi and Ferreira (1980) |
| England | All ages | +280 | 14.0 (12) | 13.0 | Bruce, Zochowski and Ferguson (1977) |
| | All ages | −156 | 0.6 | 1.9 | |
| | All ages | +182 | 7.6 | ND | Dale (1977) |
| | All ages | −60 | 0.2 | ND | |
| | All ages | +860 | 4.2 | 4.4 | Pearson and others (1977) |
| | All ages | +330 | 5.8 | ND | Tanner and Bullin (1977) |
| | All ages | −120 | 0.8 | ND | |
| Indonesia | 0–9 years | +150 | 10.0 | ND | Rockhill and others (1980) |
| | >9 years | +200 | 2.0 | ND | |
| | 0–9 years | +7[c] | 28.0 | ND | |
| | >9 years | +150[c] | 2.0 | ND | |
| | All ages | −ND | <1.0 | ND | |
| Rwanda | Children | +150 | 11 | 42 | De Mol and Bosmans (1978) |
| | Children | −58 | 0 | 31 | |
| Scotland | All ages | +196 | 8.7 (7.1) | 16 | Tefler Brunton and Heggie (1977) |
| | All ages | −50 | 0 | 0 | |
| South Africa | 0–8 months | +47 | 32 (31) | 40 | Bokkenheuser and others (1979) |
| | 0–8 months | −45 | 4 | 15 | |
| | 9–24 months | +31 | 39 (38) | 39 | |
| | 9–24 months | −18 | 44 | 50 | |
| Spain | All ages | +446 | 4.5 | 17 | Lopez Brea, Molina and Baquero (1979) |
| USA | All ages | +238 | 4.6 | ND | MMWR (1979*a*) |
| | All ages | +956 | 4.1 | ND | Blaser and others (1980*c*) |
| | All ages | −548 | 0 | ND | |
| Zaïre | Children | +70 | 8.6 (8.6) | ND | Butzler (1973) |
| | Children | −30 | 0 | ND | |

ND No data.

a. Figures in parentheses refer to *Campylobacter* isolations in the absence of other known bacterial or protozoal pathogens.

b. These patients had dysentry (bloody stools).

c. These patients had suspected typhoid.

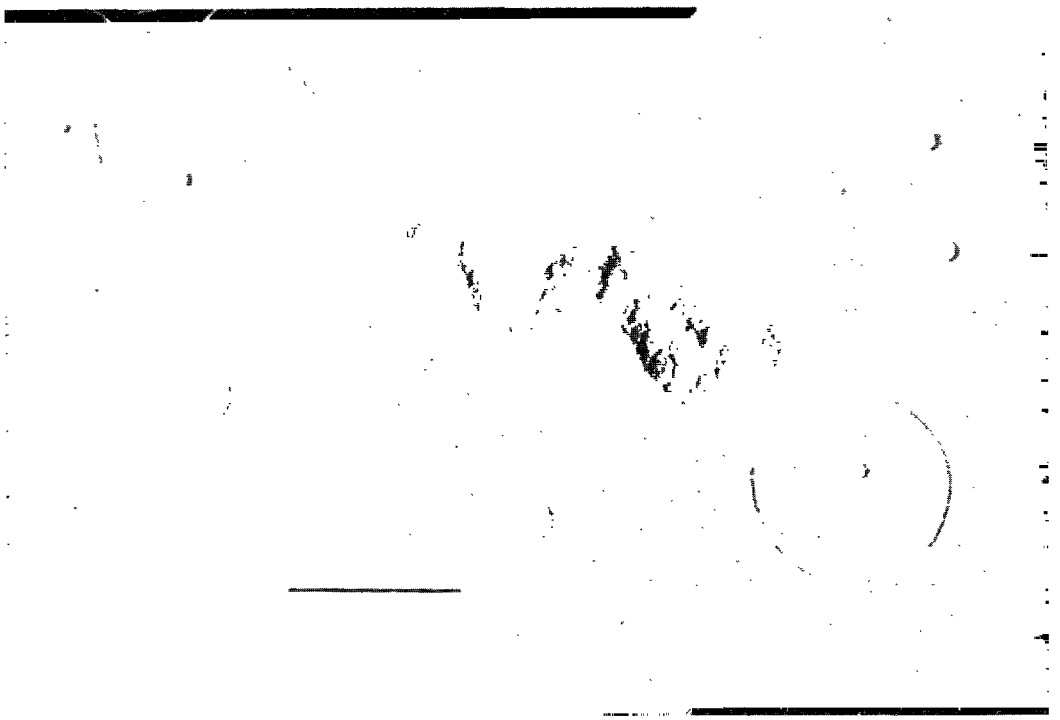

Figure 12-1. Campylobacter *under scanning electronmicroscopy.* The polar flagella are clearly seen. Scale bar = 1 micrometer. (Photo: J. P. Butzler, Hôpital Universitaire Saint Pierre, Brussels, Belgium)

considered in this chapter are catalase positive. Although campylobacters have aerobic metabolism, they are unable to grow in atmospheric oxygen. Growth occurs at oxygen concentrations of between 3 and 7 percent. Incubation on isolation media at 43°C aids the isolation of *C. fetus* ssp. *jejuni*, and incubation at 25°C favors *C. fetus* ssp. *intestinalis* and ssp. *venerealis*. *C. fetus* ssp. *jejuni* has been the most commonly described isolate associated with diarrhea, but the significance of other campylobacters has not been fully assessed. *C. fetus* ssp. *venerealis* causes enzootic sterility in cattle and is transmitted venereally. *C. fetus* ssp. *intestinalis* causes abortion in sheep and cattle, is transmitted by the fecal-oral route, and is a rare, opportunistic pathogen of man. The taxonomy of the catalase-positive campylobacters remains confused. The organism described here as *C. fetus* ssp. *jejuni* is also called *C. jejuni*, or *C. jejuni* and *C. coli*, or, in the older literature, "related vibrios."

### *Reservoirs*

Although it is known that a wide variety of animals and birds may excrete *C. fetus* ssp. *jejuni*, the reservoirs that are functionally important for human infection have not been determined. Studies in developed countries have implicated domestic animals (especially puppies), caged birds, poultry (alive or undercooked), pigs, sheep, and cows as possible sources of human infections (Blaser and others 1980*c*; Bruce, Zochowski and Ferguson 1977; Butzler and Skirrow 1979; MMWR 1978*b*, 1979*a*, 1979*b*; Pearson and others 1977). The degree to which man is an important reservoir for human infection is not clear. Most studies listed in table 12-1 found a very low prevalence (0–1.7 percent) of *Campylobacter* excretion among healthy individuals. By complete contrast, the data from Bangladesh and South Africa (table 12-1) showed that *Campylobacter* are excreted by a substantial proportion of healthy children. The important reservoirs of *Campylobacter* in poor communities in developing countries remain to be elucidated.

### *Transmission*

Transmission is presumed to be fecal-oral, from the feces of infected people, animals, or birds. Infected persons with diarrhea excrete $10^6$–$10^9$ *C. fetus* ssp. *jejuni* per gram of feces. In affluent communities there is little evidence of direct person-to-person spread, except among young children in nurseries. In poor communities and developing countries it is very probable

that person-to-person spread is of considerable importance, although studies are required to confirm this. Several reports in developed countries indicate transmission via undercooked poultry (Butzler and Skirrow 1979; MMWR 1979*a*) and unpasteurized milk (Blaser and others 1979; MMWR 1978*b*; Robinson and others 1979; Taylor, Weinstein and Bryner 1979), and human infection from contact with infected pet animals and birds is also suspected (Blaser and others 1978, 1980*c*; Butzler and Skirrow 1979; MMWR 1979*b*). The organism can persist in chicken and turkey carcasses during preparation and refrigeration for commercial marketing (Simmons and Gibbs 1979). Waterborne transmission was suspected in one major outbreak in the USA (MMWR 1978*a*).

Data on infective dose are not yet available, but a medical laboratory technician in Australia successfully infected himself by ingesting $10^6$ *C. fetus* ssp. *jejuni* in milk (Steele and McDermott 1978).

### *Incubation period*

Incubation periods for *Campylobacter* enteritis are somewhat longer than is common for other bacterial enteric infections. Recorded or estimated incubation periods are from 1.5 to 11 days, but usually are between 3 and 5 days.

### *Period of communicability*

One study in the USA (Blaser and others 1980*c*) found that fecal carriage of *C. fetus* ssp. *jejuni* was for a median period of 15 days from the onset of illness. Most patients were not excreting the organism after 21 days, and the maximum period of excretion recorded was 7 weeks. Jones (1979) reported excretion of the pathogen for 18–39 days by 12 adult patients employed at a food factory in England.

### *Resistance*

The disease has been described in both children and adults. Circulating antibody can be detected, and it may well be that some immunity is conferred by infection.

### *Epidemiology*

The epidemiology of *Campylobacter* enteritis is poorly understood in the developed countries and totally obscure in the developing countries. It seems clear that it is a zoonosis, and it may be that the nearest known parallel is with the epidemiology of the zoonotic salmonelloses (chapter 15). It is possible that the dominant route of transmission, in developed countries, is from infected animals to man, either as a result of handling pets or farm animals or as a result of ingesting poorly cooked meat (especially poultry) or unpasteurized milk. If this is the case, then its epidemiology in developed countries should indeed be very similar to the salmonelloses. This interpretation would also explain why *C. fetus* ssp. *jejuni* is such a prominent cause of bacterial diarrhea, even in affluent communities with high standards of environmental sanitation (table 12-1). Reportings of *Campylobacter* enteritis tend to peak during the warm summer months in England and Wales, Belgium, and the USA (Butzler and Skirrow 1979).

The data in table 12-1 show that *C. fetus* ssp. *jejuni* has been associated with 4–14 percent of diarrhea cases in developed countries. In these same countries, the prevalence of *Campylobacter* excretion by healthy persons is low (0–1.7 percent). The picture in developing countries is unclear. In some (for instance, Indonesia, Rwanda, and Zaïre) the prevalences of *Campylobacter* infection among those with and without diarrhea are similar to the prevalences reported from developed countries. In Bangladesh and South Africa, however, a very different picture has emerged. In Bangladesh, 18 percent of 141 village children (1–5 years old) were excreting *Campylobacter* during the dry season, whereas only 2 percent were excreting *Shigella*, and none were excreting *Salmonella* (Blaser and others 1980*a*). Fifty-two percent (13 of 25) of these *Campylobacter*-positive children had had no history of diarrhea in the 30 days prior to specimen collection. The prevalence of *Campylobacter* excretion in the 12–23 months age group was 39 percent. Similarly, in Soweto (South Africa), Bokkenheuser and others (1979) reported a 44 percent prevalence of *Campylobacter* excretion among healthy, black children age 9 to 24 months (see also Koornhof and others 1979). While it seems certain that *C. fetus* ssp. *jejuni* is a cause of some diarrhea in developing countries, the relative importance of this etiologic agent in comparison with other known major agents (especially enterotoxigenic *E. coli* and rotavirus) remains undetermined.

Data from England (Butzler and Skirrow 1979; Dale 1977) suggest that the highest incidence of disease occurs among people 5 to 34 years old, whereas some reports from developing countries suggest that infants and young children are the most affected (Blaser and others 1980*a*; DeMol and Bosmans 1978; Ricciardi and Ferreira 1980). This, in turn, suggests the possible importance of person-to-person transmission in

developing countries. However, Blaser and others (1980*a*) found that *Campylobacter* infection among children in a rural area of Bangladesh was not clustered by household and considered that there might be "relatively little person-to-person transmission."

An outbreak of *Campylobacter* enteritis affected 2,000 out of the 10,000 inhabitants of Bennington (Vermont, USA) during a 2-week period in May–June 1978 (MMWR 1978*a*). All parts of the town were involved, and there was no evidence of secondary person-to-person spread. The town water supply was partially chlorinated, but not otherwise treated, and several areas of the town were receiving water with no residual chlorine over the period of the outbreak. No *Campylobacter* was isolated from the water, but the water supply was strongly implicated as the common source of the outbreak.

## Control Measures

Very little can be said with certainty about the control of *Campylobacter* enteritis until its epidemiology is further understood. Hygienic excreta disposal, good personal and domestic cleanliness, adequate cooking of poultry and care in handling pets and farm animals are all presumed to be important protective measures.

## Occurrence and Survival in the Environment

Although it is known that *C. fetus* ssp. *jejuni* is excreted by a wide variety of animals and birds, almost no data exist on the presence of these organisms in the environment. Knill, Suckling and Pearson (1978) isolated *C. fetus* ssp. *jejuni* from 21 percent (7 of 34) of seawater samples, and from 74 percent (37 of 50) of river and pond samples, in the Southampton area (UK). All positive water samples also contained *E. coli.*

Very little is yet known about the survival of *C. fetus* ssp. *jejuni* in various environmental habitats. In one study, *C. fetus* ssp. *jejuni* was enumerated in stored feces, urine, water, and milk (Blaser and others 1980*b*). In naturally infected feces, a 7 to 9 log reduction occurred in 9 to 22 days at 4°C and in 3 to 8 days at 25°C. In urine, high initial concentrations became undetectable in less than 2 days at 37°C, but organisms were viable for up to 35 days at 4°C. In autoclaved stream water, a 7 log reduction took 5 to 33 days at 4°C and 2 to 4 days at 25°C. In pasteurized milk, maximum survival times were 22 days at 4°C and less than 3 days at 25°C. Comparative studies in acid and water showed that survival was significantly curtailed at pH values of less than 3. A 7 log reduction occurred in 20 minutes at pH 2.4

Of peripheral interest are the experiments of Lindenstruth and Ward (1948) with *Vibrio fetus*, which might now be classified as *Campylobacter fetus* ssp. *intestinalis*. They showed that, at 20°C and 37°C, inoculations of $1.5 \times 10^9$ organisms survived for 10 days but not for 20 days in hay, soil, and sheep manure. At 6°C, the same inoculation in the same environments survived for 20 days but not for 30 days.

## Inactivation by Sewage Treatment Processes

No information is available on the destruction of *C. fetus* ssp. *jejuni* by sewage treatment processes or on the occurrence of this organism in sewage.

## Inactivation by Night Soil and Sludge Treatment Processes

No information is available on the destruction of *C. fetus* ssp. *jejuni* by night soil and sludge treatment processes or on the occurrence of this organism in night soil and sludge.

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# 13

# Pathogenic and Nonpathogenic *Escherichia coli* and Other Bacterial Indicators of Fecal Pollution

THIS CHAPTER combines two distinct areas of knowledge. The first two sections ("Description of Pathogen and Disease" and "Control Measures") cover recent information on the role of certain types of *Escherichia coli* as major causes of acute diarrhea in many countries. Subsequent sections of the chapter briefly review the enormous compilation of literature on the fecal indicator bacteria, which have been used for 80 years as a measure of the degree of fecal contamination of the environment.

## Description of Pathogen and Disease

In the last 30 years, and especially in the last 10 years, it has become clear that various forms of *E. coli* are a major cause of diarrhea. This section briefly reviews *E. coli* diarrhea.

### *Identification*

Diarrhea produced by *E. coli* cannot be differentiated clinically from similar disease produced by other enteric pathogens. The spectrum of disease includes a cholera-like syndrome produced by enterotoxigenic organisms, a dysentery-like syndrome caused by enteroinvasive organisms, and many milder forms of diarrhea. Asymptomatic infection is very common. The severity of disease caused by the enterotoxigenic *E. coli* depends upon the degree of dehydration, and treatment is primarily by rehydration and electrolyte replacement—oral rehydration having proved very effective in most patients. Death rates of 5–10 percent may be experienced among untreated infants and children but are very low among those receiving rehydration therapy. Diagnosis of *E. coli* diarrhea is of limited clinical value and is, in any case, difficult because all patients are excreting large numbers of commensal *E. coli*, and the laboratory methods for identifying the suspected pathogens are complex and slow. The magnitude of the problem results from the fact that *E. coli* virulence factors are plasmid encoded and may be transmitted to many other Enterobacteriaceae.

### *Occurrence*

Gastroenteritis due to *E. coli* occurs in all parts of the world. Particular types of enterotoxigenic *E. coli* apparently cause infantile diarrhea in particular countries. It is thought that the acquisition of such infantile strains is one of the major causes of travelers' diarrhea. Enterotoxigenic *E. coli* appears to be a more important cause of diarrhea in developing countries than in developed countries. It may be that certain enteroinvasive strains, causing disease in adults, are also of restricted geographical distribution.

### *Infectious agents*

*E. coli* is a Gram-negative, rod-shaped bacterium belonging to the family Enterobacteriaceae (figure 13-1). These organisms are usually thought of as lactose-fermenting saprophytes, in contrast with the non-lactose fermenting *Salmonella* spp. and *Shigella* spp. Lactose-fermenting *Salmonella* spp. and *Shigella* spp. do occur, however, and non-lactose-fermenting *Escherichia coli* may be common from some sources.

*E. coli* is a normal inhabitant of the intestinal tract of man and many other animal species. Conventional biochemical tests used in the identification of bacteria

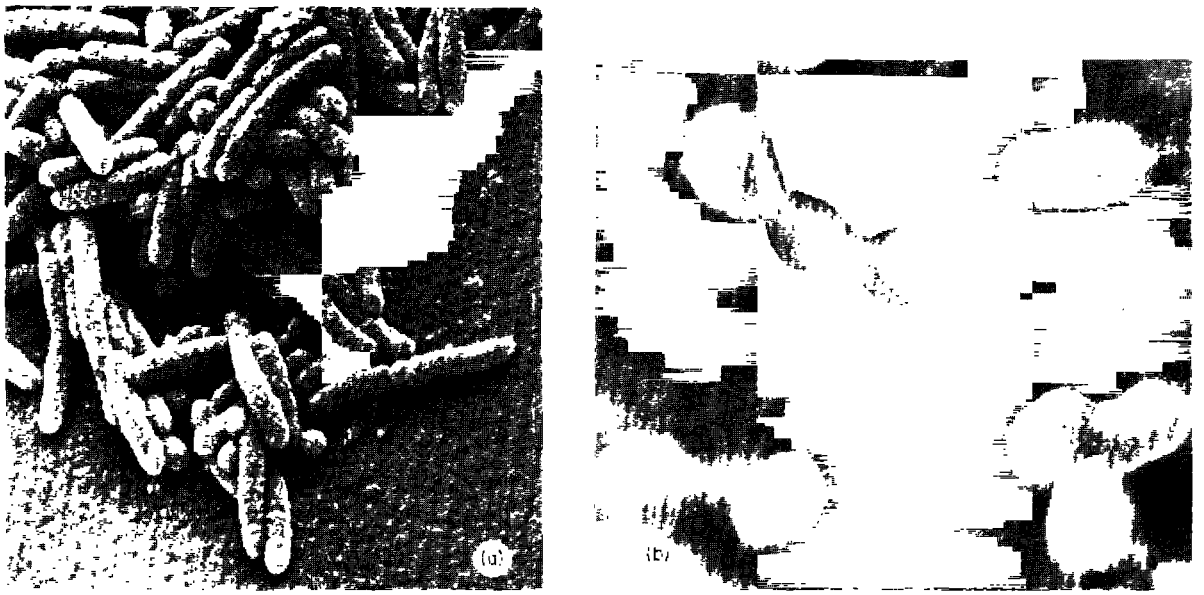

Figure 13-1. Escherichia coli *and* Streptococcus faecalis *under scanning electronmicroscopy.* (*a*) *E. coli.* Scale bar = 1 micrometer. Reproduced by permission of David Scharf 'Pile of *E. coli* cells', published in *Scientific American* Vol 237 No 1 July 1977 pp 22–23.) (*b*) *S. faecalis.* Scale bar = 1 micrometer. (Photo: N. J. Horan, Department of Civil Engineering, University of Leeds, Leeds, UK)

do not yield information that enables these saprophytic organisms to be differentiated from the pathogenic strains. Serological tests are more useful. Particular serological groups, distinguished by their somatic antigen (O antigen), are commonly associated with gastrointestinal disease. However, a particular strain cannot be assumed to be a pathogenic representative of a particular O group, unless a pathogenic mechanism (toxin production or invasiveness) can be demonstrated or epidemiological evidence links the strain to an outbreak.

It is valuable to distinguish between three different types of pathogen within the *E. coli* group, a description of which follows.

ENTEROTOXIGENIC *E. COLI* (ETEC). Enterotoxigenic strains of *E. coli* can be the cause of a cholera-like syndrome in infants, children, and adults. ETEC produce either a heat-labile enterotoxin (LT), serologically related to cholera enterotoxin, or a heat-stable enterotoxin (ST), which are structurally heterogeneous and may consist of LT complexed to endotoxin. Some strains produce both toxins. Action of LT is analogous to that of cholera toxin. Production of enterotoxin is controlled by extrachromosomal transferable DNA (plasmids). The ability to accept these plasmids may be enhanced by particular O group antigens but, although this may be important in nature, in the laboratory enterotoxin plasmids (ENT) can be transferred to nonpathogenic O groups. O groups particularly associated with toxigenicity are 6, 8, 15, 20, 25, 78, 115, 128, 148, 159.

The ability to cause disease depends not only on the production of enterotoxin but also upon the ability to colonize the intestine. Various colonization factors, or adhesins, have been described that enable the bacteria to attach to the small intestinal mucosa. These adhesins are plasmid controlled and are associated with hair-like protein structures on the bacterial cell, known as pili or fimbriae. There is now extensive evidence that the presence of one or more of three piliate bacterial antigens (K88, K99, and 987P) is required for successful colonization by ETEC of the small intestine of piglets and calves. More recent work has identified two pili, CFAI and CFAII, as the adhesins of functional importance in human infection. There is some degree of host specificity among adhesins: K88 is especially associated with piglet infections, and K99 is associated with calves and lambs.

ENTEROINVASIVE *E. COLI* (ETEC). Enteroinvasive *E. coli* produce disease by a mechanism similar to that of *Shigella* spp. These organisms invade the colonic mucosa and cause bloody diarrhea. The property seems to be restricted to a few O groups, O groups particularly implicated are 28, 112, 115, 124, 136, 143, 144, 147, 152, 164.

ENTEROPATHOGENIC *E. COLI* (EPEC). Organisms belonging to this group were first recognized as a result

of the serological examination of strains of *E. coli* isolated from outbreaks of diarrheal disease among infants. Though undoubtedly some enterotoxigenic and enteroinvasive strains have been included in this group, the pathogenic mechanism employed by most of these organisms is not known. These strains have been particularly associated with outbreaks of infantile gastroenteritis; they may, however, cause disease in adults. Experiments in adult volunteers have shown that nontoxigenic and noninvasive strains of *E. coli* isolated from epidemics are able to produce diarrhea O groups particularly implicated are 18, 20, 25, 26, 28, 44, 55, 86, 111, 112, 114, 119, 125, 126, 127, 128, 142.

*Reservoirs*

It seems likely that pathogenic *E. coli* is transmitted from man to man. Studies on infection in pigs and calves, when considered in the context of the problems that have been experienced in developing animal models of human infection, suggest a considerable degree of host specificity. Nonpathogenic or commensal *E. coli* are numerous in the gut of all warm-blooded animals and for this reason have been widely used as indicators of fecal pollution of the environment.

*Transmission*

In nursery outbreaks, the main route of transmission is by way of the hands of those nursing infected infants. It seems likely that fecal contamination of the environment, fomites, and hands constitute the primary means of transmission and infection both among children and adults. Water- and foodborne outbreaks have been described.

As with the other bacterial enteric pathogens (except *Shigella*), large numbers of ingested organisms are required to produce infection in healthy adults. Ferguson and June (1952) fed an EPEC serotype in milk to adult male prisoners in the USA. A dose of $6.5 \times 10^9$ produced moderate or severe diarrhea in seven of eleven volunteers, whereas a dose of $5.3 \times 10^8$ produced comparable symptoms in only one of twelve subjects. In subsequent experiments, in the same prison (June, Ferguson and Worfel 1953) with a different EPEC serotype, moderate or severe symptoms were produced by $1.6 \times 10^{10}$ organisms in three of eight volunteers, by $5.3 \times 10^9$ in one of eight, by $1.7 \times 10^9$ in one of seven, and by $1.4 \times 10^8$ in one of eight.

DuPont and others (1971) sought to infect adult male volunteers with ETEC and EIEC strains in milk. An ETEC strain associated with diarrhea in piglets failed to cause diarrhea in fourteen volunteers at doses of $10^6$–$10^{10}$. Two ETEC strains isolated from patients in Vietnam caused mild diarrhea (three watery stools in 24 hours) in three out of ten volunteers at a dose of $10^8$, and severe diarrhea (ten or more watery stools over 48 hours) in seven out of ten volunteers given a dose of $10^{10}$. Two EIEC strains at a dose of $10^8$ caused diarrhea (mild or severe) in eight of thirteen volunteers and dysentry in three of thirteen volunteers; at a dose of $10^6$ they caused diarrhea in one of fourteen volunteers; and at a dose of $10^4$ they caused no diarrhea in ten volunteers. However, when the $10^6$ dose of EIEC was preceded by 2 grams of sodium bicarbonate, severe diarrhea and dysentery were induced in two of three volunteers. These results suggest a median infective dose ($ID_{50}$) of around $10^9$ for the ETEC strains tested and an $ID_{50}$ of around $10^8$ for the EIEC strains tested. Those infected excreted $10^8$ EIEC per gram and $10^8$–$10^9$ ETEC per gram of feces.

*Incubation period*

Most reports suggest an incubation period of 6 to 72 hours.

*Period of communicability*

The organisms are excreted typically for 3–5 days, but sometimes for 2–3 weeks. Colonization of the intestine in which saprophytic *E. coli* is replaced by pathogenic *E. coli* can occur. Asymptomatic carriers of ETEC and EPEC have frequently been reported.

*Resistance*

The presence of the receptors for K88 antigen in the small intestine of the pig is genetically determined. Pigs lacking the receptors cannot be infected by ETEC. It seems likely that among human populations similarly unsusceptible individuals may occur. Over 50 percent of children have antibodies to common EPEC serotypes by the age of 1 year. These seem to confer resistance to infection. Adults are susceptible to strains they have not previously encountered. Neonates and infants are the most susceptible group, and breast feeding may confer some protection.

*Epidemiology*

The great importance of ETEC in childhood diarrhea in developing countries has only recently been recognized. Knowledge of which strains of *E. coli* cause diarrhea in which ways, and how these infections may

be diagnosed and categorized, is increasing rapidly. The newness and fluid state of the subject mean that understanding of *E. coli* epidemiology is limited and subject to continual revision. In addition, the laboratory techniques for declaring a particular organism to be ETEC (LT, ST, or LT + ST), EIEC, or EPEC are developing and are not entirely standardized among different laboratories. Much of the data produced by various surveys in the last five years are therefore not strictly comparable.

Several studies in different parts of the world have shown that *E. coli* is a major cause of diarrhea, especially among young children in poor communities. Sack and others (1975*a*) studied 59 Apache children, all under 5 years of age, hospitalized with acute watery diarrhea at Whiteriver (Arizona, USA). These patients had sixty-four episodes of diarrhea of which 9 percent were associated with ETEC, 11 percent with EPEC, 20 percent with *Shigella*, 3 percent with *Salmonella*, and 6 percent with ETEC plus another bacterial pathogen.

Guerrant and others (1975) studied forty infants and children (age 9 days to 10 years) admitted to the hospital in Florianópolis (Brazil) with diarrhea. A potential pathogen was isolated from thirty-one cases (78 percent). ETEC alone were isolated from twenty cases (50 percent), ETEC plus EIEC from five cases, ETEC plus *Salmonella* from two cases, EIEC alone from two cases, EIEC plus *Salmonella* from one case, and *Salmonella* alone from one case. Only one of twenty healthy controls was excreting ETEC.

Sebodo and others (1977) examined stool specimens from forty-one hospitalized children with acute diarrhea and sixteen healthy control children (all under 2 years old) in Central Java (Indonesia) in January and February. Among those with diarrhea, 15 percent excreted rotavirus, 12 percent excreted EPEC, 24 percent excreted ETEC, 2 percent excreted *Salmonella*, and 2 percent excreted *Shigella*. Among those without diarrhea, the equivalent percentages were 0, 19, 58, 0, and 0, respectively.

Freiji and others (1979) found that ETEC and EPEC accounted for around 6.6 percent of reported childhood diarrhea cases in the dry season (March–April), and around 14 percent in the wet season (May–June), in Addis Ababa (Ethiopia). The equivalent proportions for rotavirus were 11 and 34 percent in the dry and wet seasons, respectively.

Koornhof and others (1979) recorded in South Africa that 33 percent of 479 black and white children with diarrhea, mostly under 2 years of age, were excreting EPEC serotypes compared with only 15 percent of 498 age-matched healthy control children. Among those excreting EPEC, children with diarrhea excreted significantly greater numbers than healthy control children. ETEC were associated with only 10 percent of diarrhea cases, and no EIEC were recovered. EPEC appeared to be particularly prominent as a cause of diarrhea during the annual summer diarrhea peak.

In studies conducted prior to about 1970 it was common to fail to identify a known pathogen in the stools of approximately 70 percent of diarrhea cases (see, for instance, Gordon 1964). The enormous progress in diarrheal etiology is illustrated by studies in Bangladesh. One study investigated forty-eight patients with diarrhea admitted to Matlab hospital who did not have *V. cholerae*, *Salmonella*, or *Shigella* in their

Table 13-1. *Etiology of diarrhea reported to Matlab Hospital, Bangladesh, during 1977*

| | *All patients* | | *Patients under 5 years* |
|---|---|---|---|
| *Pathogen* | *Annual incidence per 1,000* | *Percentage with stated infection* | *Percentage with stated infection* |
| Enterotoxigenic *E. coli* | 8.1 | 25 | 25 |
| Rotavirus | 7.5 | 23 | 40 |
| *Vibrio cholerae* | 3.7 | 12 | 5 |
| Other vibrios | 3.1 | 9 | 5 |
| *Shigella* | 1.5 | 5 | 5 |
| *Salmonella* | | <1 | <1 |
| *Giardia lamblia* | | 2 | <1 |
| *Entamoeba histolytica* | | 3 | <1 |
| Mixed | | 6 | 10 |
| Unknown | | 15 | 10 |
| All diarrhea-causing | 30.3 | 100 | 100 |

*Note:* *Campylobacter* was not included as a possible cause of diarrhea.
*Source:* Adapted from Black and others (1979).

stools (Ryder and others 1976). Twenty-three percent had ETEC infection, and they were all over 2 years old; 29 percent had rotavirus infection, and they were all under 2 years old. ETEC was isolated from 2 percent (10 of 575) of healthy individuals in the community and from one out of thirty-nine contaminated water sources.

Black and others (1979) investigated 4,498 diarrhea cases reporting to Matlab hospital and were able to identify a pathogen in the stools of 85 percent of all cases, and in the stools of 90 percent of cases under 5 years old. Their results are reproduced in table 13-1. ETEC and rotavirus alone were associated with 48 percent of all reported diarrhea and 65 percent of diarrhea in children under 5 years. Children under 5 years constituted 57 percent of all diarrhea cases reporting to Matlab hospital. Table 13-1 also shows that the estimated incidence of reported ETEC diarrhea was over twice that of reported cholera, even though during 1977 the cholera incidence was nearly double the yearly average for that area. The incidence of reported ETEC diarrhea was around 8 times higher in children under 2 years than in all age groups over 4 years. ETEC showed a marked peak in August, compared with a cholera peak that year in September–October. Dehydration was moderate-to-severe in 33 percent of ETEC reported diarrhea cases and in 70 percent of reported cholera cases.

Further studies in Bangladesh (Black and others 1979) investigated family contacts of 82 index cases of diarrhea associated with ST- or ST + LT-producing ETEC of four serogroups (O6, O8, O78 and O115). Out of 446 family contacts, 53 (12 percent) became infected with the same serogroup and toxin type of ETEC as the index case within 10 days. Of the 53 infected contacts, 20 (38 percent) developed diarrhea. Among children under five years old, the proportion of contacts infected was 25 percent (21 of 84) and the proportion of infected individuals with diarrhea was 67 percent (14 of 21). Water sources used by households having index cases were investigated, and 10 percent (15 of 152) yielded ETEC of the same serogroup and toxin type as that infecting the index case, whereas only 0.7 percent (1 of 144) of sources used by control households were contaminated by ETEC. Ditch, tank, and canal waters were more often positive for ETEC than tubewell or river water, and 7 of the 15 ETEC-positive sources were used for drinking, whereas the remaining 8 were used for bathing and cooking. The proportions of other environmental and livestock samples containing ETEC, in case and control households respectively, were 0.8 percent (2 of 244) and 0.4 percent (1 of 242) of stored drinking water, 1.4 percent (4 of 287) and 0 percent (0 of 220) of food, 1.3 percent (3 of 237) and 0 percent (0 of 222) of heathy cows, and 0 percent (0 of 127) and 0 percent (0 of 100) of healthy goats. Contacts of index cases in houses having ETEC-positive drinking water or cooked food had a risk of infection 4.5 times that of contacts living in houses with no ETEC-positive environmental or animal specimens.

Some studies, however, have shown either that *E. coli* were not associated with a major proportion of diarrhea cases, or that the prevalence of pathogenic *E. coli* excretion among diarrhea patients was the same as that among healthy controls. Echeverria and others (1977) studied eighty infants and children (age 3 days to 4 years) with diarrhea seen at three hospitals in Taipei (Taiwan) during the summer. Fifty-six percent of cases were associated with rotavirus. The proportion of children excreting ETEC was low (7 percent) and was not significantly different from the proportion of healthy controls excreting ETEC. Three out of six sick children with bacteriological or serological evidence of LT-producing ETEC infection, and four out of five children with ST-producing ETEC, also had rotavirus infection.

Echeverria and others (1978*a*) studied eighty-two children (age 6 months to 13 years) with diarrhea in a hospital in Manila (Philippines). ETEC were isolated from 11 percent of these children and from 8 percent (4 of 49) of healthy controls. Rotavirus infection was associated with 17 percent of the diarrhea cases, *Salmonella* with 6 percent, *Shigella* with 1 percent, *Giardia* with 5 percent and *Entamoeba histolytica* with 2 percent. To determine the source of these infections, human, animal, and environmental samples were collected in the town of San Jose and examined for ETEC (Echeverria and others 1978*b*). Five out of 1,086 human fecal samples (individuals without diarrhea, ages not stated) were positive for ETEC (all LT-positive, ST-negative). ETEC were also isolated from two of twenty-eight pigs, one of ten water buffalo, zero of twenty-six pieces of beef, zero of twenty-five pieces of pork, zero of fifty-two vegetable samples and zero of forty-seven polluted water samples. The meat, vegetable, and water samples were contaminated with *E. coli*. None of the ETEC serotypes from livestock were the same as those from humans.

Spencer and others (1980) studied 156 cases of diarrhea occurring over a 1-year period among 2,400 people living in a rural, coastal area of El Salvador. Stool specimens were collected from all diarrhea cases and were compared with stools from healthy age- and sex-matched controls living nearby. ETEC were isolated as frequently from controls (13 percent) as from cases (12 percent). ETEC-producing LT were isolated more

frequently in children under 5 years among both cases and controls. ETEC isolations were more common among cases during April–June—the beginning and middle of the rainy season and the time of the annual diarrhea peak. Among controls, there was no clear seasonality of ETEC isolation. EPEC serotypes were isolated from 8 percent of cases and controls, and no EIEC were isolated. This study in a rural non-hospitalized population failed to show an association between ETEC or EPEC infections and diarrhea.

Although ETEC is a major cause of diarrhea in Bangladesh (table 13-1), Gilman and others (1980) reported that EIEC was not a major cause of dysentery. Of 132 nonamebic dysentery cases in Dacca, 81 percent excreted *Shigella*, 1 percent excreted *Vibrio parahaemolyticus*, and 1 percent excreted EIEC.

Although information is scanty, ETEC does not appear to be a major cause of diarrhea in many developed countries. Gangarosa (1978) concluded that ETEC was not readily transmitted in highly sanitized environments and had not become a significant problem in the USA despite multiple introductions by infected travelers returning from abroad (see below).

Transmission of ETEC and EPEC among small children and infants is clearly vigorous where hygiene is less than optimal. This is suggested by the very high age-specific incidences of infection and disease in children under 2 years old compared with older age groups and by serological surveys showing that a high proportion of children have antibodies to particular forms of *E. coli* or their toxins. Studies among the Apache at Whiteriver (Arizona, USA) showed widespread exposure to LT among small children (Sack and others 1975*b*).

Travelers' diarrhea has attracted increased attention recently, and it is now clear that a substantial proportion of cases are caused by ETEC. An adult visitor to a country, especially an individual from a developed country visiting a developing country, may be immunologically unprepared for the strains of *E. coli* that will be encountered. Kudoh and others (1979) studied 320 Japanese travelers returning to Tokyo with acute diarrhea. The following pathogens were associated with various percentages of the diarrhea cases: ETEC 30 percent, EIEC 1 percent, EPEC 1 percent, *Shigella* 6 percent, *Salmonella* 12 percent, *V. parahaemolyticus* 7 percent, and non-O1 *V. cholerae* 2 percent. Of the 95 individuals with ETEC infection, 20 also had other bacterial infections, most commonly salmonellosis. Echeverria and others (1979) reported that ETEC was the most common identifiable enteric pathogen among Americans with diarrhea at Clark Air Force Base in the Philippines.

Sack and others (1978) studied thirty-nine Peace Corps volunteers during the first few weeks of their work in Kenya. Eighteen of the volunteers were given a daily dose of 100 milligrams of doxycycline (a derivative of tetracycline) as a prophylactic, and only one (6 percent) of this group developed diarrhea—caused by an antibiotic-resistant strain of *Shigella sonnei*. The remaining twenty-one volunteers took a placebo, and 62 percent of them developed diarrhea during their first 4 weeks in Kenya. No salmonellae or vibrios were recovered from the placebo group, but ETEC was isolated from 57 percent (8 of 14) of individuals with diarrhea and from 57 percent (4 of 7) of those without. There were no ETEC infections among the doxycycline group.

Merson and others (1976) conducted a prospective study of 73 physicians and 48 family members (94 percent of whom were from the USA) attending the Fifth World Congress of Gastroenterology in Mexico City (Mexico) in October 1974. Of the 121 participants, 59 (49 percent) had travelers' diarrhea (51 percent of physicians and 45 percent of spouses). All pre-Mexico stool specimens were negative for bacterial pathogens. Subsequently, a pathogen was isolated from 63 percent of those with diarrhea and 21 percent of those without. ETEC was isolated from 45 percent of those with diarrhea and 7 percent of those without. In 81 percent of those with ETEC and diarrhea, ETEC was the only pathogen found. Other potential pathogens isolated from participants with diarrhea were: EIEC 4 percent, *Salmonella* 16 percent, *Shigella* 4 percent, *V. parahaemolyticus* 2 percent, *Giardia* 2 percent, and rotavirus 4 percent (there were several multiple infections). Occurrence of illness in one spouse did not increase the risk of illness in the other, and only one ill couple had the same serotype of ETEC. Illness was not associated with the consumption of water or iced beverages, but ETEC infection was associated with the consumption of salads containing raw vegetables. Nineteen percent of those with diarrhea were confined to bed. The authors point out that if these figures apply to the 3 million annual visitors from USA to Mexico (a conservative assumption, since many of the 3 million visitors are not physicians and do not stay at major hotels), then there may be 1.5 million cases of travelers' diarrhea per year among these visitors, of which 300,000 may be bedridden.

Despite dramatic figures of this kind, the problem of travelers' diarrhea is trivial compared with that of *E. coli* diarrhea among infants and children in developing countries, and deaths from travelers' diarrhea are extremely rare. Some of the recent comments (for instance, Lee and Kean 1978) concerning the

relationship between travelers' diarrhea and the economics of tourism—and the value to the general population of improving the hygiene of the hotel, restaurant, and food and beverage industries—are somewhat naïve and have led to excessive emphasis being given to travelers' diarrhea by some international development agencies.

Food is a likely route for the transmission of pathogenic *E. coli*. Barrell and Rowland (1979*a*) found that *E. coli* could be isolated from 35 percent of freshly prepared food samples in the Gambian wet season (June–October) and from 7 percent in the dry season. When food that had been stored for 8 hours or more was sampled, the percentages were 85 in the wet season and 59 in the dry season. No serotyping or toxin assays were reported. Sack and others (1977) examined 240 strains of *E. coli*, of non-EPEC serotypes, isolated from food samples collected throughout the USA and not known to be associated with diarrheal disease. Five percent of isolates produced LT alone, 6 percent produced ST alone, and 3 percent produced LT and ST—a total ETEC proportion of 14 percent (see also Mehlman and others 1976).

Polluted water is also a likely vehicle for *E. coli* transmission. Sack and others (1975*a*) examined eighteen water sources used by Apaches at Whiteriver (Arizona, USA). The water sources contained 200–300 coliforms per 100 milliliters; out of forty-seven *E. coli* isolates, three strains (6 percent) were toxin producing. Freij and others (1979) tested river water and wellwater around Addis Ababa (Ethiopia) and found ETEC in 55 percent and 14 percent of samples from the two respective sources, EPEC in 70 percent and 53 percent of samples, and EIEC in 10 percent and 20 percent of samples.

Rosenberg and others (1977) investigated an outbreak of diarrhea at Crater Lake National Park (Oregon, USA) in June 1975. Illness (defined as diarrhea or vomiting) was reported by 90 percent (288 of 320) of resident park staff, by 64 percent (68 of 107) of visitors contacted by phone, and by 44 percent (2,310 of 5,273) of visitors contacted by mail. Illness was significantly associated with drinking the park water supply, and the duration of illness increased as reported daily water consumption increased. The park water supply came from a shallow spring and was chlorinated before distribution. There was no systematic monitoring of chlorine in the supply, and dye tests showed the spring to be contaminated from an overflowing sewer above it. A single ETEC serotype (O6:H16, LT- and ST-positive) was isolated from 43 percent (17 of 40) of active diarrhea cases and from no (0 of 71) individuals who had not had diarrhea during the previous 4 days. No other viral, bacterial, or parasitic pathogen was recovered from ill or well persons. Of 396 *E. coli* isolates from the water supply, 14 (3.5 percent) were ETEC, and all of these were the same serotype as found in the diarrheal stools.

## Control Measures

Awareness that *E. coli* is a major cause of diarrhea is so recent, and understanding of *E. coli* epidemiology so partial, that few definitive statements about control can be made. Most of what can be said is based on the assumptions that, first, *E. coli* transmission is broadly similar to that of other anthroponotic, endemic bacterial agents of diarrhea (such as *Shigella*), and, second, that the information concerning *E. coli* in the environment reviewed below applies to ETEC, EPEC and EIEC as well as to nonpathogenic *E. coli*.

### *Individual*

Chemoprophylaxis with antibiotics or intestinal antiseptics is not to be encouraged. Antibiotic-resistance plasmids that may also code for virulence factors are present in the *E. coli* population in man and animals, and use of antibiotics encourages their spread. Merson and others (1976), studying travelers' diarrhea in Mexico, recorded that 33 percent of ETEC isolates showed multiple resistance to antibiotics. Kudoh and others (1979), studying travelers' diarrhea among Japanese, recorded that 38 percent of ETEC isolates had resistance to 1 or more antibiotics. Resistance to streptomycin and tetracycline was particularly common in both studies. However, there is some evidence that the prevalence of antibiotic resistance among ETEC strains is lower than among the *E. coli* population at large (Sack and others 1978).

Considerable success has been achieved in protecting suckling piglets and calves against fatal diarrhea by vaccinating their mothers with products containing K88, K99, and 987P adhesins. There is currently considerable research activity directed toward identifying the most appropriate preventive antigens for ETEC in man, and subsequently developing an oral vaccine. Prospects are reasonably good for an ETEC vaccine but are remote for a vaccine for EPEC and EIEC.

Scrupulous personal hygiene, and caution over choice and use of water and food, are probably the most effective protective methods for adults. Breast feeding appears to give some protection to infants.

### *Environmental*

Human feces, most probably from children and infected with EPEC, ETEC, or EIEC, are the major source

of infection. Control in the community therefore rests upon good water quality, adequate personal and domestic cleanliness (which will often require improved water availability), and hygienic excreta disposal. The hygiene of infants and young children—and those who handle, clean, and feed them—are of paramount importance.

## Fecal Indicator Bacteria

The previous sections of this chapter have dealt with that small subgroup of all *E. coli* which are able to cause diarrhea. The remainder of the chapter deals with the occurrence and survival of the fecal indicator bacteria in the environment. These fecal indicator bacteria include the coliforms, the fecal coliforms, *E. coli*, fecal streptococci, *Clostridium perfringens*, *Pseudomonas aeruginosa*, *Bifidobacterium*, *Bacteroides*, and other bacteria that are excreted in large numbers by healthy warm-blooded animals and that are not normally enteric pathogens. These indicator bacteria, and the concepts underlying their use as measures of the fecal contamination of the environment, are discussed in chapter 4 of Part One. In the remainder of this chapter, the most emphasis is given to coliforms, fecal coliforms, *E. coli*, and fecal streptococci because these are the excreted bacteria for which most environmental data exist and the ones which are most commonly used today as fecal indicators.

Very little information yet exists on the occurrence of pathogenic strains of *E. coli* in the environment, and little is known of their abilities to survive or multiply in extraintestinal settings. A few reports of ETEC isolations from water and food have been reviewed in the section above on epidemiology. For the time being, therefore, it must be assumed that the behavior of ETEC, EPEC, and EIEC in the environment is identical to that of the general *E. coli* population. Research currently under way, or being planned, will reveal the degree to which this assumption is valid.

## Occurrence and Survival in the Environment

Because the fecal coliforms and fecal streptococci are excreted by all warm-blooded animals, they are widespread in the environment wherever animal life is present. In addition, because the coliforms have been the main indicator of fecal pollution for over 60 years, there is more accumulated information on their presence and behavior in the environment than for any other excreted organism discussed in this book. Space only permits that a brief summary of the massive literature on coliforms in the environment, dating back to the end of the last century, can be given.

It must be stressed that the source of fecal coliforms and fecal streptococci in the environment is both humans and other animals. In an area populated mainly by humans (for instance, many urban areas), most enteric bacteria present may derive from man. However, in many rural areas the dominant source of indicator bacteria is domestic animals. An adult person may excrete only 150 grams of feces per day, but a pig may excrete 1.2 kilograms, and a cow may excrete 15–20 kilograms of feces. A hen may excrete almost the same weight of feces per day as a man. Feces from all these sources may contain comparable numbers of indicator bacteria per gram.

### *In surface waters*

Rain and snow generally contain no fecal bacteria. Geldreich (1978) collected rainwater in Cincinnati (Ohio, USA) and found no fecal coliforms or fecal streptococci in forty-six of forty-nine samples. Fallen precipitation, however, becomes stormwater or overland flow and may pick up a considerable load of fecal bacteria before it reaches a stream or soaks into the ground (Geldreich and others 1968). In urban areas, stormwater becomes contaminated by human and animal feces, and in some towns also by sewage, which may collect in drains and pools. Geldreich (1978) cited estimates that 70,000 kilograms of dog feces are deposited in the streets and parks of New York City (USA) every day. He also reported (1978) up to $10^5$ fecal coliforms and fecal streptococci per 100 milliliters of stormwater in suburban Cincinnati. Olivieri, Kawata and Krusė (1978) found that stormwater in Baltimore (Maryland, USA) contained indicator concentrations similar to weak raw sewage, with geometric mean values for fecal coliforms and fecal streptococci being generally over $10^5$ per 100 milliliters.

Stormwater run-off in rural areas may also collect large concentrations of indicator bacteria from animal feces and septic tank discharges (Dudley and Karr 1979). Typically, concentrations of fecal coliforms and fecal streptococci in stormwater are somewhat lower in rural catchments than in urban areas (Davis 1979), but this is not the case where manure, sewage effluent, or sludge is being applied to fields (Dunigan and Dick

1980; Evans and Owens 1972) or where the catchment supports intensive livestock farming (Geldreich 1976). For example, in the highlands of Papua New Guinea the large herds of domestic pigs (Feachem 1973), and the tendency of these pigs to pass the day foraging near streams, caused elevated fecal coliform and fecal streptococci concentrations in streams and rivers used for domestic purposes (table 13-2; Feachem 1974).

Major rivers flowing through populated areas always contain a considerable concentration of fecal indicator bacteria. Berg and Metcalf (1978) reported that the Seine (France) contained up to $5 \times 10^4$, the Missouri (USA) $4 \times 10^4$, and the Mississippi (USA) $3 \times 10^5$ fecal coliforms per 100 milliliters. Poynter and Stevens (1975) reported that water supply intake points on the Thames (UK) contained up to $3.5 \times 10^4$ *E. coli* per 100 milliliters. Evison and James (1973) found up to $10^5$ fecal coliforms and up to $10^4$ fecal streptococci per 100 milliliters of Nairobi River (Kenya) water, and Petr (1980) recorded over $10^4$ fecal coliforms per 100 milliliters in some sections of the Purari River (Papua New Guinea). Saleh (1980*a*, 1980*b*) found up to $10^3$ fecal streptococci per 100 milliliters in the Nile at Cairo (Egypt).

There is a growing tendency in Europe and North America to set indicator bacterial standards for body-contact recreational waters. A commonly adopted standard in the United States is less than 200 fecal coliforms per 100 milliliters (Cabelli 1978; Train 1979), and the European Economic Community has proposed a guideline of less than 100 fecal coliforms or fecal streptococci per 100 milliliters and a mandatory limit of 2,000 fecal coliforms per 100 milliliters. These standards for bathing water remain a controversial subject, and there is no clear consensus of opinion among regulatory authorities or individual experts (Evison 1979). Very few developing countries have standards of this kind, and most governments take the very reasonable view that, with the exception of schistosomiasis (see chapter 32), the risk of disease related to recreational bathing comes very low on the list of national health priorities.

Early studies on coliform die-off in water (see the appendixes to Feachem and others 1980) reported the time required for the bacterial population to become undetectable. These data are of limited value because a small proportion of organisms will be especially hardy and because the time over which residual numbers can be detected is very dependent upon the isolation technique being employed. Therefore, most recent work has concentrated on the rate of die-off that may be expressed as the time required for a given level of reduction; for instance, the time required for a 90 percent reduction ($t_{90}$) or the time required for a 50 percent reduction (half life, or $t_{50}$).[1]

Chamberlin and Mitchell (1978) and Mitchell and Chamberlin (1978) reviewed the literature on coliform decay rates in streams and found that $t_{90}$ values ranged from 20 to 115 hours, with a median value of about 60 hours. Chamberlin and Mitchell (1978) went on to analyze physical, chemical, and biological influences on coliform death rates and concluded that solar radiation is the dominant lethal factor (see also Chojnowski, Mancini and Jeris 1979).

Zanoni and others (1978) found a $t_{90}$ value for coliforms in Lake Michigan (USA) in July (water temperature 15°C) of only 6 hours. Dutka and Kwan (1980) reported $t_{90}$ values of 23–720 hours for *E. coli*, 19–360 hours for *Streptococcus faecalis*, and 19–180 hours for *Salmonella thompson* in Lake Ontario (Canada) at 17–19°C. Poynter and Stevens (1975) recorded $t_{90}$ values of 88 hours for *E. coli* and 96 hours for fecal streptococci in reservoir water stored at 15°C. McFeters and others (1974) found that the $t_{50}$ times for coliforms and fecal streptococci in well water at 9–12°C were around 20 hours, which was similar to times for shigellae and some salmonellae, but considerably

1. It has been often found experimentally, and it is usually assumed, that bacterial survival in water follows an exponential curve; that is, that the probability of a bacterium dying in a given time interval is independent of its age. In other words, the reduction in bacterial concentration in water follows a first-order equation of the form:

$$\frac{dC}{dt} = -kC,$$

where $C$ = concentration of bacteria per volume of water (say, organisms per 100 milliliters) at time $t$
$t$ = time (say, hours)
$k$ = first order decay or die-off rate constant (expressed as reciprocal of units of $t$; say per hour).

Integrating:

$$C = C_0 e^{-kt}$$

where $C_0$ = concentration of bacteria at $t = 0$.

Changing to base 10 and rearranging:

$$k = \frac{2.3}{t} \log_{10} \frac{C_0}{C}$$

If $C = 0.1C_0$, then:

$$k = \frac{2.3}{t_{90}}$$

In much of the literature, death rates are expressed as $k$, in hourly or daily units, rather than as $t_{90}$ values. The significance of $t_{90}$ is that it is the time for reduction to a fraction of 1/10 of the starting population, or a 1 log reduction. Since large changes in bacterial populations are best handled in logarithmic terms, it is of particular convenience. Times required for greater reductions may be readily calculated: $t_{99} = 2 \times t_{90}$, $t_{99.9} = 3 \times t_{90}$, and so forth.

Table 13-2. *Some reported concentrations of fecal bacteria in untreated domestic water sources in developing countries*

| *Country* | *Water source* | *Fecal coliforms per 100 milliliters* | *Fecal streptococci per 100 milliliters*[a] | *Source* |
|---|---|---|---|---|
| Gambia | Open hand-dug wells, 15–18 meters deep | Up to 100,000 | ND | Barrell and Rowland (1979*b*) |
| Indonesia | Canals in central Jakarta | 3,100–3,100,000 | ND | Gracey and others (1979) |
| Kenya | Springs | 0 | 0 | Evison and James (1973) |
| | Dam | 0–2 | 0–14 | |
| | Waterhole | 11–350 | 50–90 | |
| | Large river | 10–100,000 | 10–10,000 | |
| Lesotho | Unprotected springs | 900 | 1,700 | Feachem and others (1978) |
| | Waterholes | 860 | 1,610 | |
| | Small dams | 260 | 360 | |
| | Streams | 5,000 | 4,100 | |
| | Protected springs | 200 | 250 | |
| | Tap water from springs | 9 | 29 | |
| | Tap water from bored holes | 1 | 10 | |
| Nigeria | Ponds | 1,300–1,900 | 1,300–3,900 | Essien and Osuhor (1979) |
| | Open hand-dug wells | 200–580 | 180–630 | |
| | Tap water from bored hole | Up to 35 | Up to 6 | |
| | Ponds | 4,000,000[b] | ND | Tomkins and others (1978) |
| | Open hand-dug wells, 6–12 meters deep | 50,000[b] | ND | |
| | Stored in home | 100[b] | ND | |
| Papua New Guinea | Streams | 0–10,000 | 0–4,000 | Feachem (1974) |
| Tanzania | Rainwater | 3 | 13 | Brokunsult and Ross Institute (1978) |
| | Waterholes | 61 | 974 | |
| | Ponds | 163 | 590 | |
| | Streams | 128 | 293 | |
| | Unprotected springs | 20 | 58 | |
| | Protected springs | 15 | 40 | |
| | Open wells | 343 | 1,761 | |
| | Protected wells | 7 | 33 | |
| | Bored holes | 1 | 11 | |
| | Treated tap water | 3 | 13 | |
| Uganda | Rivers | 500–8,000 | ND | White, Bradley and White (1972) |
| | Streams | 2–1,000 | ND | |
| | Unprotected springs | 0–2,000 | ND | |
| | Protected springs | 0–200 | ND | |
| | Hand-dug wells | 8–200 | ND | |
| | Bored holes | 0–60 | ND | |

ND No data.

*Note:* These figures are not necessarily typical of the domestic water quality in the countries concerned. They are measurements taken from selected sources during specific investigations. It is generally true, however, that people in developing countries who must use surface sources or open wells are often drinking water with $>10^3$ fecal coliforms per 100 milliliters.

a. When only a single value is given, it is a geometric mean.

b. Total coliforms rather than fecal coliforms.

longer than those for *Vibrio cholerae* and other salmonellae.

Waters with little or no microbial life will sustain indicator bacteria for considerably longer than similar waters with an active flora and fauna. Geldreich (1976) reported that the $t_{90}$ for fecal coliforms in filtered stormwater was 40 hours at 20°C and 230 hours at 10°C. Gallagher and Spino (1968) found that the $t_{90}$ for fecal coliforms in filtered streamwater was 168 hours at 20°C.

As with all the microbial survival data discussed in this book, temperature is a crucial factor. Mancini (1978) used published data to compute a relationship between decay rate and temperature in fresh water. The results showed a $t_{90}$ of about 120 hours at 0°C, falling to about 15 hours at 30°C. Evison and James (1973) found that indicator bacteria in sewage effluent were reduced by 96.5 percent within 5 miles following discharge into the Nairobi River (Kenya; temperature 18.5°C), whereas only a 56 percent reduction took place over an equivalent distance in the River Tees (UK; temperature 2°C). Davenport, Sparrow and Gordon (1976) found fecal coliform and fecal streptococci reductions of only 84 and 67 percent, respectively, after 170 hours of travel under the ice of the frozen Tanana River (Alaska, USA), whereas a similar travel time in a tropical river might cause a reduction of over six log units assuming that there were no additional inputs of fecal pollution.

Many investigators have found that total coliform and fecal coliform decay rates are similar, whereas fecal streptococci often persist for longer (for instance, Cohen and Shuval 1973; Poynter and Stevens 1975).[2] *Str. bovis*, and to a lesser extent *Str. equinus*, however, die-off considerably faster than fecal coliforms and other species of fecal streptococci (Geldreich 1976; Geldreich and Kenner 1969; Guy and Small 1977; McFeters and others 1974). Therefore, because *Str. bovis* and *Str. equinus* are the dominant streptococcal species in some animal feces but never in human feces, it is sometimes the case that in stored samples containing mainly human fecal pollution the fecal coliform to fecal streptococci ratio falls over time, whereas when nonhuman pollution predominates the ratio may rise (Feachem 1975; see chapter 4).

As a general rule indicator bacteria die in fresh water, and the warmer the temperature the higher the death rate. Death rates are also higher in natural waters with an active biological population, than in sterilized, filtered, or other "dead" waters (Poynter and Stevens 1975). Under certain special conditions, however, growth of indicator bacteria may occur. The growth phase is usually of limited duration and is especially likely where nutrient levels are high, temperatures are warm, but overall microbial and zoological activity is low. Thus chlorinated effluents sometimes provide suitable growth environments for indicator bacteria (see the section below on effluent chlorination). Hendricks (1972) found that *E. coli* would grow at 30°C in autoclaved river water collected downstream of a sewage outfall, but not at 20°C or 5°C and not in autoclaved water collected upstream of the outfall (see also Gorden and Fliermans 1978; Hendricks 1971; McFeters, Stuart and Olson 1978).

Coliform growth is more likely than fecal coliform growth, which is more likely than fecal streptococcal growth (see, for instance, Allen, Pasley and Pierce 1952). The growth of indicator bacteria is more likely than the growth of pathogenic bacteria. This latter fact seriously reduces the value of the indicator bacteria as indicators of pathogenic microbes in situations where growth is possible or suspected; also, the human excreted viruses (chapters 9–11) can *never* increase in numbers in the aquatic environment.

### *In groundwater*

The microbiological quality of groundwater is becoming an increasing cause for concern worldwide as greater use is made of limited groundwater resources and as the practice of disposing of fecal wastes in on-site sanitation systems or by land application becomes more common. The potential for groundwater contamination depends upon a complex of factors including the rainfall, the rate of groundwater abstraction, groundwater depth and flow patterns, the method of waste disposal, and the type, texture, and depth of the overlying soil or rock. Marzouk, Goyal and Gerba (1980) reported on the quality of ninety-nine groundwater samples in Israel. Measures of fecal coliforms per 100 milliliters were range $0–2 \times 10^4$, mean $10^3$, and median 0. Measures of fecal streptococci per 100 milliliters were range $0–10^4$, mean 300, and median 0. Tjostem and others (1977) found low levels of coliform contamination in groundwater pumped from a limestone aquifer in northeastern Iowa (USA). Below the limestone, and separated from it by a shale band, was a sandstone aquifer. Water pumped from the sandstone in uncased wells was also contaminated, but

2. A recent report, however, suggested that *Str. faecalis* survived for a shorter period in Canadian lake water than *E. coli* (Dutka and Kwan 1980). There is probably considerable inter- and intra-species variation in survival ability, and studies on mixed populations of fecal streptococci and *E. coli* cannot be compared directly with studies on the survival of single laboratory-maintained strains.

wells that were fully cased and grouted through the limestone into the sandstone produced relatively unpolluted water. This illustrates an important general principle—that unpolluted groundwater can often be obtained from below polluted shallow aquifers if carefully designed abstraction technologies are employed.

Over the past 60 years, studies have been conducted to determine the risks of shallow groundwater pollution from pit latrines and septic tank soakage systems. Stiles and Crohurst (1923) recorded the movement of *E. coli* for 20 meters horizontally through fine sand in the direction of groundwater flow. In an early study in Singapore (Yeager 1929) to investigate the required separation of bored hole latrines from shallow unprotected wells, it was found that coliforms traveled for more than 23 meters but less than 31 meters through very permeable soils. Similar studies in West Bengal (India) showed that wells located 1.6 meters from a bored hole latrine (dug in alluvial sandy loam with a percolation rate of about 56 meters per day) became heavily contaminated with fecal coliforms, whereas most wells located 3.3 meters distant remained uncontaminated (Dyer, Bhaskaran and Sekar 1945). Further information on the ability of excreta disposal systems, especially septic tank drainfields, to pollute groundwater is reviewed below in the sections on septic tanks and land treatment.

Groundwater pollution by fecal coliforms and fecal streptococci may also be due to the deliberate recharge of sewage effluents to groundwater. Vaughn and others (1978) studied three sewage recharge installations on Long Island (New York, USA). At site 1, the chlorinated effluent contained up to $2.4 \times 10^6$ fecal coliforms per 100 milliliters, and the groundwater (9 meters below the recharge basins) contained up to 150 fecal coliforms per 100 milliliters. At site 2, the chlorinated effluent contained up to $4.3 \times 10^5$ fecal coliforms per 100 milliliters, and the groundwater (24 meters below the recharge basins) contained up to 930 fecal coliforms per 100 milliliters. At site 3, the tertiary effluent contained up to $9.3 \times 10^5$ fecal coliforms per 100 milliliters, and the groundwater (5.5 meters below the recharge basins) contained up to 150 fecal coliforms per 100 milliliters at a point 46 meters horizontally downslope from the recharge site. Slade and Edworthy (1981) isolated up to $1.2 \times 10^5$ *E. coli* and fecal streptococci per 100 milliliters of groundwater from a chalk aquifer directly below groundwater recharge lagoons receiving raw comminuted sewage. Bacteriological aspects of groundwater recharge in Israel are summarized by Goldshmid (1974). Groundwater contamination may also result from seepage through the base of waste stabilization ponds (see, for instance, Ciravolo and others 1979).

There are few data available on the survival of indicator bacteria in groundwater. It may be anticipated that survival will be for longer than in must surface waters because of the absence of sunlight, cool temperatures, and a low level of microbial and biological activity. Kudryavtseva (1972) reported that coliforms introduced into saturated alluvial sands in the USSR during the summer survived for up to 3.5 months. A pathogenic serotype of *E. coli* similarly inoculated into the groundwater survived for 3 months. In groundwater samples returned to the laboratory and stored in darkness, coliforms survived for up to 5.5 months, and pathogenic *E. coli* survived for up to 4 months.

These and other data (Allen 1979) show that whether or not on-site soakage or land application cause bacterial pollution of the groundwater depends on numerous site-specific factors. Where soils are of fine or medium texture, unsaturated, and more than 1 meter deep, little or no bacterial contamination of the underlying aquifer may occur. Where wastewater can drain down through "macropores" (such as root channels, structural voids, rodent burrows, solution channels or fissures) the groundwater may become significantly polluted with fecal coliforms and fecal streptococci. Even in this latter case, however, the enteric bacterial concentrations in groundwater are likely to be far less than in surface waters in the same location and will be readily eliminated by any water treatment process including chlorination. Even where untreated waters are being used for domestic purposes, contaminated groundwater will usually pose a lesser health risk than available surface water.

### *In drinking water*

Treated and chlorinated drinking water should contain no fecal indicator bacteria. Most people in developing countries, however, drink unchlorinated and untreated water. In cases where this water derives from protected groundwater, or upland surface water sources, it may be of moderately good quality (say $< 100$ fecal coliforms per 100 milliliters). In other cases the water used may be highly polluted and, on occasions, has an indicator bacteria concentration similar to that of a weak raw sewage. In table 13-2, some information on the pollution of drinking water sources in developing countries is summarized.

This problem of fecally polluted drinking water is by no means restricted to the developing countries. Sandhu, Warren and Nelson (1979) reported that

around 60 percent of people in three counties in South Carolina (USA) were served by wells, springs, and other private water sources. In one county, private water sources had a mean *E. coli* count of $1.4 \times 10^6$ per 100 milliliters and a mean fecal streptococci count of $2.6 \times 10^6$ per 100 milliliters. It was concluded that defective septic tank systems were responsible for most of the fecal contamination.

The question of bacteriological standards for drinking water remains a subject of considerable debate. In countries where all, or nearly all, the population drink treated piped water it is reasonable and correct to stipulate that no coliforms or other indicator bacteria should be detected in tap water. Failure to meet this standard indicates a malfunction of the treatment plant (especially of the chlorination unit) or an inflow of pollution through a damaged section of the distribution system, which should be immediately investigated and rectified. In developing countries, however, the great majority of the population drink water that is untreated, either from improved but untreated supplies (such as handpumps) or from unimproved supplies (such as ponds). This water, as indicated in table 13-2, may be grossly polluted, and it is pointless for the government of such a country to require that all water supplies contain no fecal coliforms. At the best such a ruling will simply be ignored and thus bring similar regulations into disrepute; at the worst it may force people to abandon improved but lightly contaminated supplies in favor of the only alternative, which may be unimproved and heavily polluted supplies. For example, there have been cases where overzealous health officials have closed down contaminated shallow tubewells in a village because the wells were found to contain 50 fecal coliforms per 100 milliliters and have thus forced the villagers to use polluted irrigation canals containing $10^4$ fecal coliforms per 100 milliliters.

The World Health Organization (WHO) has generally advocated standards or guidelines for small untreated water supplies that stipulate less than 10 coliforms and zero fecal coliforms per 100 milliliters (WHO 1971). These recommendations have been questioned by those primarily concerned with water supplies in developing countries on the grounds that they are too stringent (for instance, see Feachem 1977). Even if great attention is paid to selecting the purest available water source and distributing the water through a well-designed and well-maintained system, it will not in general be possible to meet a zero fecal coliform standard without incorporating chlorination. Well-designed untreated spring supplies, for instance, will typically contain up to about 25 fecal coliforms per 100 milliliters, and this level of contamination can only be removed by adding a chlorination unit. Therefore, to set a zero fecal coliform standard is equivalent to requiring that all water supplies be chlorinated (at least). Many developing countries, however, have decided to adopt a flexible policy toward water treatment and are installing numerous spring, well, or upland stream supplies that have no treatment processes. The advantage of designing a supply without treatment is that it is somewhat cheaper, and much easier to operate and maintain, than a similar supply with treatment (for instance, slow sand filtration and chlorination). For many developing countries, therefore, a zero fecal coliform standard is inappropriate. A preferable approach is to set flexible quality goals that can be changed as the water supply sector progresses. To install an improved supply providing water with up to 50 fecal coliforms per 100 milliliters, for instance, is a great advance when many people in the same country may be drinking water containing over $10^3$ fecal coliforms per 100 milliliters.

The effect collecting, carrying, and storing water have on bacteriological water quality has attracted increasing concern in recent years. Clearly, there is less purpose in supplying good quality water at a public tap if it is to become subsequently polluted prior to use. If water is collected in clean vessels and stored in such a way that polluting material cannot enter, water quality is likely to improve—as suggested by the data from Malumfashi (Nigeria) presented by Tomkins and others (1978) and summarized in table 13-2. It may be more usual, however, for water quality to deteriorate between collection and use because the water collection vessels are contaminated and the water is stored in the home in such a way that it can be further contaminated by children and animals. Studies in Lesotho (Feachem and others 1978) showed that clean water collected from a handpump (0–6 fecal coliforms and 0–1 fecal streptococci per 100 milliliters) could become considerably contaminated before use (maximum of 1,340 fecal coliforms and 4,280 fecal streptococci per 100 milliliters). Similarly, Oluwande (1980) reported that public tap water in Western State (Nigeria) contained 0–3 coliforms and 0 fecal coliforms per 100 milliliters, whereas stored water in homes contained 0–1,800 coliforms and 0–10 fecal coliforms per 100 milliliters. The epidemiological significance of water pollution occurring after collection is different from that of pollution of the water source. The first type of pollution promotes intrafamilial disease transmission, whereas the second allows the spread of infection throughout a community using a common source.

In concluding this section on indicator bacteria in drinking water, a note of caution must be sounded about the validity of the standard tests for fecal coliforms, which were developed in Europe and North America, when they are applied to tropical waters. Some workers studying water pollution in upland tropical areas, where surface water temperatures are not greatly higher than in temperature zones, have obtained satisfactory results using standard methods for enumerating fecal coliforms [for instance, Feachem (1974) in the highlands of Papua New Guinea and White, Bradley and White (1972) in Uganda]. Other studies in the tropics [for instance, Banerjea and Sen (1940) and Raghavachari and Iyer (1940) in India; Boizot (1941) in Singapore; Evison and James (1973) in Kenya; Katugampola and Assim (1958) in Sri Lanka; and Moussa (1965) in Egypt] have detected a considerable proportion of coliforms of probable nonfecal origin that have the ability to ferment lactose at 44°C. In this respect they mimic the truly fecal coliforms and are thus able to give false positive reactions on standard fecal coliform tests. Recent water testing in the Gambia (Barrell and Rowland 1979*b*) and Tanzania (Brokunsult and Ross Institute 1978) has shown a high prevalence (up to 55 percent) of false positive results presumably caused by nonfecal coliforms that reside in warm tropical waters and have the ability to ferment lactose at 44°C. There is an urgent need for the development of a test for fecal indicator bacteria in tropical waters that will reliably and simply distinguish between organisms of enteric origin and others that are free-living and adapted to warm, aqueous habitats. [See note on page 66.]

*In seawater*

The great majority of coastal towns and cities that have a sewerage system discharge their sewage into the sea following little or no treatment. This is true throughout the world. The design of these marine outfalls has attracted considerable interest over the past two decades and involves complex decisions about the degree of treatment and the design of the outfall and complex tradeoffs between costs and environmental hazards. The principal health-related fecal hazards are the risks to swimmers and the contamination of fish and shellfish.

To design outfalls in such a way that fecal bacteria and viruses do not pollute beaches or seafood requires a detailed knowledge of the dispersion, sedimentation, and death of fecal microorganisms discharged into coastal waters. The information on viruses is briefly reviewed in chapter 9, and the available knowledge of fecal bacteria, especially coliforms, is considerably more extensive.

Numerous studies have documented high levels of indicator bacteria (up to $10^3$–$10^5$ per 100 milliliters) in ocean or estuarine waters near sewage outfalls. Recent examples from the USA include studies at Miami Beach (Florida; Edmond, Schaiberger and Gerba 1978), Honolulu (Hawaii; Loh, Fujioka and Lau 1979), the Texas Gulf Coast (Gerba and others 1977; Goyal, Gerba and Melnick 1977, 1978, 1979), Long Island, (New York; Vaughn and others 1979), and the New York Bight (Berg and Metcalf 1978). Studies from other countries include those at Tel Aviv (Israel; Shuval 1978), Alexandria (Egypt; Hakim 1978), Kerala (India; Raveendran, Gore and Unnithan 1978), Naples (Italy; Evison and Tosti 1980), Tuscany (Italy; Petrilli and others 1979), Whitely Bay (UK; Evison and Tosti, 1980), Liverpool (UK; Karthegisan and Pugh Thomas 1980), Belgium (Yde and de Maeyer-Cleempoel 1980), and New South Wales and Tasmania (Australia; Roper and Marsall 1979). In several of these studies (for instance, those in Texas) fecal indicator bacteria were isolated at higher concentrations (10–1,000 times higher) from bottom sediments than from the overlying waters. Roper and Marshall (1979) showed that *E. coli* in saline sediments were protected against attack by viruses, bacteria, and amoebae, and growth of coliforms in marine sediments has been demonstrated (Gerba and McLeod 1976).

Numerous estimates of coliform death rates have been made. Chamberlin and Mitchell (1978) and Mitchell and Chamberlin (1978) reviewed eighty-seven of these estimates and concluded that the times of 90 percent reduction ($t_{90}$) lay between 0.6 and 8 hours, with a geometric mean of about 2 hours (corresponding $k$ values are 0.3–4 per hour with a mean of 1.15 per hour—see footnote 1, above). These values reveal considerably faster death of coliforms in seawater than in fresh water (where $t_{90}$ values are between 20 and 115 hours, with a median of about 60 hours). Death rates of coliforms in seawater are also considerably faster than the death rates of viruses in seawater ($t_{90}$ values in the range of 15–70 hours—see chapter 9). There is now widespread agreement that, owing to the greater persistence of enteric viruses, fecal coliforms are an inadequate index of saline water quality, especially in shellfish-growing areas (Berg and Metcalf 1978). Fecal streptococci survive longer in marine environments than fecal coliforms (Baross, Hanus and Morita 1975; Hanes and Fragala 1967; Petrilli and others 1979; Vasconcelos and Swartz 1976), but not sufficiently long for them to act as an adequate indicator of the enteroviruses. Pichot and Barbette (1978) found a $t_{90}$

of 3.7 hours for fecal coliforms and 5.7 hours for fecal streptococci under the same experimental conditions.

The reasons for the rapid death of coliforms in seawater have been the subject of many investigations (Mitchell 1968). Faust, Aotaky and Hargadon (1975) found temperature, dissolved oxygen, and salinity to be the major determinants of the rate of death, and Enzinger and Cooper (1976), McCambridge and McMeekin (1979) and Mitchell and Yankofsky (1969) drew attention to the important role of protozoan predators. Gerasimenko (1977) found that oil pollution did not affect coliform death rates.

An increasingly convincing case has been built for the importance of light-induced cell damage in determining coliform death rates in sea water (Chamberlin and Mitchell 1978; Chojnowski, Mancini and Jeris 1979; Gameson and Gould 1975; Gameson and Saxon 1967; Mitchell and Chamberlin 1975, 1978). Experiments on fecal coliforms in Sydney harbor (Australia) showed a minimum daytime $t_{90}$ of 1.9 hours and a night time $t_{90}$ of 40 hours (Bellair, Parr-Smith and Wallis 1977). Fecal streptococci appear to be substantially less sensitive to light than coliforms (Chamberlin and Mitchell 1978).

Little information is available on the survival of indicator bacteria in tropical seawater. In extrapolating results from temperate areas, temperature is the variable of most importance. Even relatively small temperature differences can substantially affect the death rate. Jamieson, Madri and Claus (1976) reported that in sterilized saline waters a pathogenic serotype of *E. coli* had a $t_{90}$ of about 40 hours at 4°C and about 8 hours at 37°C. Vasconcelos and Swartz (1976) reported that *E. coli* concentrations in seawater declined by less than 2 log units at 8.9°C, but by 7 log units at 14.5°C, after 6 days. Burdyl and Post (1979) studied *E. coli* survival in the Great Salt Lake (USA) and estimated a $t_{90}$ of about 110 hours at 9°C and about 21 hours at 19°C. Faust, Aotaky and Hargadon (1975) reported $t_{50}$ values for *E. coli* in estuarine water of 39 hours at 0°C and 14 hours at 30°C. Mancini (1978) reviewed reported death rates and temperatures and computed typical $t_{90}$ values of 60 hours at 0°C and 7 hours at 30°C. Clearly, coliforms discharged into tropical seawater will rapidly decline in numbers, as will other excreted bacteria, although not necessarily at the same rate. Excreted virus concentrations will decline considerably more slowly (see chapter 9).

An active debate continues about the magnitude of the health risk associated with swimming in fecally polluted seawater and the correct approach to water quality standards and legislation (Cabelli 1979; Evison and Tosti 1980; Moore, Perin and Maiden 1979). Recent evidence from Egypt and the USA (Cabelli 1979; Cabelli and others 1979) revealed a small but measurable difference in the incidence of gastrointestinal illness between swimmers and nonswimmers at polluted beaches. The recorded risks of swimming in seawater containing $10^2$–$10^3$ fecal coliforms per 100 milliliters were an additional attack rate of 1–2 cases of gastrointestinal illness per 100 people in the 8–10 days following the visit to the beach. It must be kept in mind, however, that especially in developing countries the infections that may be transmitted to swimmers at polluted beaches will usually be highly endemic in the community at large (the community producing the wastes which are polluting the sea), and swimming may constitute a negligible additional risk. Set against this is the possibility that swimmers from high socioeconomic strata (who experience a low risk at home due to adequate water supply, sanitation, and hygiene) may be exposed to a substantially increased risk of infection when they bathe in seawater polluted by the wastes of all socioeconomic strata. The same level of additional risk may apply to tourists—who are usually either local residents from upper socioeconomic groups or foreign visitors.

### *In feces and night soil*

The fecal indicator bacteria are excreted by almost all people, and by almost all warm-blooded animals, nearly all of the time. They are therefore ubiquitous and numerous in all materials containing fresh human or animal feces. The fecal indicator bacteria are a part of the vast total intestinal microflora. A healthy individual may commonly excrete $10^{11}$–$10^{12}$ bacterial cells per wet gram of feces, and these cells may constitute about 9 percent of the total fecal wet weight or 25 percent of the fecal dry weight (Geldreich 1978). The composition of this total bacterial population is set out in table 1-6. It is usual for the anaerobes, especially *Bifidobacterium* and *Bacteroides*, to be a more numerous and more stable component of the fecal flora than the fecal coliforms or fecal streptococci (see, for instance, Mata, Carrillo and Villatoro 1969; Tomkins and others 1981; Zubrzycki and Spaulding 1962).

The numbers and the serotypes or species of fecal coliforms and fecal streptococci excreted by humans vary considerably between individuals according to age (Gorbach and others 1967), diet (Bettleheim and others 1977), state of health, and chemical and microbiological properties of the intestine. Wheater, Mara and Oragui (1979) studied twelve adults in Dundee (Scotland) and found between $8 \times 10^4$ and

$8 \times 10^7$ fecal coliforms, and between $3 \times 10^2$ and $2 \times 10^7$ fecal streptococci, per gram of wet feces. However, community averages are less variable and are in the ranges of $10^6$–$10^9$ fecal coliforms per gram and $10^5$–$10^8$ fecal streptococci per gram (table 1-6). Fecal coliform concentrations are usually higher than those for fecal streptococci by a factor of 4 or more, at least in the developed countries whence most such data come.

The numbers and serotypes of fecal coliforms excreted by a single individual vary through time due to the influence of many factors. In particular, the proliferation of a pathogenic bacterium may modify the commensal flora in the intestine. Dale and Mata (1968) found that eight children in Guatemala with shigellosis excreted between $10^4$ and $10^8$ coliforms per gram and that coliform excretion had a roughly inverse relation to *Shigella* excretion. Streptococci were excreted by the same children in concentrations of $10^8$–$10^9$ per gram.

Most nonhuman animals excrete $10^5$ or more fecal coliforms and fecal streptococci per gram of feces. However, some animals excrete $<10^5$ per gram, as reported for horses and rabbits, respectively, by Geldreich (1978) in the USA and by Wheater, Mara and Oragui (1979) in Scotland. It has often been claimed in the literature from the USA (for instance, Geldreich 1976) that fecal streptococci concentrations generally exceed fecal coliform concentrations in animal feces and that the reverse is true for human feces. However, Wheater, Mara and Oragui (1979) showed that this was not the case for sheep, pigs, cats, dogs, hens, ducks, pigeons, and seagulls in Scotland (see also Williams Smith and Crabb 1961). This variability in fecal coliform to fecal streptococci ratios is one of the reasons for the current rejection of the ratio as a method for distinguishing between human and nonhuman fecal pollution (see chapter 4).

Jordan (1926) studied the survival of *E. coli* in stored feces. At room temperature, numbers increased to $10^8$–$10^{11}$ per gram after 2–5 days and subsequently decreased to undetectable levels in 6–12 weeks. At 37°C, numbers initially increased markedly but then declined to zero within 1–3 weeks. At 10°C, a slower increase in numbers occurred, to a maximum of $10^8$–$10^{10}$ per gram after 20 days, and $10^4$ per gram could still be detected after 23 weeks.

### *In sewage*

Because the fecal indicator bacteria are ubiquitous and numerous in feces, they are also ubiquitous and numerous in raw sewage and in most treated sewage effluents. Concentrations of indicator bacteria in sewage vary through the day, but this variability is considerably less in the sewage derived from large communities than in that derived from small communities. Communities with a high water use per capita produce a sewage with a lower concentration of indicator bacteria than communities with lower water usage. Thus, indicator bacteria concentrations in sewage in developing countries are generally higher than those reported from industrialized countries.

Raw sewage typically contains between $10^5$ and $10^8$ fecal coliforms and fecal streptococci per 100 milliliters. Berg and Metcalf (1978) reported between $3.8 \times 10^4$ and $4.6 \times 10^6$ fecal coliforms per 100 milliliters of sewage in the USA. Geldreich (1978) reported that twenty-one towns in the USA had between $3.4 \times 10^5$ and $4.9 \times 10^7$ fecal coliforms and that seven towns had between $6.4 \times 10^4$ and $4.5 \times 10^6$ fecal streptococci per 100 milliliters of sewage. Davis (1979) found that raw sewage in Houston (Texas, USA) contained $3 \times 10^6$ to $3 \times 10^7$ fecal coliforms and $5 \times 10^5$ to $2 \times 10^6$ fecal streptococci per 100 milliliters. In the Dundee area (Scotland), raw sewage contained $5.8 \times 10^6$ to $1.5 \times 10^7$ *E. coli* per 100 milliliters (Wheater and others 1980).

Evison and James (1973) reported that raw sewage in Nairobi (Kenya) contained up to $1.6 \times 10^8$ *E. coli*, and up to $3.5 \times 10^7$ fecal streptococci, per 100 milliliters. In contrast, and presumably because of higher levels of water use, raw sewage in Pietermaritzburg (South Africa) contained only $1.5 \times 10^4$ *E. coli* per 100 milliliters (Grabow and Nupen 1972). In Brazil, Mara and Silva (1979) reported a mean of $5 \times 10^7$ fecal coliforms and $7 \times 10^6$ fecal streptococci per 100 milliliters of raw sewage.

Concentrations of indicator bacteria in sewage may be affected by the presence of industrial wastes that often contain chemicals antagonistic to enteric bacteria. Data on raw sewages from different areas of Birmingham (UK) showed *E. coli* concentrations of $1.7$–$3.7 \times 10^8$ per 100 milliliters where sewage was principally of domestic origin, compared with only $9 \times 10^5$ per 100 millimeters where sewage flow was 60 percent of industrial origin and contained 20–30 milligrams per liter of phenols (Pike and Carrington 1979).

Studies of sewage produced by small rural communities in the hills of Yorkshire (UK) have shown the fecal coliform concentrations to vary between $10^5$ and $10^8$ per 100 milliliters, whereas fecal streptococci vary between $10^4$ and $10^7$ per 100 milliliters (Feachem, unpublished data). Sanitation technologies that use little water, for instance the pour-flush designs, will

produce a sewage with an exceptionally high concentration of indicator bacteria. Daniel and Lloyd (1980) reported a geometric mean from twelve samples of $8.4 \times 10^8$ coliforms per 100 milliliters of sewage flowing into an Oxfam sanitation unit installed in a refugee camp near Dacca (Bangladesh).

Some studies on the survival of coliforms and streptococci in sewage are listed in the appendixes of Feachem and others (1980). Survival is greatly prolonged at cool temperatures, when dissolved oxygen is low (Hanes, Sarles and Rohlich 1964), or when the overall microflora have been reduced by chlorination or some other means. In warm climates, with sewage temperatures around 25–30°C, a >99 percent reduction in indicator bacteria concentrations may be expected in about 10–15 days, depending on the level of oxygenation of the sewage. It is generally reported that fecal streptococci survive for a little longer than fecal coliforms in sewage (see, for instance, Berg and Metcalf 1978; Cohen and Shuval 1973).

One study recorded a far more rapid rate of death for fecal indicator bacteria in sewage under natural conditions (Dor, Schechter and Shuval 1976). The undiluted raw sewage of Jerusalem (Israel) entered the Nahal Soreq wadi at a rate of about 21,000 cubic meters per day. After a flow of 45 kilometers (which took about 44 hours), at a temperature of around 20°C (March), the fecal coliform and fecal streptococci concentrations were reduced by 99.9 and 99 percent, respectively. The combination of warm temperatures, highly turbulent flow in some sections of the wadi, and a rich algal community contributing photosynthetic oxygen produced a warm and well-aerated environment that caused rapid death of excreted bacteria.

### *In sludge*

Fecal indicator bacteria are always present in high concentrations in fresh sewage works sludge. Concentrations of $10^6$–$10^8$ total coliforms, $10^5$–$10^7$ fecal coliforms, and $10^4$–$10^6$ fecal streptococci per gram are normal. Dudley and others (1980) investigated an anaerobically digested sludge and two primary sludges at San Antonio (Texas, USA) and found $5 \times 10^5$–$5 \times 10^6$ fecal coliforms and $7 \times 10^4$–$5 \times 10^5$ fecal streptococci per gram of suspended solids.

As with coliforms in feces, night soil, and soil, coliforms in sludge may survive for several months in cool, moist conditions. Growth may also occur, and this will be more rapid at warmer temperatures. Edmonds (1976) reported that fecal coliforms in sludge applied to forest soil in Washington State (USA) survived for longer in summer than in winter. At warmer temperatures (say >25°C), however, it is likely that a vigorous growth period would be followed by a rapid decline. Overall survival times of indicator bacteria in sludge in the tropics will typically be shorter than in temperate climates.

### *In soil*

Fecal indicator bacteria occur in only very low concentrations (typically less than 2 per gram) in most uncontaminated soils (Geldreich and others 1962). In contrast, they are found in high concentrations in soil wherever effluent, night soil, sludge, or manure are being used for irrigation or fertilization or where livestock are grazing. The literature on the survival of enteric bacteria in soil is extensive and dates back to the early 1920s (see the appendixes of Feachem and others 1980). This literature has been periodically reviewed (see, for instance, Elliott and Ellis 1977; Gerba, Wallis and Melnick 1975*a*; Rudolfs, Falk and Ragotzkie 1950).

Reported survival times vary widely. The work of Van Donsel, Geldreich and Clarke (1967) clearly demonstrated the importance of sunlight and temperature in determining the death rate of fecal bacteria in soil. Times for 90 percent reduction ($t_{90}$) varied from a minimum of about 3 days in summer in exposed sites to maxima of about 14 days for *E. coli* and 20 days for *Str. faecalis* in autumn and winter at shaded sites. In spring and winter *Str. faecalis* survived for approximately twice as long as *E. coli*, whereas in summer and autumn the two survival times were similar. This may have been because some *E. coli* growth was occurring in summer and autumn.

In Alberta (Canada) studies were conducted on the weekly application of 45 millimeters of unchlorinated waste stabilization pond effluent to plots of canary grass (Bell and Bole 1978). The effluent contained between $2.3 \times 10^4$ and $1.7 \times 10^5$ fecal coliforms per 100 milliliters, and most coliforms were retained in the upper 80 millimeters of the loamy sand. Fecal coliform dieoff occurred in two phases. Over the first 2 days after effluent application a 90 percent reduction occurred. Subsequently, the reduction was about 33 percent per day at 15°C and about 25 percent per day at 10°C. In spring and summer, little or no fecal coliform contamination could be detected 2 weeks after the cessation of irrigation.

Chandler and Craven (1978*a*) studied the disposal to land of piggery waste containing $10^5$–$10^8$ *E. coli* per 100 milliliters in Victoria (Australia). *E. coli* concentrations in the soil declined by 99 to 99.99 percent

after 3–6 weeks, and rapid downward movement of *E. coli* was recorded in late summer when the soil was dry and cracked. In other experiments, Chandler and Craven (1978*b*) recorded a 99 percent reduction of *E. coli* in 1 day in dry soil, whereas in saturated soil the reduction was less than 90 percent after 3 weeks. Similarly, Chandler and Craven (1980) recorded $t_{90}$ values for *E. coli* at 20°C of 18 days in soil with 30 percent moisture and 2.5 days in soil with 10 percent moisture.

Kibbey, Hagedorn and McCoy (1978) found that the survival of *Str. faecalis* in various loams was mostly dependent upon temperature and moisture levels. The time for 95 percent reduction ($t_{95}$) in saturated soils was 94 days at 4°C, 80 days at 10°C, 53 days at 25°C, and 29 days at 37°C. In air-dried soil the $t_{95}$ values were 23 days at 4°C, 18 days at 10°C, 9 days at 25°C, and 5 days at 37°C.

Under certain soil conditions coliform and fecal coliform concentrations will increase, and this increase will be more rapid at warmer temperatures (see, for instance, Guy and Small 1977). Survival times and the potential for growth are influenced by the availability of nutrients in the soil. Thus, survival is prolonged in soil that is regularly receiving effluent or night soil applications. Dazzo, Smith and Hubbell (1973) recorded $t_{90}$ values for *E. coli* of 4 days in soil receiving no manure and 8.5 days in soil receiving 50 millimeters of cow manure slurry per week.

Survival of indicator bacteria in soil is influenced by moisture content, temperature, shade, soil organic content and the overall biological activity present in the soil. These conditions are so variable that reported survival times and $t_{90}$ values cover a wide range. From an overview of the literature (see the appendixes of Feachem and others 1980), it appears that fecal coliforms generally survive for less than 10 weeks, with a 90 percent reduction taking place within 10 days. Under cool, moist conditions a hardy residual fraction of fecal coliforms may survive for many months. Where conditions are hot and arid very limited survival can be expected, and it is probable that almost complete elimination of fecal indicator bacteria will occur within 2 weeks.

### *On crops*

Crops irrigated or fertilized with effluent, night soil, sludge, or manure may be heavily contaminated by fecal indicator bacteria, whereas untreated plants and crops are not (Geldreich, Kenner and Kabler 1964).

Studies in Victoria (Australia) demonstrated the very limited survival times of excreted bacteria on plants compared with their survival on the underlying soil (Chandler and Craven 1978*a*, 1978*b*, 1980). Under conditions in which *E. coli* survived on soil for 8 weeks, no *E. coli* were recovered from grass after 16 days. Persistence of *E. coli* on the grass stems was greatest near the soil surface.

Experiments in Alberta (Canada) on the spray irrigation of fodder crops with waste stabilization pond effluent (containing $10^2$–$10^6$ fecal coliforms per 100 milliliters) have demonstrated the importance of climatic conditions and the anatomy of the plant in determining the survival of excreted bacteria (Bell 1976; Bell and Bole 1976). Fecal coliforms on alfafa (*Medicago sativa*) declined by over 99 percent in 1 day when temperatures were warm (12–23°C), relative humidity was low (20–65 percent) and there were about 9 hours per day of bright sunshine. When temperatures were cooler (9–18°C), relative humidities higher (48–95 percent), and the sky was overcast, a 99 percent reduction required 4 days. In contrast, fecal coliforms on reed canary grass (*Phalaris arundinacea*), bromegrass (*Bromus inermus*) and orchard grass (*Dactylis glomerata*)—grasses that, unlike alfafa, possess leaf sheaths—only underwent a 99 percent reduction in 5 days even in bright weather conditions. The authors argued that sunlight is an important determinant of bacterial death rates and that plant anatomy controls the degree to which effluent droplets on plant surfaces are exposed to sunlight. Greenhouse studies (Brown, Jones and Donnelly 1980) showed that 25 millimeters of simulated rainfall, falling in 1 hour, reduced *E. coli* on grass by 90–99.9 percent. In the absence of rainfall, the same level of reduction under the same conditions took 10–25 days.

Sadovski and others (1978) studied the survival of a mutant *E. coli* inoculated into waste stabilization pond effluents that were applied to drip irrigation to cucumber plots on two farms in Israel. At one site (air temperature 13–30°C, soil temperature at noon 22–30°C, sunlight 9.5 hours per day, relative humidity 27–55 percent), a single irrigation was performed with inoculated effluent containing $10^7$ mutant *E. coli* per 100 milliliters. Mutant *E. coli* were still detectable in the irrigation water, at a concentration of $>10^3$ per 100 milliliters, 8 days after the flow of inoculated effluent. The soil contamination immediately after irrigation with inoculated effluent was $10^7$ mutant *E. coli* per 100 grams of dry soil and persisted at a level of $>10^6$ per 100 grams for at least 8 days. Cucumbers grown in exposed soil were contaminated by $10^4$ mutant *E. coli* per 100 grams immediately following the inoculated irrigation, and this contamination fell to 65 per 100 grams after 8 days. When the soil and drip lines were

covered with polyethylene sheets to reduce evaporation and raise temperature, no mutant *E. coli* could be detected on cucumbers 1 day following the inoculated irrigation. At the second site (air temperature 23–28°C, soil temperature at noon 40–43°C, sunlight 11.8 hours per day, relative humidity 62–70 percent), three irrigations were performed with inoculated effluent containing $4.4 \times 10^{11}$ mutant *E. coli* per 100 milliliters. After the third inoculated irrigation the soil contained $1.3 \times 10^6$ mutant *E. coli* per 100 grams (dry weight), and this contamination fell to 130 per 100 grams after 9 days and maintained this level for a further 11 days. Unlike at the first site, where irrigation with uninoculated effluent had continued throughout the study, in this case the irrigation terminated 5 days after the third inoculated irrigation, and consequently the soil moisture content fell from 15 percent to 3 percent. Bacterial contamination on cucumbers grown in exposed soil rose to $1.7 \times 10^3$ mutant *E. coli* per 100 grams but rapidly declined following the last inoculated irrigation. When the soil and drip lines were covered with polyethylene sheets, no mutant *E. coli* could be detected on the cucumbers. Earlier studies at the first site by the same workers (Sadovski, Fattal and Goldberg 1978) showed that harvested cucumbers and eggplants, irrigated with sewage effluent containing $10^6$ fecal coliforms per 100 milliliters, were contaminated by, on average, 389 fecal coliforms per 100 grams when drip-irrigated throughout the growing season on exposed soil, but by only 30 per 100 grams when drip-irrigated on soil covered by polyethylene sheets. Irrigating with sewage effluent only during the early stage of growth (up to flowering) produced a final contamination level on harvested vegetables similar to that found when irrigation was with fresh water (around 2.5 fecal coliforms per 100 grams).

Other reports of coliform survival on crops are listed in the appendixes of Feachem and others (1980) and have been reviewed elsewhere (for instance, Elliott and Ellis 1977; Geldreich and Bordner 1971; Rudolfs, Falk and Ragotzkie 1950). Fecal bacteria in soil may be in relatively sheltered and supportive microhabitats where moisture, shade, and nutrient availability permit survival for many weeks. In contrast, bacteria on crop surfaces will in general be exposed to desiccation and sunlight, and their survival is very much shorter than in soil. Rapid death is promoted by high air temperatures, bright sunlight, low relative humidity, and a plant anatomy that does not offer many sheltered sites. Under most conditions, fecal indicator bacteria are unlikely to survive for more than 4 weeks on crop surfaces with at least a 99 percent reduction taking place within 1 week. In arid regions with low cloud cover, complete elimination is likely to take place within 1 week.

The reduction of fecal contamination of harvested crops depends not only on the microbial death rates on plant surfaces but also on the technology employed for irrigation or fertilization. The use only of treated effluent, night soil or sludge; the application of the fecal material to the soil (for instance, by drip irrigation) rather than over the crops (for instance, by spray irrigation); covering the soil with plastic sheets to reduce evaporation; and the cessation of irrigation about two weeks prior to harvesting will all greatly reduce crop contamination.

### *In fish and shellfish*

Fish and shellfish that live in water contaminated by fecal discharges are frequently found to contain fecal indicator bacteria. Several studies have shown that these bacteria are not part of the normal flora of the intestines of freshwater or saltwater fish (Geldreich and Clarke 1966; Guélin 1962). Fish intestines may contain fecal coliforms and fecal streptococci only when the fish have been living in fecally contaminated water, and these bacteria may survive, and perhaps multiply, for periods of up to 14 days (Glantz and Krantz 1965) in the fish intestines.

Most investigations have concentrated on the bacterial contamination of shellfish rather than fish. This is because the method of filter feeding of bivalve molluscs concentrates bacteria in the same way as it concentrates viruses (see chapter 9; Metcalf 1978; Wood 1979) and because molluscs are often eaten raw or only lightly cooked. Goyal, Gerba and Melnick (1979) investigated oyster beds in Galveston Bay (Texas, USA) and found fecal coliform concentrations per 100 milliliters of up to 2,400, 46,000, and 46,000, respectively, in water, sediment, and oysters. Similar results were obtained by Slanetz, Bartley and Stanley (1968). Munger, Heyward and Dutton (1979) recorded that fecal coliform concentrations in clams in the Seattle area (Washington, USA) were up to 59 times higher than in the surrounding water.

Mitchell and others (1966) studied the uptake and elimination of *E. coli* by the Eastern oyster (*Crassostrea virginica*) in sterilized seawater at 20°C. When the seawater was inoculated with $10^3$ *E. coli* per milliliter, the oysters accumulated over $10^4$ *E. coli* per gram within 4 hours. When the seawater contained about 10 *E. coli* per milliliter, the oysters accumulated over 100 per gram within 4 hours. When the contaminated oysters were rinsed in clean water and

placed in sterilized seawater, the *E. coli* concentrations declined by 99 percent in 10 hours, and fell below the level of detection after 50 hours. In similar experiments Hoff and Becker (1969) reported that Olympia oysters (*Ostrea lurida*), in sterilized seawater containing 10 *E. coli* per milliliter, accumulated 110–320 *E. coli* per gram after 24 hours at 6–11°C. When the oysters were rinsed and replaced in sterilized seawater, *E. coli* levels fell to about 1 per gram after 48 hours.

Jegathesan and others (1976) purchased three species of commonly eaten shellfish (cockles, *Anadura granosa*, and two species of mussels, *Modiolus senhaussi* and *M. metcalfi*) from markets in Malaysia and examined them for bacterial enteric pathogens. Of twenty cockles examined, nine contained coliforms (mean concentration of $8.9 \times 10^4$ per gram), and seven isolations of pathogenic serotypes of *E. coli* were made. Of eighteen mussels examined, six contained coliforms (mean concentration of $1.2 \times 10^6$ per gram), and twelve isolations of pathogenic serotypes of *E. coli* were made. The authors noted that these shellfish are normally eaten partially cooked in Malaysia.

Although most attention in the developed countries has turned to the risks of contaminated shellfish transmitting excreted virus (especially hepatitis A virus, rotavirus, and Norwalk agent), the risks of bacterial infections due to pathogenic *E. coli*, shigellae, salmonellae, and *Vibrio cholerae* being spread by shellfish harvested from polluted waters are very real (Hughes, Merson and Gangarosa 1977; Janssen 1974).

Considerable debate surrounds the use of quality standards for shellfish growing waters based on permissible concentrations of fecal indicator bacteria (Cabelli 1978; Evison 1979; Metcalf 1978). The USA sets limits of 70 coliforms per 100 milliliters (median value) and 14 fecal coliforms per 100 milliliters (median value) for waters used for shellfish production. It is widely accepted, however, that these bacteriological measures are poor indicators of the risk of viral contamination, and most countries have yet to legislate for shellfish water quality or to decide whether a standard based on indicator bacteria alone is adequate.

### *In the air*

Airborne droplets of water and wastewater may contain excreted bacteria, and these may cause respiratory or enteric infection if inhaled in sufficient numbers by a susceptible host. The mechanisms by which aerosolized bacteria may be produced are similar to those that generate viral aerosols (see chapter 9). Baylor, Peters and Baylor (1977) bubbled air through a column of liquid containing *E. coli* and produced droplets that contained an average concentration of *E. coli* 30 times greater than that in the column. In similar experiments Blanchard and Syzdek (1970) produced droplets that contained up to 1,000 times more *Serratia marcescens* per milliliter than the water column from which the droplets were produced. The concentration of bacteria in the droplet depended on the size of the droplet, with highest concentrations occurring in droplets 60–80 micrometers in diameter.

The aerosolized excreted bacteria most encountered by people in developed countries are those produced by the flush toilets in their houses. Darlow and Bale (1959) inoculated wash-down toilet bowls with $10^{11}$–$10^{12}$ *Serratia marcescens* and investigated the production of airborne organisms when the low-level, 9-liter cistern was flushed. At the level of the seat, near the bowl, $7 \times 10^4$ *Serratia* per cubic meter of air were isolated, and 1.2 meters above the seat the concentration was $7 \times 10^2$ per cubic meter. About 10 minutes after flushing there remained 70 bacteria per cubic meter widely distributed about the toilet room. A tenfold reduction in the inoculum to the bowl reduced the aerosol concentrations by about one-quarter. A second flush, 15 minutes later and without reinoculation of bacteria, still produced $2.8 \times 10^4$ bacteria per cubic meter at seat level. Swabs taken from surfaces throughout the room yielded *Serratia*. Adding sheets of toilet paper to the bowl before flushing did not affect aerosol production by the first flush, but increased aerosol production by the second flush by retaining a greater number of organisms in the bowl during the first flush. Even with the lid closed, the first flush produced $3.1 \times 10^4$ bacteria per cubic meter of air at seat level. Aerosol production was unaffected by whether the seat was covered by its lid or by a cardboard replica of the human buttocks.

Bound and Atkinson (1966) compared aerosol production by wash-down and syphonic types of flush toilets, each flushed by a low-level, 9-liter cistern. The bowls were inoculated with a suspension of *E. coli*, and air samples were taken 0.4 meters from the bowl at seat level. Wash-down toilets produced on average 13 *E. coli* per cubic meter of air, whereas syphonic bowls produced only 0.9 per cubic meter. Closing the lid before flushing did not affect aerosol production.

Newson (1972) studied hospital toilets in England. He sampled water from the bowls of toilets in normal use and found no *E. coli* in 62 percent of samples (109 of 176); only 5 percent of samples (9 of 176) contained more than $2.5 \times 10^5$ per 100 milliliters. The seats, lids, flush handles, and door handles in the same toilets were swabbed, and 6 percent of specimens (19 of 293) were positive for *E. coli*. When $10^{11}$ *E. coli* were added to a

bowl, an aerosol of up to 186 *E. coli* per cubic meter of air was produced. The same procedure also produced 70–80 visible droplet splashes, of which 39–75 contained *E. coli*. More droplet splashes were produced by high-level cistern flushes and, surprisingly, syphonic bowls generated more splashes than wash-down bowls. The first flush reduced the concentration of inoculated *E. coli* in the bowl water from $10^8$ to $10^6$ per 100 milliliters; the second flush took the concentration down to $10^3$ per 100 milliliters; and the third flush left no *E. coli* detectable in bowl water.

Gerba, Wallis and Melnick (1975*b*) seeded $10^{11}$ *E. coli* into a flush toilet bowl with a 14-liter cistern flush. Total numbers of *E. coli* in the bowl water were reduced to $10^7$ after one flush, $10^4$ after two flushes, and remained at around $10^4$ for at least seven flushes. This was because *E. coli* were adsorbed to the porcelain inner bowl surface and were gradually eluted by successive flushes. Public toilet bowls in normal use were sampled and found to contain between $10^2$–$10^8$ coliforms per total bowl volume. One flush ejected 27–104 visible droplets, and up to $6.6 \times 10^4$ *E. coli*, when $10^{11}$ organisms were seeded into the bowl. A reduction of 5 log units in the number of *E. coli* in the bowl reduced the number ejected by a flush by under 3 log units. The numbers of *E. coli* ejected were not appreciably affected by whether the bowl contained similar original numbers in culture, homogenized stool, or fecal pellets. An estimated 800–1000 bacteria fell out on bathroom surfaces after a flush ($10^{11}$ seeded *E. coli* in the bowl), and 75–80 percent of these fell out in the first 2 hours after the flush. Coliforms were detected on surfaces of all of twenty private and public toilets in normal use. Walls, floors, toilet seats, and bowl rims were the most contaminated surfaces.

Sewage treatment plants, especially those that involve the pumping of air or oxygen into sewage or the mechanical aeration of a sewage tank, produce aerosol droplets that contain excreted bacteria as well as excreted viruses (see chapter 9). Bitton and Smith-Holmes (1978) sampled air above the aeration tank of a package sewage treatment plant, above an activated sludge plant, and on the roof of a building in Florida (USA). In a cubic meter of air above the package plant there were 154 coliforms, 22 fecal coliforms, and 2 fecal streptococci; above the activated sludge plant there were 165 coliforms, 27 fecal coliforms, and 5 fecal streptococci; and on the roof there were 16 coliforms, 2 fecal coliforms, and 1 fecal streptococcus.

Fannin and others (1977) investigated airborne coliform bacteria near an activated sludge and a trickling filter sewage treatment plant in Michigan (USA). The sewage contained $2 \times 10^{10}$ coliforms per 100 milliliters. Within 5 meters of the source, the air at the activated sludge plant contained 130–390 coliforms per cubic meter, whereas at the trickling filter plant the air contained 21–79 coliforms per cubic meter. Wind speed and air temperature were significantly correlated with the concentration of airborne coliforms. Katzenelson, Teltch and Shuval (1977) isolated up to 452 coliforms per cubic meter of air 30 meters downwind of an aerated lagoon in Israel.

Goff and others (1973) isolated up to 965 coliforms per cubic meter of air 100 meters downwind from trickling filter plants in the USA. Coliform aerosol detection increased at high relative humidities and low solar radiation. Cronholm (1980) detected aerosolized enteric bacteria up to 930 meters downwind from small activated sludge plants in Kentucky (USA). Foliage downwind of the plants was contaminated by 1–830 enteric bacteria per square millimeter of leaf surface. Six mice forced to inhale air at a sewage treatment plant and observed for 2 weeks exhibited no symptoms, and cultures of their respiratory organs were negative for enteric bacteria.

Fedorak and Westlake (1980) sampled total airborne bacteria at an activated sludge plant at Edmonton (Canada) and found up to $1.8 \times 10^3$ per cubic meter of air, compared to a background level of 27 per cubic meter. Similar bacterial concentrations (up to $2.6 \times 10^3$ per cubic meter) were found indoors near taps used to sample sewage and sludge from the treatment plant. Increased airborne bacterial concentrations occurred in the laboratory when sludge was being dispensed for analysis and when the floor was being mopped.

Randall and Ledbetter (1966) studied the aerosols produced by an activated sludge plant in Texas (USA). The opportunistic respiratory pathogens *Klebsiella*, *Aerobacter*, and *Proteus* constituted 11 percent of all isolates, 19 percent of isolates of enteric origin, and 56 percent of Enterobacteriaceae. The *Klebsiella-Aerobacter* group survived for longer in the air than other enteric bacteria and made up an increasingly high proportion of isolates further from the activated sludge tanks. The authors suggested that *Klebsiella* should be used as an indicator of bacterial air pollution from sewage treatment plants.

Crawford and Jones (1978) studied the emissions of airborne bacteria from aerated grit removal tanks at a sewage treatment plant in Toronto (Canada). The rate of bacterial emission from the tanks was strongly inversely correlated to relative humidity but not to air temperature, pressure, or sewage flow rate. The rate of decay of downwind airborne bacterial concentrations

was correlated only to wind speed. When the decay rates of various indicator bacteria were compared it was found that fecal coliforms decayed faster than total coliforms, which decayed considerably faster than fecal streptococci. By the use of a mathematical model, the authors predict a maximum downwind travel distance of 505 meters for fecal coliforms and 757 meters for *Str. faecalis*.

The source of excreted bacterial aerosols that is receiving the most scientific attention at present is the spray irrigation of sewage effluents. Sorber and others (1976) investigated airborne bacteria produced by the spray irrigation of secondary sewage effluent at a golf course in Arizona (USA). The effluent contained a mean of $3.7 \times 10^5$ coliforms and 170 fecal streptococci per 100 milliliters, and the average background level of coliforms in the air was 2.4 per cubic meter. At 47 meters downwind of the sprinklers up to 330 coliforms per cubic meter were recovered, and at 152 meters only 30 coliforms per cubic meter were found. Coliform levels significantly higher than the background were detected up to 198 meters downwind. The aerosolization efficiency (the proportion of effluent that was divided into droplets sufficiently small to remain airborne) was estimated as 0.32 percent. The decay rate of airborne bacterial concentrations was markedly reduced at night. In subsequent experiments at the same site, effluent contained $2.8 \times 10^5$ coliforms, $2.3 \times 10^4$ fecal coliforms, and $1.3 \times 10^3$ fecal streptococci per 100 milliliters (Bausum, Schaub and Kenyon 1978). The concentrations of aerobic bacteria-bearing particles per cubic meter of air, downwind of the sprinklers, were up to 10,500 at 46 meters, up to 4,700 at 76 meters, up to 3,200 at 100 meters, up to 500 at 150 meters, and up to 13 at 560 meters. (All these concentrations are expressed as values in excess of the background levels of 15–198 colony-forming particles per cubic meter.) When the effluent was heavily chlorinated prior to spraying, colony-forming particles fell to between 0 and 57 per cubic meter at 46 meters downwind. The proportion of Enterobacteriaceae in the total aerobic bacterial flora was 2 percent in the effluent but 26 percent in the aerosol isolates.

Parker and others (1977) investigated a spray irrigation system that utilized effluent from a potato-processing plant. The sprayed effluent contained $1.6 \times 10^6$ coliforms per 100 milliliters. At 15 meters downwind there were up to 1,100 coliform-bearing particles per cubic meter, and at 1–1.5 kilometers downwind there were up to 30 coliform-bearing particles per cubic meter.

A series of experiments were conducted in Israel to investigate the production of airborne bacteria at spray irrigation sites utilizing sewage effluents (Katzenelson and Teltch 1976; Katzenelson, Teltch and Shuval 1977; Teltch and Katzenelson 1978). Coliform bacteria were found in the air up to 350 meters downwind from the irrigation site. The concentration of bacteria in the air was directly related to the concentration in the effluent, and coliforms could only be detected in the air when their concentration in effluent exceeded $10^5$ per 100 milliliters. Coliforms survived for longer in aerosols when relative humidity was high, when solar radiation was low, and when the effluent contained a higher concentration of organic matter. Up to 10 times more aerosolized bacteria were detected at night than during the day. Earlier studies in Israel (Katzenelson, Buium and Shuval 1976) had suggested that residents on kibbutzim that practised spray irrigation with sewage effluent experienced a higher incidence of some bacterial excreted infections (shigellosis, salmonellosis, and typhoid) than members of kibbutzim that practiced no form of wastewater irrigation. These findings have been doubted and appear to be contradicted by subsequent work (Feliciano 1979).

Airborne excreted bacteria can be produced by many situations other than toilet flushing, sewage treatment, or spray irrigation. Edmonds and Littke (1978) sampled airborne coliforms 80 millimeters above anaerobically digested, dewatered (20–40 percent solids) sludge applied to land. Coliforms in the sludge over a 7-month sampling period were between $5 \times 10^5$ and $3.5 \times 10^7$ per gram, while coliforms in the air ranged from 0 to $1.5 \times 10^4$ per cubic meter. Maximum coliform emission was associated with no rainfall and high air temperature, wind speed, and solar radiation. Adams and others (1980) have reported on bacterial aerosol emissions from cooling towers using disinfected sewage effluents or polluted river water.

The risks to health associated with inhaling aerosolized bacteria depend on factors such as the dose inhaled, the dose required to cause an infection, and the aerosol size. Katzenelson, Teltch and Shuval (1977) estimated that an individual working 100 meters from an effluent sprinkler in Israel would inhale about 36 coliforms every 10 minutes. Considering the very high ratio of coliforms to pathogens, this rate of inhalation appears low. Infective doses for some pathogens may be lower, however, by the respiratory route than by the alimentary route. Crozier and Woodward (1962) reported that typhoid fever was established in chimpanzees by the respiratory route with doses of *S. typhi* a thousandfold less than those needed for infection by oral challenge.

The infective dose of inhaled bacteria depends in part upon the ability of the aerosols to penetrate deep

into the lungs. Lung penetration is especially important in the establishment of respiratory infections. Aerosols that penetrate best are those which are less than 5 or 6 micrometers in diameter (Druett, Henderson and Peacock 1956; Druett and others 1953; Harper and Morton 1953; May and Druett 1953). Reported aerosol sizes from sewage sources vary considerably but, in general, smaller aerosols predominate as distance from the source increases because larger particles have settled. Although only small aerosols are likely to penetrate to the lower respiratory tract, it is probable that bacteria (and viruses) in larger aerosols caught in the upper respiratory tract may subsequently be swallowed.

Baylor, Peters and Baylor (1977) found a significant negative correlation between droplet size and the concentration of *E. coli* within a droplet. Droplets produced by surf and blown into the beach had a mean size of 35 micrometers at the water's edge and 21 micrometers 10 meters up the beach (Baylor and others 1977). Sorber and others (1976) found that 50 percent of airborne bacteria at a spray irrigation site were associated with particles in the range of 1–5 micrometers. Darlow and Bale (1959) found that a toilet flush produced aerosols with a mean diameter of 2.3 micrometers, 87 percent of them being less than 4 micrometers. Goff and others (1973) recorded that the majority of aerosols emitted by trickling filter plants were less than 5 micrometers in diameter. Randall and Ledbetter (1966) found that 40 percent of viable airborne bacteria in the immediate vicinity of activated sludge units were associated with particles of less than 5 micrometers in diameter, but that this proportion rose to 70 percent at a downwind distance of 6 meters. Teltch and Katzenelson (1978) found that the median aerosol size produced by effluent sprinklers in Israel was greater than 7 micrometers but that 50 percent of bacteria sampled were associated with aerosols of less than 7 micrometers in diameter.

The studies reported above, and the laboratory investigations of Poon (1968), clearly show that, although bacterial aerosol production may be increased by low relative humidity, high temperature, high solar radiation, and high wind speed, these same conditions also promote rapid death and dispersion of bacteria in the air. Thus the dissemination of aerosolized bacteria from treatment plants and spray irrigation sites in hot arid climates should be appreciably less than that reported from temperate climates. In addition there is no convincing epidemiological evidence that those exceptionally exposed to sources of bacterial aerosols (for instance, workers in fields receiving sprayed effluent or sewage plant operators) have higher infection or disease rates than others (Feliciano 1979). It may be that, in most situations, the inhalation of excreted bacteria is insignificant compared with the ingestion of the same pathogens and that the numbers of bacteria inhaled are usually well below the required infective doses.

## Inactivation by Sewage Treatment Processes

The fate of bacterial indicators of pollution in sewage treatment processes has been the subject of a number of studies over the past 50 years. The literature is not, however, as extensive as might be expected, partly because it is only very recently that the importance of *E. coli* as an enteric pathogen has been recognized and partly because the traditional focus of attention has been the ability of sewage treatment processes to improve the physicochemical quality, rather than the microbiological quality, of sewage. Indeed, interest in the performance of sewage treatment plants in removing enteric bacteria has been so low that many engineers are unaware of the poor bacterial quality of secondary effluents from conventional treatment plants.

The interest in enteric bacteria in sewage treatment processes is now increasing for two reasons. First, more stringent effluent quality legislation is being introduced in the developed countries, and this has promoted research into disinfection and tertiary treatment as methods for improving the quality of unsatisfactory secondary discharges. Second, there is growing awareness in developing countries of the dangers of discharging highly pathogenic secondary effluents into streams that downstream communities may use for domestic purposes.

### *By primary and secondary sedimentation*

Primary sedimentation tanks, with retention times of 2–6 hours, produce quite variable results in removal of indicator bacteria (see the appendixes to Feachem and others 1980). Usually a reduction of fecal coliforms and fecal streptococci is reported in the range 0–60 percent. For instance, at two sewage treatment plants in Scotland, primary sedimentaton removed on average 32 and 50 percent of *E. coli* and 57 and 53 percent of *Pseudomonas aeruginosa* (Wheater and others 1980). Removal is primarily caused by settlement of bacteria, which are adsorbed to, or entrapped within, solid particles in the influent sewage. The data on bacterial dieoff in sewage reported above

suggest that only a very small reduction (< 10 percent) would be expected due to natural death in the short period during which the sewage resides in the sedimentation tank. Growth of indicator bacteria, especially of total coliforms, is sometimes recorded in sedimentation tanks.

*By storage*

Storage will be an especially effective method of reducing enteric bacteria in sewage at warm tropical and subtropical temperatures (say, >25°C). Although little specific information is available, it may be anticipated that fecal bacteria in warm sewage would have a $t_{90}$ of 120 hours or less.

*By septic tanks*

The removal of fecal indicator bacteria by septic tanks has attracted increased interest recently because of concern about the pollution of groundwater by septic tank effluents. A septic tank is simply a settling chamber, or chambers, usually having retention time of 3 days or less. In poorly designed tanks, or those requiring desludging, there is very considerable solids carryover into the effluent. Enteric bacteria are removed both by death in the anaerobic liquor and by association with solids that settle to the sludge layer. The short retention times, and poor sedimentation performance that is often the result of insufficiently frequent desludging, are the reasons why high concentrations of fecal indicator bacteria are found in septic tank effluents.

Viraraghavan (1978) reported that a septic tank in Canada produced an effluent containing geometric mean values of $2.3 \times 10^6$ coliforms, $1.6 \times 10^5$ fecal coliforms, and $1.1 \times 10^5$ fecal streptococci per 100 milliliters. Brandes (1978*a*) reported that approximately 2.5 million residents of Ontario (Canada) use septic tank systems. He studied three septic tanks with retention times of 2–10 days and recorded the following ranges of concentrations of fecal coliforms per 100 milliliters: $4 \times 10^5$–$2 \times 10^6$ in first compartment supernatant, $1 \times 10^5$–$1 \times 10^6$ in second compartment supernatant, $9 \times 10^5$–$8 \times 10^6$ in first compartment sludge, $6 \times 10^4$–$6 \times 10^5$ in second compartment sludge, and $5 \times 10^5$–$4 \times 10^6$ in the effluents (see also Brandes 1978*b*). McCoy and Ziebell (1976) sampled effluents from five septic tanks in the USA with retention times of 2–13 days and found geometric mean values of $4.2 \times 10^5$ fecal coliforms and $3.8 \times 10^3$ fecal streptococci per 100 milliliters.

McGarry and Stainforth (1978) have described a three-compartment septic tank used in Kiangsu Province, China (see also figure 6-1 in Part One). The first two compartments have retention times of 10 days each, and the third has a retention time of 30 days. Tests on these tanks showed that, in winter at temperatures of 3–7°C, *E. coli* reductions of 4 log units were achieved (suggesting a $t_{90}$ of 300 hours).

The data (see the appendixes of Feachem and others 1980) suggest that in a well-designed and well-maintained septic tank with 3 days' retention in a warm climate (>25°C), a reduction of fecal indicator bacteria of 50–95 percent may be achieved. In poorly designed and poorly maintained tanks (the most usual kind), little or no reduction can be expected. It must be stressed, however, that the distinction between a 90 percent removal (say, influent = $10^6$ per 100 milliliters; effluent = $10^5$ per 100 milliliters) and a 10 percent removal (say, influent = $10^6$ per 100 milliliters; effluent = $9 \times 10^5$ per 100 milliliters) is trivial. In either case the effluent is heavily contaminated with enteric bacteria, and the ultimate fate of these bacteria depends on the method of disposal of the effluent and the sludge.

Effluents are normally discharged to soakaway pits or drainfields where bacteria may be retained in the soil and eventually die. Under certain conditions, however, fecal bacteria may travel from the drainfield to pollute shallow groundwater aquifers or nearby wells. Kudryavtseva (1972) recorded that coliforms inoculated into saturated alluvial sands (percolation rate 13 meters per day) in the USSR traveled for a horizontal distance of not more than 3 meters in the direction of groundwater flow. However, the same author cited other data from the USSR indicating a horizontal travel of indicator bacteria of 850 meters through pebble deposits and 1 kilometer through weathered limestone.

Several studies have shown that the travel of bacterial indicators through soil from pit latrines or septic tank drainfields decreases over time as the soil becomes increasingly clogged with fecal solids and biological slime. Caldwell and Parr (1937) found that, after two months of operation, fecal coliforms and *Clostridium perfringens* could be recovered at 8 meters, and occasionally at 11 meters, from a bored hole latrine. After 7 months, however, bacterial travel was less than 1.5 meters. McCoy and Ziebell (1976) applied septic tank effluent ($5.1 \times 10^6$ fecal coliforms and $7.3 \times 10^6$ fecal streptococci per 100 milliliters) to 0.6 meter deep columns of loamy sand at 25°C. At an application rate of 0.1 cubic meters per square meter per day, 92 percent of fecal coliforms were removed over the first 100 days of application. After this time a

clogging zone developed at the top of the column, and removal of fecal coliforms improved to 99.999 percent. Fecal streptococci removal rates were around 3 log units during the first period and over 6 log units after 100 days. In field experiments it was found that an effluent contained $1.9 \times 10^6$ fecal coliforms per 100 milliliters; the soil in the clogging zone at the base of the drainfield trench contained $4 \times 10^6$ per 100 grams; and the soil 0.3 meters below the clogging zone contained less than 200 fecal coliforms per 100 grams. The authors noted, however, that these are optimal removals under ideal conditions in nonaggregated soils.

Brown and others (1979) studied the movement of septic tank effluent containing $10^6$ fecal coliforms per 100 milliliters through a sandy loam, a sandy clay, and a clay, with percolation rates of 6, 0.9, and 0.06 meters per day, respectively. Fecal coliform concentrations decreased greatly with increasing distance from the septic line, and a 90–99 percent reduction in 50 millimeters was common. In most tests fecal coliforms were not present more than 0.3 meters below the septic line, and only very occasionally were they detected in leachate drawn from 1.2 meters below the septic line. The tendency for nearly all fecal coliforms to be concentrated at the interface between the gravel packing around the septic line and the soil was especially marked in soils of low permeability and increased with time of effluent application for all soils. Fecal coliform concentrations in the soil decreased by about 99 percent in 2 weeks following the termination of effluent application.

Studies on septic tank drainfields have clearly shown that the risks of groundwater pollution are very much greater if the drainfield is located in, or only a little above, the saturated zone. Viraraghavan (1978) studied the movement of indicator bacteria horizontally through sandy clay and clay (percolation rates 0.02–1.0 meters per day) from a septic tile discharging effluent containing about $10^5$ fecal coliforms and fecal streptococci per 100 milliliters. The septic line was 0.6 meters below ground level, and the water table was only 0.15 meters or less below the line. Fifteen meters downslope from the septic line the groundwater contained about $10^2$ fecal coliforms and $10^2$–$10^3$ fecal streptococci per 100 milliliters.

Hagedorn, Hansen and Simonson (1978) seeded antibiotic-resistant *E. coli* and *Str. faecalis* into two pits, 0.3 and 0.6 meters deep, dug into saturated silty loam and clay loam (percolation rates 0.01–0.2 meters per day) in Oregon (USA). The bacteria were detected in wells 15 meters from the pits within 8–16 days of inoculation, and they survived in appreciable numbers in the saturated soils throughout a 32-day sampling period. Reneau and others (1977) recorded the travel of fecal coliforms for a horizontal distance of at least 28 meters from a septic tank drainfield through saturated sandy loam in Virginia (USA). At one site, fecal coliform concentrations were up to $4.6 \times 10^4$ per 100 milliliters adjacent to a drainfield but were never more than 430 per 100 milliliters at a distance of 28 meters (see also Reneau and Pettry 1975). Further important work on bacterial movement through saturated soil was reported by McCoy and Hagedorn (1979) and Rahe, Hagedorn and McCoy (1979).

Other literature on the retention and survival of bacteria in soil is reviewed above in the sections on groundwater and soil and below in the sections on filtration and land treatment. A detailed review of the fate of enteric bacteria in septic tank and drainfield systems has been published (Small Scale Waste Management Project 1978).

Septic tank sludge is rich in excreted bacteria (Brandes 1978*a*, 1978*b*) and requires treatment by digestion, drying, or composting prior to application to agricultural land. The destruction of fecal indicator bacteria in tanks, in drainfields, and in sludges will be more rapid at warmer temperatures; therefore, given correct design and good maintenance, performance in developing countries may often be better than that reported from temperate areas.

### *By trickling filters*

Little information is available on the performance of trickling filters as a unit process. Most studies report the removal of fecal indicator bacteria across a complete trickling filter plant (pretreatment–primary sedimentation–trickling filters–secondary sedimentation).

Literature on fecal indicator bacteria removal in trickling filters is listed in the appendixes of Feachem and others (1980). In a well-operated plant the trickling filter itself may remove 20–80 percent of influent enteric bacteria, whereas the total treatment plant will remove 70–97 percent. Poor maintenance or overloading will result in considerably lower removal rates. Fecal coliform and fecal streptococci removal rates are generally similar.

In Britain it is common for the removal of fecal coliforms across complete trickling filter plants to be 90–95 percent. This poor removal performance results in an effluent containing $10^4$–$10^7$ fecal coliforms per 100 milliliters. Wheater and others (1980) recorded that two trickling filter plants in Scotland achieved average overall *E. coli* reductions of 87 and 90 percent

and produced effluents containing up to $6.9 \times 10^6$ and $1.4 \times 10^6$ *E. coli* per 100 milliliters.

*By activated sludge*

The removal of fecal indicator bacteria by activated sludge processes is poor, although removal is somewhat better than by trickling filters. The mean retention time in the aeration tanks (6–12 hours) is such that only a less than 50 percent reduction by natural die-off would be expected, even at warm temperatures and assuming that all the liquor were held for the mean retention time. Most reduction of indicator bacteria is in practice achieved by adsorption to flocs, which are subsequently removed in secondary sedimentation tanks, and by the grazing of ciliated protozoa (Van der Drift and others 1977).

Studies on removal of fecal indicator bacteria by activated sludge are listed in the appendixes of Feachem and others (1980). Reductions of influent fecal bacteria are between 0 and 99.9 percent, with some experimenters reporting increasing numbers during aeration. Most experience suggests that reductions will be 80–99 percent across a complete activated sludge plant that is well-operated and well-maintained but that does not include effluent disinfection or tertiary treatment. Overloaded or poorly maintained plants will achieve very much lower removal rates. These low levels of removal (always less than 2 log units) mean that activated sludge plant secondary effluents contain high concentrations of enteric bacteria. Berg and Metcalf (1978) reported $1.1 \times 10^5$–$4.9 \times 10^6$ fecal coliforms per 100 milliliters of activated sludge effluent. The settled sludge from the secondary sedimentation tanks will also contain a high concentration of fecal indicator bacteria that have adsorbed to flocs in the aeration tanks.

*By oxidation ditch*

Practically no information has been published on removal of fecal indicator bacteria by oxidation ditches, although a certain amount is known about removal of *Salmonella* (see chapter 15). The process is essentially similar to that of activated sludge, but the longer hydraulic retention times (1–3 days), and the higher proportion of sludge recycling giving a solids retention time of 10–30 days, are features that should produce improved bacterial removal. Laboratory studies in the USSR indicated that coliforms declined by 6 log units after 3 days in an oxidation ditch, and that an EPEC strain (O111) survived for 3–7 days when seeded at a concentration of $10^5$ per 100 milliliters and for 15–18 days when seeded at $10^7$ per 100 milliliters (Goncharuk and others 1970).

*By waste stabilization ponds*

The removal of *E. coli* and other fecal indicator bacteria in waste stabilization ponds has been studied by many investigators throughout the world during the last 30 years (see the appendixes of Feachem and others 1980). It is now well established that ponds, if properly designed, can achieve substantially higher removal rates of fecal bacteria (and indeed of other excreted pathogens) than other forms of sewage treatment. For example, Mara and Silva (1979) report the reduction of fecal coliform bacteria in a series of five ponds in northeast Brazil, with a total retention time of 29 days and an average temperature of 26°C, from $5 \times 10^7$ per 100 milliliters in raw sewage to 17 per 100 milliliters in the final effluent; this represents a very high overall reduction of 99.99996 percent.

MECHANISMS OF *E. COLI* REMOVAL IN PONDS. There is a variety of environmental factors that are considered to be responsible, or at least partially so, for the removal of *E. coli* and other fecal indicator bacteria in ponds. These factors include time and temperature, ultraviolet radiation, the antibacterial effect of extracellular algal toxins, low concentrations of dissolved carbon dioxide, high pH, high (especially supersaturated) concentrations of dissolved oxygen, and predation by the microinvertebrate fauna. These factors are reviewed briefly below, but it should be emphasized that their relative importance, apart from perhaps that of time and temperature, is largely unknown.

Compared with other forms of sewage treatment, ponds are characterized by long mean hydraulic retention times, ranging from a few weeks in hot climates to several months in cold climates. Thus, ponds provide a considerably greater opportunity for fecal bacterial removal than other treatment processes. It is now well established (Mara 1976), both theoretically and from field observation, that removal of fecal bacteria is greater in a series of ponds than in a single pond providing the same overall hydraulic retention time, and that this efficiency increases with the number of ponds in the series. The microbial flora and fauna vary considerably from pond to pond in a series of ponds, and it is therefore likely that the relative effect on fecal bacterial removal of the environmental factors discussed below changes in a similar manner.

Moeller and Calkins (1980) investigated the effect of the ultraviolet component of solar radiation (wavelength range: 280–320 nanometers) on the removal of fecal coliforms in a series of four maturation ponds treating the effluent from a conventional activated sludge plant in Kentucky (USA). The overall hydraulic

retention time in the pond series was 5–7 days, and the pond temperatures varied from below 10°C to above 25°C. No relationship between fecal coliform removal and either temperature or algal density was found, but a significant correlation between fecal coliform removal and the received dosage of ultraviolet radiation was apparent, with the bacterial removal rate being directly proportional to the dose received.

Several investigators have studied the direct and indirect effects of pond algae on the removal of fecal bacteria. Many common pond microalgae produce antibacterial substances that have been shown to be inhibitory to *E. coli* (Davis and Gloyna 1972), although *in vitro* toxicity tests have shown that the degree of inhibition effected by different algae not only varies from alga to alga, but that there appears to be a marked synergistic effect when two or more algae are present, with the synergism increasing with the number of algal species present. Moreover, removal of fecal bacteria in samples of pond water is higher than in laboratory mixtures of large numbers of different algal species (Jackson 1979).

Algal demand for carbon dioxide is often greater than its supply as an end-product of pond bacterial metabolism, with the result that bicarbonate ions reverse to carbon dioxide and hydroxyl ions, leaving an excess of the latter that raises the pH of the pond. High pH values (above 9.5) are known to be detrimental to the survival of *E. coli* in ponds (Parhad and Rao 1974), although Gray (1975) suggests that, since carbon dioxide is an essential growth factor for *E. coli*, its unavailability to *E. coli* as a result of its rapid utilization by photosynthesizing algae is an important factor in determining the removal of *E. coli* in natural environments.

The concentration of dissolved oxygen in ponds is controlled by the pond algae. In facultative ponds there is a diurnal variation in dissolved oxygen concentration at any depth above the oxypause and also in the position of the oxypause itself. The survival of *E. coli* is enhanced under anaerobic conditions (Klock 1971; Marais 1974). In thermally stratified facultative ponds in northeast Brazil, *E. coli* forms a reasonably stable layer some 10–20 centimeters below the oxygen-supersaturated algal zone, presumably to provide protection against the detrimental effect of very high dissolved oxygen concentrations (Pearson and Mara, unpublished data). However, Davis and Gloyna (1972) have shown that in Texas (USA) pretreatment in anaerobic ponds is advantageous in that the overall removal in a series of ponds which includes an anaerobic pond is greater than in one which does not. From the details given by these authors it is not possible to determine whether this is due merely to the anaerobic pond functioning as an additional unit in the series, or whether solids removal in anaerobic ponds permits an enhanced efficiency of whatever factors are responsible for fecal bacterial removal in facultative ponds. The results obtained by Mara and Silva (1979) in northeast Brazil suggest, however, that the latter explanation is not valid under all climatic conditions.

Loedolff (1965) examined the removal of *E. coli* through predation by microinvertebrates in experimental ponds in Pretoria (South Africa). Cladocera were found to be the numerically greatest group of microinvertebrates, with *Moina dubia* and *Daphnia magna*, respectively, predominating in facultative and maturation ponds. *In vitro* studies showed that *M. dubia* remove 93 cells of *E. coli* per individual per hour and *D. magna* 55, the difference being ascribed to the difference in coarseness of the filtering setae of the two species. At these rates of predation it was concluded that Cladocera do not contribute significantly to bacterial removal in ponds because, in practice, their numbers never rise to the level required for them to have a major effect on bacterial numbers.

KINETICS OF *E. COLI* REMOVAL IN PONDS. Removal kinetics have been studied in detail by only a few investigators. The most favored approach (for example, Marais 1974) is the assumption that removal of fecal bacteria follows first-order kinetics and that the pond is a completely mixed reactor.[3] Although this assumption undoubtedly represents a gross

3. The resulting kinetic equation is thus:

$$N_e = N_i/(1 + K_b t^*),$$

where $N_e$ and $N_i$ are the numbers of a fecal indicator bacterium (or bacterial pathogen) per 100 milliliters of pond effluent and influent, respectively; $K_b$ is the first-order rate constant for the removal of the bacterium in reciprocal days; and $t^*$ is the mean hydraulic retention time in the pond in days ( = the pond volume in cubic meters divided by the influent flow rate in cubic meters per day). A formal derivation of the equation is given, for example, by Mara (1976). A more rigorous approach to pond kinetics would be to use the Wehner and Wilhelm (1956) equation for first-order removal in dispersed flow reactors; such an approach is not normally possible, however, as pond dispersion numbers are usually unknown. For a fuller discussion of this point, see Mara (1976).

The value of $K_b$ is strongly temperature dependent and is usually described by an Arrhenius equation of the form:

$$K_{b(T)} = K_{b(20)}\theta^{T-20},$$

where $K_{b(T)}$ is the value of $K_b$ at $T$°C, $K_{b(20)}$ its value at 20°C, and $\theta$ the dimensionless Arrhenius constant. Marais (1974), using the results reported by Slanetz and others (1970), calculated values of 2.6 reciprocal days and 1.19 for $K_{b(20)}$ and $\theta$, respectively, for facultative and maturation ponds in the temperature range 5–20°C.

simplification of the environmental factors involved in fecal bacterial removal in ponds and also of the hydraulic regime therein, it is nonetheless empirically justified and in the past has served as a reasonable basis for design (Marais 1974; Mara 1976). However, a more recent and more rigorous analysis of removal of fecal bacteria in ponds is given by Dissanayake (1980), who studied the removal of fecal coliforms in laboratory-scale, pilot-scale, and full-scale ponds in Bangkok (Thailand). He found that the first-order rate constant for fecal coliform removal ($K_b$, in reciprocal days) was best described by the following multiple linear regression equation:

$$\exp(K_b) = 0.7716(1.0281)^T(1.0016)^{C_s}(0.9990)^{\lambda},$$

where $T$ is the temperature in degrees Celsius, $C_s$ the average concentration of algae in the pond in milligrams per liter,[4] and $\lambda$ the organic loading on the pond in kilograms of chemical oxygen demand per hectare per day. The intensity of ultraviolet radiation was shown to be an unimportant factor in influencing the value of $K_b$, and no account was taken of predation by microinvertebrates (which, as noted above, is insignificant). When used with the Wehner and Wilhelm (1956) model for first-order removal of fecal coliforms in dispersed flow reactors, this equation was found to be very satisfactory in predicting fecal coliform removal in full-scale ponds. Dissanayake (1980) also gives regression equations for predicting the value of $C_s$, so that his model for fecal coliform removal can be used by design engineers. Application of Dissanayake's model has of course been limited because of its recentness, and further work is required to determine the global applicability of its regression constants. Nonetheless, the model at least gives some idea of the relative importance of the principal environmental factors involved in removal of fecal bacteria in ponds.

Much of the large volume of literature in removal of fecal bacteria in ponds does not contain all the information required for a kinetic analysis of the results given therein; for example, many publications do not contain details of the retention time, and almost none gives information on the dispersion number or algal biomass of the ponds studied. The complexity of the removal of fecal bacteria in ponds is shown in table 13-3, which presents values of the first-order rate constant for the removal of various fecal bacteria in four series of ponds located in Australia, Brazil, and South Africa.[5] These series of ponds were selected because they are representative of well-operated ponds, with sufficient information given about their performance to permit the kinetic constants to be calculated. It is apparent from table 13-3 that there is considerable variation in the values of the kinetic constants for each bacterial group—even for ponds in the same series at the same temperature—and that little can be concluded about the relative removal rates of the different groups. Insufficient data were reported for this series of ponds to permit the validity of Dissanayake's (1980) model to be ascertained.

At present it appears, therefore, that design engineers have no alternative but to follow the design procedure based on the work of Marais (1974) and Mara (1976) for the removal of fecal bacteria in a series of ponds, even though its only environmental parameter is temperature. It is clear, however, that in the future pond design will have to include the effect of other variables such as algal biomass and organic loading. The pioneering approach shown by Dissanayake (1980) requires that it be followed by further work to determine its validity as a design tool.

### *By aerated lagoons*

The survival of fecal indicator bacteria in aerated lagoons has scarcely been studied. Menon and Bedford (1973) reported that coliform and fecal coliform removal rates were 38 and 63 percent, respectively, in an aerated lagoon treating wood pulp processing effluents at Fort Frances (Canada). The study was conducted during summer when the lagoon temperature was 28°C, but other process details were not given. There was evidence of coliform and fecal coliform growth on some occasions in the aerated lagoon, and laboratory studies showed that in sterilized aerated lagoon liquor total coliforms multiplied at 15°C and 28°C, that fecal coliforms multiplied at 28°C but not at 15°C, and that fecal streptococci died at both temperatures. This particular study was not well designed or reported, and it may be that the results are not typical. From a theoretical standpoint, an aerated lagoon (retention time 2–6 days) may be expected to have bacteria removal properties similar to, or a little better than, an oxidation ditch. If the effluent is treated

4. Rather than using algal dry weight in milligrams per liter, it is preferable to express algal biomass concentrations in ponds in terms of photosynthetic pigment; for example, micrograms of chlorophyll a per liter.

5. Since the dispersion numbers for these ponds were not given in the literature cited, it is only possible to analyze the results therein on the assumption of either complete mixing or plug flow. For the purpose of table 13-3, the former was used, although the latter would have served the argument equally well. The kinetic equation for a completely mixed pond is given in footnote 3, above.

Table 13-3. *Fecal bacteria removal rate constants in series of waste stabilization ponds*

| *Country reference* | *Temperature* (°C) | *Pond number* | *Retention time (days)* | *First-order rate constant (reciprocal days, base e) for removal of:* | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | *Fecal coliforms* | *Fecal streptococci* | Clostridium perfringens | *Salmonellae* | Pseudomonas aeruginosa |
| Australia (Parker 1962)[a] | 21 | 1 | 3.8 | 0.13 | 0.21 | | | |
| | | 2 | 8.0 | 1.13 | 1.30 | | | |
| | | 3 | 13.0 | 0.07 | 0.13 | | | |
| | | 4 | 18.0 | 0.12 | 0.61 | | | |
| | | 5 | 23.0 | 1.11 | 0 | | | |
| | | 6 | 28.5 | 0.55 | 0.91 | | | |
| | | 7 | 33.5 | 0.38 | 0.99 | | | |
| | | 8 | 38.5 | 0.19 | 0.23 | | | |
| Australia (Parker 1962)[a] | 9 | 1 | 4.1 | 0.11 | 0.16 | | | |
| | | 2 | 8.6 | 0.44 | 0.21 | | | |
| | | 3 | 14.0 | 0.23 | 0.17 | | | |
| | | 4 | 19.2 | 0.28 | 0.04 | | | |
| | | 5 | 24.6 | 0.14 | 1.01 | | | |
| | | 6 | 30.5 | 0.28 | 3.17 | | | |
| Brazil (Mara and Silva 1979)[b] | 26 | 1 | 6.75 | 2.17 | 3.69 | | | |
| | | 2 | 5.46 | 2.26 | 2.17 | | | |
| | | 3 | 5.46 | 2.40 | 2.38 | | | |
| | | 4 | 5.46 | 15.00 | 5.79 | | | |
| | | 5 | 5.79 | 1.91 | 0.12 | | | |
| South Africa (Coetzee and Fourie 1965)[c] | ND | 1 | 20.0 | 1.96 | | 2.44 | 0.55 | 4.19 |
| | | 2 | 15.0 | 8.47 | | 1.40 | 1.27 | 0.26 |
| South Africa (Coetzee and Fourie 1965)[d] | ND | 1 | 2.5 | 1.71 | | | 0.17 | 3.73 |
| | | 2 | 2.5 | 0.43 | | | 0.19 | 0.24 |
| | | 3 | 2.5 | 0.35 | | | 0.62 | 2.36 |
| | | 4 | 2.5 | 1.11 | | | 0.15 | 3.60 |

ND No data.

a. Data for full-scale ponds receiving raw sewage (anaerobic, facultative and six or four maturation ponds).

b. Data for pilot-scale ponds receiving raw sewage (anaerobic, facultative and three maturation ponds).

c. Data for full-scale ponds receiving raw sewage (facultative and maturation ponds).

d. Data for full-scale ponds receiving humus tank effluent (four maturation ponds).

in maturation ponds, removal as in waste stabilization ponds is anticipated. The sludge drawn off from secondary sedimentation tanks or settling ponds will be rich in excreted bacteria.

*By tertiary treatment*

The growing environmental concern in developed countries in recent years, coupled with some awareness that the effluents from conventional secondary processes (trickling filter and activated sludge) are heavily contaminated with excreted viruses and bacteria, has led to an increasing use of tertiary treatment. This section discusses the effect on fecal indicator bacteria of some of the tertiary processes that are being used to upgrade the chemical or microbiological quality of secondary effluents prior to discharge or agricultural reuse.

This section does not consider the advanced wastewater reclamation plants designed to transform sewage into drinking water. Such plants incorporate a complex combination of biological, physicochemical, and disinfection processes and can eliminate excreted bacteria completely. Grabow and Isaacson (1978) reported that the water produced by the sewage reclamation plants in Windhoek (Namibia) and Pretoria (South Africa) contained no fecal coliforms in 94 percent of samples, no fecal streptococci in 100 percent of samples, no *Pseudomonas aeruginosa* in 96 percent of samples, no *Staphylococcus aureus* in 91 percent of samples, and no *Clostridium perfringens* in 92 percent of samples. The Windhoek and Pretoria plants receive secondary effluents and treat them by lime treatment, primary coagulation and sedimentation, ammonia stripping, primary chlorination, recarbonation, secondary coagulation and sedimentation, pH adjustment, sand filtration, secondary chlorination, activated carbon, and final chlorination (Grabow, Bateman and Burger 1978). A treatment train of this complexity, although highly effective, has no application in most countries (whether developed or developing) because of its high cost and excessive operation and maintenance problems.

LAGOONING. If retention times are several days, lagooning can be highly effective in removing excreted bacteria. Removal mechanisms and rates are as reported above for waste stabilization ponds. With short retention times, lagooning will not achieve worthwhile reductions in concentrations of excreted bacteria. Grabow, Middendorff and Basson (1978) report a removal of only 31 percent of coliforms and 18 percent of enterococci from lime-treated tertiary effluent (pH 9.6) held in an aerated pond for a mean period of 10 hours in Pretoria (South Africa).

COAGULATION. Coagulation or flocculation of a secondary effluent, followed by solids removal in sedimentation or flotation chambers, will remove those bacteria which are bound up within, or adsorbed to, the flocs. This bacterial removal mechanism is analogous to that of activated sludge, although the retention times in the sedimentation tanks are considerably shorter and poor removal percentages may be expected. Coagulation is commonly promoted by the addition of alum [$Al_2(SO_4)_3$], iron salts (for example, $FeCl_3$), or polyelectrolytes.

High removal rates can be achieved by lime treatment followed by sedimentation. The lime treatment raises the pH to a level that is extremely hostile to many bacteria and viruses, although some Gram-positive bacteria (for example, fecal streptococci) and spore-forming bacteria (for example, *Clostridium* spp.) are comparatively resistant to high pH. Grabow, Middendorff and Basson (1978) studied the effect of lime treatment, plus sedimentation with added $FeCl_3$ (1.5–2.5 milligrams per liter as Fe) and polyelectrolyte (0.5 milligrams per liter), on enteric bacteria in activated sludge effluent. With a final pH of 9.6, 62 percent of coliforms and 68 percent of enterococci were removed. When the lime dose was raised to give a final pH of 11.1, the removals were 99.98 and 97 percent, respectively, for coliforms and enterococci.

FILTRATION. The data on bacterial retention and death in soil, reviewed elsewhere in this chapter (see above, the sections on groundwater, soil, and septic tanks and below, the section on land treatment), show that a well-designed sand filter, with a sufficiently deep bed and a sufficiently low filtration rate, will remove a considerable proportion of fecal indicator bacteria in the effluent. In particular, a slow sand filter, receiving 2–5 cubic meters per square meter per day of effluent, should remove around 99 percent of enteric bacteria. Rapid processes, however, may be relatively ineffective. A combination of ammonia stripping, recarbonation, secondary clarification (with added $FeCl_3$ and polyelectrolyte), and sand filtration reduced the average concentration of enterococci by only 98 percent (Grabow, Middendorff and Basson 1978). Similarly, a tertiary effluent from a gravity sand filter on Long Island (New York, USA) contained up to $9.3 \times 10^5$ fecal coliforms per 100 milliliters (Vaughn and others 1978). Removal rates will be enhanced at warm temperatures.

DISINFECTION. The realization that secondary effluents from conventional sewage treatment plants (trickling filters or activated sludge) contain high concentrations of excreted viruses and bacteria has caused a growing interest in effluent disinfection as a means of achieving a major improvement in microbiological quality prior to discharge. The technique most practiced and most studied is effluent chlorination.

As with water chlorination, the level of bacterial kill achieved in effluent chlorination increases as chlorine dose, temperature, and contact time increase and as pH decreases. The additional factor of critical importance is the chemical quality of the effluent being chlorinated. When chlorine is added to an effluent, free chlorine ($HOCl + OCl^-$) disappears almost immediately, and the chlorine rapidly combines with ammonia and organic compounds. This combined chlorine is very considerably less bactericidal than free chlorine—although, as with free chlorine, it will be more effective as contact times and temperatures increase.

When a chlorinated effluent is discharged into a river, it is possible that the fecal indicator bacteria in the river will also be reduced in the stretch of river immediately downstream of the outfall (Snow 1977). The chlorine may also have a negative impact on the receiving water by killing certain species of flora and fauna and thus upsetting the aquatic ecology (Silvey, Abshire and Nunez 1974). In addition, the discharge of chlorinated effluents will add to the load of chlorinated organic compounds in the water and in the food chain, and some of these compounds are known or suspected carcinogens.

Properly controlled effluent chlorination can produce a very dramatic reduction in the concentrations of excreted bacteria. The better the quality of the effluent, the greater will be the bacterial reduction achieved by a given dose of applied chlorine. Berg and others (1978) added sodium hypochlorite (NaOCl) to primary effluents to achieve final combined chlorine residuals of 11 to 23 milligrams after 15 minutes at pH 8.2–9.2 and 22°C. This treatment reduced enterovirus (initial concentrations 109–427 per liter) by 85–99 percent, fecal coliforms (initial concentrations $10^4$–$10^7$ per 100 milliliters) by 99.95–>99.99998 percent, and fecal streptococci (initial concentration $10^5$ per 100 milliliters) by 99.997–>99.9998 percent (see also Berg and Metcalf 1978).

Kott and others (1974) reported that the addition of 8 milligrams per liter of chlorine to waste stabilization pond effluent (1 hour contact time at 20°C) reduced the concentration of coliforms from $10^5$–$10^7$ per 100 milliliters down to less than 2 per 100 milliliters in 50 percent of tests. Stenquist and others (1977) found that treating a secondary effluent ($BOD_5$ = 19 milligrams per liter) with 39 milligrams per liter of chlorine reduced coliform concentrations from $10^6$ to <2 per 100 milliliters after a 1-hour contact time.

Although effluent chlorination can produce very low concentrations of indicator bacteria at the point of discharge, it does not always do so. Vaughn and others (1978) reported that chlorinated secondary effluents on Lond Island (New York, USA) contained between 0 and $2.4 \times 10^6$ fecal coliforms per 100 milliliters. Kampelmacher, Fonds and van Noorle Jansen (1977) found that the chlorination of three secondary effluents in the Netherlands (2–6 milligrams per liter of chlorine added) reduced *E. coli* by between 24 and 99.999 percent and fecal streptococci by 0 to 99.99 percent. This great variability of bacteria removal is characteristic of effluent chlorination systems and in part is a result of the variable chlorine demand of the effluent.

The environment produced by effluent chlorination, namely one of high nutrients but low microbiological activity, is ideal for the growth of some excreted bacteria. Several studies have reported massive regrowth of fecal indicator bacteria, especially coliforms, in chlorinated sewage effluents. Shuval, Cohen and Kolodney (1973) studied trickling filter effluent in Jerusalem (Israel) that was treated with 10 milligrams per liter of chlorine and then stored for 3 days prior to agricultural use. The geometric mean concentrations of coliforms per 100 milliliters were $5 \times 10^6$ in the secondary effluent, 120 in the chlorinated effluent, and 800 in the stored effluent. Laboratory experiments with 5 milligrams per liter of chlorine added to secondary effluent stored at 20°C showed that coliform concentrations fell to 0.001 percent of their initial value after chlorination but 4 days later had regained their initial level, whereas fecal coliform concentrations fell to 0.0001 percent of initial levels and had climbed back to 0.1 percent after 5 days.

Kinney, Drummond and Hanes (1978) compared bacterial death rates in chlorinated secondary effluent mixed with stream water and in unchlorinated tertiary effluent mixed with stream water (three parts effluent to one part of stream water). The effluent had a $BOD_5$ value of 7–11 milligrams per liter. The applied chlorine dose was 2.3–3.5 milligrams per liter, which produced a total residual of around 1 milligram per liter after 15 minutes. The mixtures were held in the dark at 20°C. The chlorinated mixture contained 6 *E. coli* per 100 milliliters, which rose to $10^2$ per 100 milliliters after 5 days. The unchlorinated mixture contained $1.9 \times 10^3$–$2.2 \times 10^4$ *E. coli* per 100 milliliters, which fell to $10^2$ per 100 milliliters after 5 days. Similar

experiments were conducted with total coliforms, *Klebsiella*, *Enterobacter*, and *Citrobacter*. In all cases there was no statistically significant difference in bacterial concentrations between the chlorinated and unchlorinated mixtures after 4 days of storage.

Irving (1980) reported great variability in the bacterial reductions obtained when chlorine was added to raw sewage. Average results showed that, after a contact time of 30 minutes, the coliform reductions were 3 log units at 5 milligrams per liter of applied chlorine, 4 log units at 10 milligrams per liter of applied chlorine, and $>5$ log units at 15 milligrams per liter of applied chlorine. To investigate regrowth, samples of chlorinated sewage were added to seawater and stored in the dark at 15°C. When the effluent to seawater mix was 1:10, coliform concentrations increased in the chlorinated mixture to more than the initial (pre-chlorination) level after 160 hours' storage, whereas the unchlorinated control had concentrations of only 10 percent of initial values. When the effluent to seawater mix was 1:100, no regrowth occurred and the chlorinated mixture had coliform concentrations of 0.001 percent of initial levels after 160 hours' storage, whereas the unchlorinated control had concentrations of 1 percent of initial values. The ratio of effluent to seawater, and thus the concentration of bacterial nutrients, was found to influence strongly the coliform regrowth potential. When actual marine outfall conditions were simulated (ten-fold dilution of sewage initially rising to a hundredfold dilution after 3.5 hours), no regrowth of coliforms or fecal coliforms was recorded in the chlorinated effluent and seawater mixture.

Fecal coliforms are more susceptible to chlorination, and undergo lesser regrowth in chlorinated effluents, than coliforms as a whole. Fecal streptococci may be slightly less susceptible to chlorination of effluents than fecal coliforms. Major regrowth of fecal indicator bacteria in chlorinated effluents and their receiving waters is more likely in fresh water than in seawater, where dilution is low (less than ten-fold) and where temperatures are warm. Thus, the discharge of chlorinated effluents into tropical streams, which have little or no flow during the dry season, is a situation almost certainly accompanied by major regrowth of coliforms, fecal coliforms, and possibly other excreted bacteria.

Chlorine dioxide ($ClO_2$) has attracted interest recently as a disinfectant of water and wastewater, although its mode of action is not elucidated (Roller, Olivieri and Kawata 1980). Longley, Moore and Sorber (1980) compared chlorine and chlorine dioxide applied to secondary effluent at doses of 5 milligrams per liter (as $Cl_2$) with a contact time of 3 minutes at 22°C. Reduction of fecal coliforms averaged over 5 log units with chlorine dioxide and over 3 log units with chlorine. This and other studies (for instance, Cronier, Scarpino and Zink 1978) have shown that chlorine dioxide is often a more powerful bactericide than chlorine and has other advantages such as being less affected by pH and less prone to generate carcinogenic trihalogenated methanes. Similarly, Keswick and others (1980) found that bromine chloride (BrCl) had several advantages over chlorine as a wastewater disinfectant.

Various other disinfecting methods have been tried, at least on an experimental basis. Ozone is able to eliminate fecal solids-associated coliforms after 20 seconds with an applied dose of less than 0.1 milligram per liter (Foster and others 1980; see also Wyatt and Wilson 1980). Ultraviolet and gamma radiation have also been shown to be effective in killing bacteria in effluent (Myhrstad 1979; Woodbridge and Cooper 1979). These techniques are very much at the experimental stage, and their economical and technical appropriateness are doubtful. The same comments apply to disinfection by photodynamic oxidation (Gerba, Wallis and Melnick 1977).

LAND TREATMENT. The treatment of a primary or secondary effluent by application to land, with subsequent flow through the soil to underdrains or to groundwater, can be an effective method of removing high concentrations of excreted viruses (see chapter 9) and bacteria. Lance, Rice and Gilbert (1980) reported a 5 log removal of fecal coliforms from primary effluent (settled sewage) as it passed through 2.5 meters of loamy sand at the rate of 0.2 cubic meters per square meter per day and at a temperature of 24°C. Under similar conditions, but with double the infiltration rate, there was a 3 log removal of fecal coliforms from secondary effluents. A theoretical approach to computing the degree of bacteria removal by land treatment systems has been proposed by Hendricks, Post and Khairnar (1979).

Gilbert and others (1976) recorded that the fecal coliforms and fecal streptococci in a secondary effluent were reduced by 99.9 percent after flow through 9 meters of soil (fine loamy sand underlain by coarse sand and gravel) under groundwater recharge basins at Phoenix (Arizona, USA). The application rates average 0.25 cubic meters per square meter per day, and the mean daily air temperatures ranged between 10°C in January and 32°C in July. Fecal coliforms were not detected in water from a well 90 meters distant from the recharge basins (Bouwer and others 1980). Schaub

and Sorber (1977) studied a rapid infiltration site (1.4 cubic meters per square meter per day) in Massachusetts (USA) where primary effluent had been applied to unconsolidated silty sand and gravel for over 30 years. Nearly all indicator bacteria were removed in the top 1 meter of soil, but, on occasion, up to $10^4$ fecal streptococci per 100 milliliters were detected in the groundwater, as compared with $10^5$–$10^6$ per 100 milliliters of effluent.

Land treatment will in general be more effective in warm climates than in temperate ones. Maintenance and management of these installations must be highly efficient, otherwise, the treatment site will degenerate into an unsanitary bog.

OTHER PROCESSES. A variety of other processes for the treatment of sewage are being tried on a laboratory or pilot scale. Many of these research and development projects focus on the need to produce a final effluent almost completely free of excreted organisms and attempt to reclaim sewage for agricultural or other productive purposes. Some of the technologies being developed have high performance in removing fecal indicator bacteria—for instance, solar distillation (Qasim 1978)—but are unlikely to be economically and technically appropriate for application in developing countries. Technologies that are simple, relatively low cost, and highly effective in removing excreted bacteria are already known (for instance, waste stabilization ponds or land treatment of secondary effluents), and the priority in most developing countries is the successful and widespread application of these established technologies.

## Inactivation by Night Soil and Sludge Treatment Processes

Since fresh feces commonly contain $10^5$–$10^9$ fecal coliforms and fecal streptococci per gram, the fecal products from dry sanitation systems contain high concentrations of fecal indicator bacteria. It is this very concentration of excreted organisms that makes the dry systems attractive compared with the wet systems, which mix the fecal material with large volumes of water that are then difficult to purify and contain. From a microbiological viewpoint the essential principle of the dry sanitation systems is that they should concentrate and contain the excreted organisms in the smallest possible volume where they may then be killed. The sludge from a sewage treatment plant contains those bacteria which were adsorbed to, or trapped within, settleable solids, and high concentrations of fecal indicator bacteria are typically found ($10^5$–$10^8$ per gram). This section describes methods of killing bacteria contained in night soil or sludge and emphasizes the important role of time and temperature in creating conditions lethal to bacteria.

### *By pit latrines*

Pit latrines are an effective method of containing and storing excreted bacteria. Death rates will be very much more rapid at warm temperatures than at cold temperatures and may be more rapid in dry pits than in flooded pits. A pit in use will always contain fresh layers of pathogenic material. The contents of a sealed pit in a warm climate, however, should contain a very low concentration of fecal indicator bacteria after 3 months' storage.

Stiles and Crohurst (1923) buried human feces in pits in an area with a high water table and covered them with sawdust. Three years and 2 months later, the feces were both macroscopically, and, microscopically recognizable but had developed a "musty" odor. Three out of five samples contained viable *E. coli.* Jordan (1926) found that *E. coli* in stored human feces became undetectable after 6–12 weeks at room temperature and after 1–3 weeks at 37°C. At 10°C they were still detectable in high concentrations after 23 weeks.

### *By anaerobic digestion*

Anaerobic digestion is the most commonly adopted method of sludge treatment at large sewage treatment works. The process is usually mesophilic (35°C) but is sometimes heated to the thermophilic range (55°C). Retention times are typically 10–60 days.

Cooke, Thackston and Malaney (1978) studied mesophilic anaerobic digestors at three treatment plants near Nashville (Tennessee, USA). Concentrations of coliforms in the raw primary sludge were $10^8$–$10^{10}$ per 100 milliliters and in the digested sludge were $10^4$–$10^9$ per 100 milliliters. All digesters were operating at 33–37°C, but retention times varied considerably. The mean removal rates at different retention times were less than 2 log units after 9 days, 3 log units, after 38 days, and nearly 4 log units after 50 days. Berg and Metcalf (1978) reported that a continuously operated mesophilic digester (35°C for 20 days) removed 95 to 99 percent of fecal coliforms and 80 to 97 percent of fecal streptococci, whereas a thermophilic digester (50°C for 20 days) removed 99.9 to >99.9999 percent of fecal coliforms and 99.8 to >99.999 percent of fecal streptococci. Studies in the USSR showed that an enteropathogenic serotype of *E.*

*coli* survived thermophilic digestion for not more than 10 days (Grigoryeva, Korchak and Bey 1969).

Continuously fed mesophilic digesters will produce sludge that still contains high concentrations of fecal indicator bacteria ($10^3$–$10^6$ per 100 milliliters), even if retention times are around 50 days. For substantially higher levels of bacterial reduction the process should be thermophilic, with retention times of at least 20 days and preferably with batch operation. Thermophilic operation is more economical in warm climates than in temperate areas because heat loss to the environment is reduced; therefore less energy input is required (from digester biogas or other sources). However, the most economical method of achieving thermophilic conditions, and thereby high levels of excreted bacterial reduction, is often by aerobic composting. Finstein and others (1980) compared anaerobic digestion with composting and found that, although digestion required less labor and produced methane, composting was advantageous with respect to pathogen kill, process stability, decomposition of industrial compounds, drying, and residue acceptability.

*By heating*

Any process that heats night soil or sludge to over 60°C for the required length of time will eliminate fecal indicator bacteria. Most of the heating processes—such as pasteurization (80°C), wet oxidation (120–370°C), incineration, and pyrolysis—will completely destroy all excreted organisms but are technically complex and require considerable energy input. More attractive is thermophilic, aerobic composting, which elevates temperatures to above 60°C without the need for any external energy source. Time-temperature combinations that will destroy enteroviruses (see chapter 9) and *Ascaris* eggs (see chapter 23) will certainly destroy the nonsporulating excreted bacteria.

*By composting*

The composting of night soil and sludge is a simple means of producing a fecal product that is safe and convenient to use in agriculture. Of greatest interest are the aerobic processes that generate sufficient heat to raise the temperature of the composting mass to 55°C or above and thus kill a large proportion of excreted pathogens.

Savage, Chase and MacMillan (1973) studied the composting of pig waste (uneaten garbage and pig feces) in four windrows, which were mechanically turned. Windrow 1 contained pig waste turned twice per week; windrow 2 contained pig waste turned 20 times per week; windrow 3 contained pig waste plus old compost turned 20 times per week; windrow 4 contained pig waste plus straw turned 20 times per week. The speed of temperature rise declined in the order of windrows 4, 3, 2, 1. All windrows eventually reached 60°C. Windrow 4 reached 60°C in 3 days, after which it rose to 72°C, stayed above 60°C for over 30 days, and fell back to ambient temperature (20–30°C) by day 38. In windrow 1, fecal coliforms and fecal streptococci numbers increased for the first 40 to 60 days until temperatures reached 50°C, after which bacterial concentrations decreased as the temperatures rose into the thermophilic range. In windrows 2 and 3, the concentrations of fecal coliforms fell from $10^7$ to $10^3$ per gram after 35 days, whereas in windrow 4 fecal coliforms became undetectable after 14 days. The combination of frequent turning and the addition of straw (which provided a source of carbon and improved the structure of the compost by furnishing more opportunity for aeration) produced a windrow that achieved very high bacterial death rates (7 log units in 14 days).

Burge, Cramer and Epstein (1978) have reviewed the composting work conducted at Beltsville Agricultural Research Center (Maryland, USA). In early experiments raw or digested sludge filter cake (20 per cent solids) was mixed with woodchips (1 volume sludge to 3 volumes of woodchips), placed in windrows, and turned daily for 2 weeks, after which it was stockpiled for a minimum of 30 days. Temperatures in the windrows rose to 50–70°C in 3 days with raw sludge, and to 40–60° in 14 days with digested sludge, except in rainy winter weather when temperatures stayed at 20–30°C. Fecal coliforms typically declined from $10^7$ per gram to undetectable levels during the windrow phase at depths of 0.8 meters and more. At depths of less than 0.4 meters, where the windrow was cooler, fecal coliform concentrations declined from $10^7$ to $10^2$–$10^4$ per gram during the windrow phase and maintained these concentrations during 30 days of stockpiling. Because of lowered temperature during heavy rain, poor bacterial kill at the edges of the windrows, and bad odor release from the raw sludge windrows, new experiments were started in which windrows (raw sludge plus woodchips) were aerated (by sucking air downwards into slotted pipes laid under the windrow) and lagged by covering with a layer of old compost. Composting by forced aeration was continued for 21 days, after which the material was stockpiled for a minimum of 30 days. Temperatures rose into the thermophilic range within 5 days, irrespective of the weather conditions. Fecal coliforms

were reduced to undetectable levels after 10 days in the windrows, and this bacterial destruction occurred at all parts of the composting mass (see also Kawata, Cramer and Burge 1977).

Unpublished data obtained by Vietnamese scientists have suggested that *E. coli* are reduced to undetectable levels after 7 weeks treatment in a sealed vault of a double-vault composting latrine (Nimpuno, personal communication). McGarry and Stainforth (1978) describe aerobic (average pile temperature 40°C) and anaerobic (average pile temperature 29°C) composting with equal parts by weight of human feces and urine, animal feces, rubbish (sweepings, brushwood, grass, ashes, weeds, leaves), and soil held for one month in China. Initial *E. coli* concentrations of the mixture were $2.5 \times 10^5$ per gram, and these were reduced to 91–233 per gram in both the aerobic and anaerobic processes.

It is clear from these and other studies (listed in the appendixes of Feachem and others 1980) that well-managed composting plants can reduce fecal indicator bacteria in night soil and sludge to undetectable levels in under 1 month. For this to occur, the process must become thermophilic rapidly. This in turn requires the addition of carbon, aeration by turning or forced ventilation, moisture control, and a good physical structure with adequate air voids for the pile. To ensure that all parts of the pile achieve a disinfecting temperature, the compost must be turned or lagged. The experiments at Beltsville have shown that forced ventilation and lagging can create high temperatures throughout a windrow even during cold and rainy winters.

*By lime treatment*

Lime treatment, resulting in high pH values, should be effective in reducing fecal indicator bacteria in night soil and sludge, although little specific information is available. Lime treatment is certainly effective in eliminating salmonellae from sludges and animal slurries (see chapter 15). Polprasert and Valencia (1981) applied various concentrations of lime to fecal samples collected from school children in Bangkok (Thailand). Initial pH was 5.8–6.0, and the fecal coliform concentrations were $4.6 \times 10^{10}$ to $1 \times 10^{11}$ per milliliter. The addition of 5,700 milligrams of CaO per liter raised the pH to 9 and reduced the fecal coliform concentration by 0–1 log units. The addition of 19,000 milligrams of CaO per liter raised the pH to 12 and reduced the fecal coliform concentration by 4–6 log units. Intermediate doses produced intermediate pH values and bacterial reductions. Fecal coliform death occurred in the first 3 hours after lime dosing, and no significant additional dieoff occurred during 2 days storage at 25°C.

*By other processes*

Various other methods have been proposed for the disinfection of sludges prior to agricultural use. Irradiation of sludges by 3–3.5 kilogray produces a 4 to 9 log reduction in Gram-negative enteric bacteria such as fecal coliforms. Gram-positive bacteria such as fecal streptococci appear to be somewhat more resistant and may be reduced by only 2 to 3 log units (Lessel and Suess 1978; Osborn and Hattingh 1978). A radiated sludge, however, is a nutrient-rich but biologically impoverished medium, and major regrowth of fecal indicator bacteria may be predicted and has been reported (Osborn and Hattingh 1978). Superchlorination of sludges (chlorine doses of 700–4,000 milligrams per liter applied under pressure) will certainly destroy fecal bacteria, but it has to be questioned seriously on environmental grounds (Kamlet 1979).

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# 14

# *Leptospira* and Leptospirosis

LEPTOSPIRAS are quite distinct from the other bacteria discussed in chapters 12 through 18 in that they are not normally transmitted from person to person. They infect rodents and other animals and occasionally infect man when he comes into contact with infected animal urine. Leptospiras are included here because sewer workers are exceptionally exposed to the risk of leptospirosis.

## Description of Pathogen and Disease

Leptospirosis can be a severe illness, but rarely is it sufficiently common to constitute a major public health problem. Good accounts of leptospirosis have been published by Alston and Broom (1958) and Turner (1967, 1968, and 1970).

### *Identification*

Leptospirosis is an infection with bacteria of the genus *Leptospira* that may take several forms. One of these forms is Weil's syndrome, which develops in about 40 percent of cases of infection by the more virulent serotypes, notably *L. icterohaemorrhagiae*.[1] It is a severe illness with jaundice, neck stiffness, hepatomegaly, sometimes splenomegaly, hemorrhages in the eyes and skin, albuminuria, and hematuria. It has a high fatality rate unless diagnosed and treated in the early stages. However, milder forms of illness can also result from infection by *L. icterohaemorrhagiae* as well as by the many other serotypes (over 100) that are pathogenic to man. In such cases the disease cannot be diagnosed clinically with accuracy because of its protean nature. Its signs and symptoms may mimic many other illnesses such as influenza, septic meningitis, Q-fever, enteric-like illnesses, glandular fever, and brucellosis. In tropical countries it has to be differentiated from malaria, scrub-typhus, dengue, and sand fly fevers. Such illnesses can only be diagnosed as leptospirosis by laboratory tests—isolation of the organisms in blood culture and serological tests that show a rise in the specific antibody levels.

1. See the subsection "Infectious agent," below, for a note on taxonomic nomenclature.

### *Occurrence*

Leptospiras exist in most countries of the world, but whereas certain serotypes (for example, *icterohaemorrhagiae* and *canicola*) are widespread, others tend to be restricted only to certain regions, where their natural (reservoir) hosts are found. In most tropical areas multiple serotypes are circulating.

Although it appears that the incidence of human leptospirosis is low compared with that of other infectious diseases and that cases tend to be sporadic rather than epidemic, it is likely that in certain parts of the world many cases are being missed because they are misdiagnosed and leptospirosis is not being looked for.

At certain times and under certain circumstances, leptospirosis may become a major problem, as it was among the British forces stationed in the jungles and swamps of Malaya and Burma, and as it still is among rice field and sugarcane plantation workers.

### *Infectious agent*

Bacteria of the genus *Leptospira* are essentially parasites of animals, notably rodents—rats, mice, voles, and the like. Leptospiras are slender, flexuous, spiral organisms that are actively motile by means of internal axial filaments analogous to the external flagella of Gram-negative bacilli. They are approximately 0.1 micrometers in width and 6–20 micrometers in length, with bent or hooked ends (see figure 14-1). Some of them are free-living, nonpathogenic, common in fresh water; others are parasites of animals and potential pathogens of man. Leptospiras are

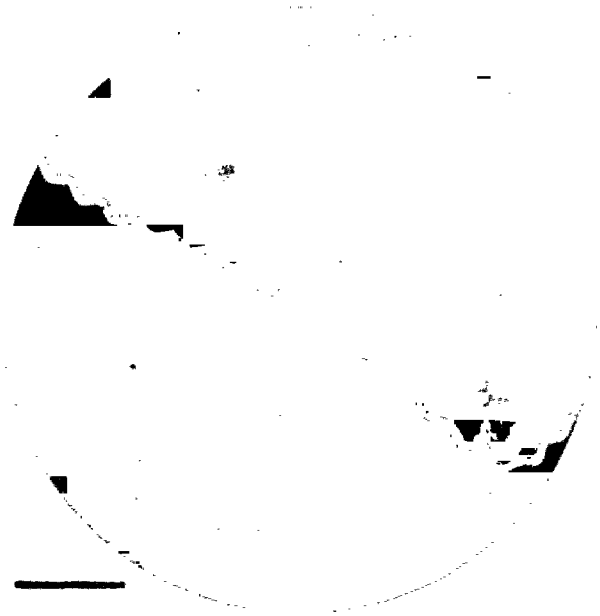

Figure 14-1. Leptospira *under scanning electronmicroscopy*. The organism is spiral and has bent or hooked ends. Scale bar = 1 micrometer. (Photo: J. D. Fulton and D. F. Spooner, National Institute for Medical Research, London, UK)

strictly aerobic. They require animal serum or a serum derivative for growth. The optimum temperature for growth is 28–30°C.

Approximately 150 serotypes of pathogenic leptospiras are recognized. Before 1967 these serotypes were considered as separate species. Recently the antigenically distinct types have been classified as serovarieties (or serotypes) of a single species, *Leptospira interrogans*. For example, an organism referred to as *L. canicola* in the older literature would now be called *L. interrogans* serovar (serotype) *canicola*. Free-living saprophytic leptospiras are now classified as serovars (serotypes) of the species *L. biflexa* and are commonly isolated from surface waters, sediments, and soils (Henry and Johnson 1978).

The first pathogenic serotype to be isolated from human leptospirosis was named *icterohaemorrhagiae*. It is one of the more virulent of the leptospiral serotypes and is the main cause (but not the only cause) of the severe form of the disease, Weil's syndrome. Many different serotypes pathogenic to man and animals have since been reported.

### *Reservoirs*

Each serotype of *Leptospira* has its own preferred host (mainly rodents) in which it exists as a commensal bacterium without causing any apparent harmful effects. It colonizes the kidney tubules, from which it is periodically washed out in the urine.

The rat, *Rattus norvegicus* (brown rat, sewer rat), is the reservoir host of the *icterohaemorrhagiae* serotype, and any environment where these rats are found is a potential hazard to the men who may work there, should they handle material contaminated with rat urine. Persons who have been infected with *icterohaemorrhagiae* include fish workers, butchers, refuse collectors, bricklayers, dock laborers, factory workers, miners, sewer workers, and many others. Any environment that encourages the proliferation of rats is potentially a source of infection with *icterohaemorrhagiae*. Domestic animals may also be infected by this serotype, and they transmit the infection to each other and to the people who look after them.

### *Transmission*

Provided that the external conditions are favorable (moist, relatively warm, shaded from the ultraviolet light of the sun, not salty, and of a neutral pH), leptospiras can remain viable for a time outside the animal body and are then transmissible to other hosts, including man or his domestic animals. The organisms gain access to the body through its contact with the urine-contaminated environment by way of cuts and abrasions of the skin or by way of the mucous membranes of the nose and mouth during immersion in water containing leptospiras. If the new, accidentally infected host is a human being or an animal of a different species than the reservoir host, a disease state may result. In the convalescent stage of the infection, man and these animals may become urinary excretors of leptospiras although, unlike the reservoir hosts, they tend to be temporary rather than chronic shedders. Dogs, cattle (including milking cows), pigs, and horses have all been incriminated in the spread of the disease via their urine. Man-to-man transmission is rare and usually limited to direct contact (for example, by venereal or transplacental routes), but an exceptional case from Vietnam has been reported (Spinu and others 1963) and is discussed below.

Although it is not possible to determine the infective dose of virulent leptospiras in man, experiments on guinea pigs have indicated that the *icterohaemorrhagiae* serotypes (derived directly from an infected guinea pig) may have a lethal dose, and consequently an infective dose, as low as 1 organism.

### *Incubation period*

Symptoms develop after a period ranging from 2 to 14 days, with an average of 7–8 days.

*Period of communicability*

The length of time a patient may be considered to be capable of communicating leptospiras to other individuals within his environment depends on the time he carries the organism in the kidney tubules. Unlike some animal hosts that may remain carriers for the whole of their life span, humans rarely shed leptospiras in their urine for longer than 4–6 weeks from the onset of infection (see, for instance, Ido and others 1917).

*Resistance*

All known vertebrate animals are susceptible to leptospiral infection, and there is no known example of natural resistance. Acquired immunity may help to control the incidence of clinical disease in communities living and working in close proximity to sources of infection. This applies especially to rural communities.

*Epidemiology*

Many of the accounts of leptospirosis record outbreaks of infection among groups of workers in developed countries who have been occupationally exposed to animal urine. For instance, Fairley (1934) made the first report of a fatal case of leptospiral jaundice in a sewer worker in Britain. The patient had been working in a London sewer for only 3 weeks before becoming infected. A serological investigation revealed that eight other sewer workers who had suffered from jaundice had also been infected by the same leptospiral serotype (*icterohaemorrhagiae*), and five other cases of leptospiral jaundice in London sewer workers were subsequently diagnosed. Alston (1935) described three of them. The first was a man, age 52, employed intermittently for 5 years as a flusher; the second was a man 35 years old who had been employed for 3 years, also as a flusher; the third, a fatal case, was a man 52 years old employed relaying sewers. He worked in old open trenches and in sewers that contained much slime and silt. He cut a finger on a broken drain pipe. The wound was dressed, and he went on working until his illness occurred about 2 weeks later. An investigation into the length of service of the men involved in these cases showed that it was not only recent recruits who were liable to infection. It occurred in men who had worked in sewers for a few months to 5 years or more. Infection occurred among those who worked as building laborers as well as among sewer flushers. Laborers who chiseled out and handled slime-covered brickwork in sewers were in direct contact with leptospiras subsequently shown to be present in the slime. These men frequently sustained abrasions and cuts on the hands. The flushers were less likely to sustain abrasions of the skin. They may, however, have become infected through the mucous membranes of the alimentary or respiratory tract by touching the mouth and nose during work. Virulent strains of *icterohaemorrhagiae* were isolated from sewer rats, caught in sewers in eight different parts of London, and also from slime taken from the floor of the sewer near where the first patient (the fatal case) had been working and from the outlet of a house drain into another sewer. The isolation of virulent leptospiras from sewers recalls the isolation of *icterohaemorrhagiae* serotype from slime in a coal mine recorded in 1927 (Buchanan 1927). Johnson, Brown and Derrick (1937) recorded the first three cases of classical Weil's disease in Australia.

Cabelli (1978) stated that leptospirosis has been reported with increasing frequency in the USA over the past five decades and that many cases are no longer related to occupational exposure but to contact with soil or water contaminated by urine. Swimming or wading in small ponds or creeks, recently used by cattle or receiving run-off from nearby pastures, is a common setting for infection. In 1975, 119 cases of leptospirosis were reported in the USA and 36 (29 percent) were attributed to contact with water containing cattle urine. Diesch and McCullouch (1966) isolated serotype *pomona* from bathing water implicated in an outbreak of 15 human cases of *pomona* leptospirosis in Iowa (USA) in 1964; these authors induced leptospiral infections by inoculating guinea pigs with the suspect water (see also Gillespie and Ryno 1963).

In 1977, 86 cases of leptospirosis were reported in the USA, of which 69 percent were males, and there were four deaths. The most probable sources of infection were livestock, domestic pets, and contaminated water. Only 26 percent of cases could be linked to particular vocational activities; of these, farmers were at the greatest risk (Centers for Disease Control 1979). The association between leptospirosis and farming in developed countries is further highlighted by the fact that 42 percent of leptospirosis cases in the UK in 1979–80 were farmers (Coghlan 1981).

Detailed accounts of leptospirosis epidemiology in developing countries are few. The pattern of infection normally depends upon the interaction between man and infected animals in a particular environment. Willis and Wannan (1966) found leptospiral antibodies in the blood of between 31 and 79 percent of people in six villages in different parts of Papua New Guinea. Antibody prevalence rose with age and was similar in males and females. Antibodies, to the same leptospiral

serogroups as were found in humans, were also found in rats, dogs, and pigs. The authors noted that in Papua New Guinea the daily pattern of village life brings most individuals into close contact with these animals. Thus, high antibody prevalence is not associated with a particular sex or occupation. By contrast, Damude and others (1979) found that 43 percent of sanitation workers and 39 percent of sugarcane workers in Barbados had antibodies to leptospiras and that this antibody prevalence was significantly higher than that for all other occupations studied. Further, 74 percent of the 215 reported cases of human leptospirosis occurring on Barbados during 1968–74 were males. Caldas and Sampaio (1979) reported on leptospirosis in Salvador (Bahia, Brazil) during 1975. The highest incidence occurred during the rainy season, especially April and May, and possible sources of infection were sewage, rats, water, dogs, mud, and garbage—in that order of frequency.

The operation of foreign armies in tropical areas has stimulated important research on a number of tropical infections for over a century. The study of leptospirosis in Malaysia during the 1950s and 1960s is a relatively recent case in point (Alexander and others 1975; Baker 1965; Baker and Baker 1970; Gordon Smith and others 1961; McCrumb and others 1957). About 40 percent of nonmalarial fever among foreign troops was due to leptospirosis. Infection was strongly associated with jungle maneuvers, and an average case rate of about 1 per battalion per day of jungle duty was reported. The primary rain forests of Malaysia were found to be a hyperendemic focus of leptospirosis. Infection of wild and domestic animals was common, and rats were implicated as the reservoir of greatest importance in the epidemiology of human infection. Pathogenic leptospiras (serotypes of *L. interrogans*) were readily isolated from stagnant and swamp waters in the jungle and also from streams, especially following periods of rain; soil near the stream banks, presumably contaminated by animal urine, frequently contained pathogenic leptospiras, and it was thought that these were being washed into the streams during rain.

A report from Vietnam (Spinu and others 1963) tells a different and unusual story. In Vietnam, as elsewhere, cases of human leptospirosis tend to occur sporadically, usually on the plains among workers in the flooded rice fields, where there is a great diversity of rodent species as well as domestic animals to act as the reservoir hosts for the various serovars that are responsible for human infection. During 1959, however, an epidemic amounting to 121 cases occurred among 240 soldiers working in the jungle territory of northeast Vietnam. Two groups of 80 soldiers were employed in two different forest sites, cutting down trees and bringing them down to the beds of several streams, where a third group of 80 men with a team of buffaloes had the task of dragging the logs downstream and across marshland to the place where the logs were stacked before being loaded onto lorries for further transport. Cases of leptospirosis occurred among soldiers in this third group and among men from the other two teams who were drafted to do the same work. No cases developed among the soldiers felling trees. The pH of the mud and water was 7–7.2, and the atmospheric shade temperature 22–26°C—conditions favorable to the survival of leptospiras. Leptospiras inoculated into samples of water taken from the streams remained viable in the laboratory for 2–5 days. There was no evidence that either the buffaloes or rodents in the area were carriers of the infecting strains of *Leptospira*. The inability of the investigators to find an animal reservoir of infection led to the conclusion that the epidemic had been spread by person-to-person transmission. Soldiers urinated directly onto the waterlogged track down which the logs were dragged and around the loading platforms where the ground was equally boggy. The soldiers, clad only in shorts, worked barefoot and were liable to sustain scratches and abrasions of the skin, and these small wounds allowed the ready entry of leptospiras into the body. The prolonged period of leptospiruria (up to 97 days) shown by some of the patients was thought to be due to the near normal pH of the urine (pH 6.2–7.2), a result of their predominantly vegetarian diet.

## Control Measures

Both individual and environmental approaches to leptospirosis control are employed, although in general environmental approaches are more effective.

### *Individual*

Persons who are known to have been at risk through contact with material contaminated with the urine of infected animals should be given a course of penicillin, 2 mega-units per day for 5 days, administered intramuscularly. No special prophylactic drugs are available for leptospirosis. Avoidance of contact with any material containing animal urine (especially rat urine) will greatly reduce the risk of infection. Cuts and abrasions should be covered and protective clothing worn.

In high-risk occupations, vaccination has proved to be effective. It is necessary to prepare the vaccine from

strains prevalent in the area, since the level of cross-protection afforded by one serotype may not be adequate for another. Undesirable side effects have been reported, but these have been minimized by the use of vaccines prepared by methods similar to that of Babudieri (1962).

### *Environmental*

To avoid direct transmission of leptospiras contained in animal urine, working premises should be made rat-proof: refuse and food waste should not be left lying around to attract rodents, dogs, foxes, and other scavengers. Chlorination of water supplies will prevent transmission by this route.

Human urine is an uncommon source of infection because prevalence of human infection is relatively low and leptospiras do not multiply or survive for long in urine. Leptospirosis is not an intestinal infection, and the organisms are not likely to be present in the feces. Workers in occupations likely to bring them into contact with human or animal urine should be encouraged to wear protective clothing, rubber boots, and gloves. This applies especially to anyone employed to remove night soil, especially urine, manually.

## Occurrence and Survival in the Environment

Leptospiras that infect animals may be found in the environment where animal urine is present, especially in water, mud, slime, or soil.

### *In water and sewage*

Chang, Buckingham and Taylor (1948) found that the survival of *L. icterohaemorrhagiae* in water was heavily dependent on the temperature and the level of bacterial contamination. In river water, the leptospiras survived for 8–9 days at 5–6°C, but at 20–27°C they survived for only 5–6 days, and at 31–32°C their life span was reduced to 3–4 days. In tap water containing 10 per cent human sewage they survived for 6–7 days at 5–6°C, 3–4 days at 25–27°C, and 2–3 days at 31–32°C. At atmospheric temperature, survival times were 18–20 days in sterile tap water, 10–12 days in tap water with added mixed bacterial flora, and only 12–14 hours in undiluted sewage. When the sewage was aerated, however, the leptospiras survived for 2–3 days, indicating that the adverse effect of heavy bacterial growth may be due to anaerobic conditions and to a lowered pH value.

Chang, Buckingham and Taylor (1948) also found that survival in water was greatly reduced by high or low pH and by salinity. In tap water without bacterial contamination, the leptospiras remained viable for over 4 weeks at neutral pH, provided that some nutrients were present. At pH 5 the survival time was reduced to less than 2 days. High pH values were also detrimental. Leptospiras survived for only 18–20 hours in seawater. The hostility of salinity to leptospiras is confirmed by the work of Jamieson, Madri and Claus (1976). At a range of salinities (0.5, 2.0, and 3.5 percent) and temperatures (4, 25, and 37°C), serotype *pomona* survived for less than 24 hours.

The addition of nutrients to sterile water enhances the survival of leptospiras. In sterile tap water plus 1 percent tryptose, survival was up to 50 days, whereas the addition of 0.1 percent horse serum increased the survival time still further to 102 days. However, in the presence of other bacteria the addition of nutrients had the opposite effect. By favoring the multiplication of the other bacteria, addition of nutrient reduced the survival of the leptospiras to approximately 40 hours (Chang, Buckingham and Taylor 1948).

Spinu and others (1963) reported that leptospiras survived for 2–5 days in stream water at 22–26°C. Diesch (1971) recorded a 3-day survival period for leptospiras in stream water and well water.

Noguchi (1918) reported that in unpolluted water, such as drinking water, leptospiras (serotype *icterohaemorrhagiae*) did not remain infectious for longer than one week. Further experiments proved that leptospiras will not grow or survive for long in highly contaminated water, such as polluted river water, sewage, or stagnant cesspools. They invariably disappeared in 48 hours. Noguchi tested the survival time of *icterohaemorrhagiae* in culture medium to which various species of aerobic bacteria had been added. He showed that bacilli of intestinal origin were extremely antagonistic to the growth of the leptospiras.

In summary (see also the appendixes of Feachem and others 1980), leptospiras in clean, sterile water at cool temperatures may survive for up to 20 days, and they may grow and survive for 100 days in the presence of suitable nutrients. However, in water with a rich bacterial flora and at warm temperatures, precisely the environment in which leptospiras are likely to be found after being shed from an infected animal in the tropics, survival times are probably between 1 and 5 days.

### *In urine*

Leptospiras are shed in the urine, and therefore survival in urine is an important determinant of the risk

of new infections, especially in excreta disposal systems such as the Vietnamese double-vault latrine, which disposes of urine separately from feces. Noguchi (1918) added serotype *icterohaemorrhagiae* to normal human urine and found that it survived for less than 24 hours due to the acidity. When the urine was neutralized or made slightly alkaline they survived for 24 hours but not for 48 hours. However, when nutrients were added in the form of rabbit serum plasma to neutral or slightly alkaline urine, growth occurred for approximately 10 days. Highly alkaline conditions were as detrimental to leptospiras as was acidity.

*In feces and night soil*

Leptospiras are not passed in the feces of infected animals but will be mixed with feces in most excreta disposal systems in which urine and feces are treated together. Noguchi (1918) showed that leptospiras would not survive for longer than 24 hours in an emulsion of feces, either normal or from jaundiced patients, with or without added nutrients. This was thought to be due to bacterial contaminants that outgrew the leptospiras, since in sterile fecal material with added nutrients leptospiras survived and remained virulent to guinea pigs for 4 days. Diesch (1971) found that serotype *pomona* survived for up to 5 days in liquid cattle manure.

*In soil*

Noguchi (1918) added leptospiras to samples of soil rich in organic matter and neutral in pH. The organisms were not detected after 72 hours because of bacterial overgrowth. The more vigorous the growth of bacteria, the less able were the leptospiras to survive.

Karaseva, Chernukha and Piskunova (1973) contaminated measured areas of soil with urine of voles shown to be carriers of leptospiras belonging to serogroups Grippotyphosa or Hebdomadis. It was found that the shortest survival time occurred in areas where the moisture content was low (9.5–16.5 percent), where there was little shade, and where the pH was 5.5. Under these conditions the leptospiras survived for only 6–12 hours. In marshy areas where the moisture content was high (40–60 percent) and the pH 6.9–7.4, leptospiras were seen actively motile for 4–7 days. In deep shade provided by reeds on the shore of a lake, the survival time was 15 days. In one experiment a leptospirosis-free vole was infected with Grippotyphosa serogroup by inoculation with washings from soil contaminated with infected vole urine 5 days before. Thus, the survival time of pathogenic leptospiras in soil under favorable conditions was 15 days, with preservation of their pathogenic properties for at least 5 days.

## Inactivation by Sewage Treatment Processes

Chang, Buckingham and Taylor (1948) conducted experiments to determine the resistance of leptospiras to halogen compounds and synthetic detergents and showed that leptospiras are more sensitive to chlorine than are the enteric bacteria. Synthetic detergents that are cationic were shown to be highly leptospiricidal, whereas the anionic detergents were inactive except at high dosage.

Diesch (1971) seeded a laboratory model oxidation ditch with a virulent strain of serotype *pomona* to simulate the shedding of leptospiras by beef cattle. Leptospiras were recovered for at least 61 days. Diesch concluded that an oxidation ditch containing cattle manure is an adequate environment for the survival of leptospiras. If sludge or effluent from such a ditch is used to fertilize land, or if effluents are discharged to rivers, there is clearly a risk of leptospiral transmission to men and other animals.

Diesch (1971) also reported a 5-day survival in the effluent, and a 4-day survival in the sludge, from a cattle manure settling chamber. When compared with the 61-day survival time in the oxidation ditch, these results highlight the importance of oxygen to the survival of leptospiras. Similarly, McGarry and Stainforth (1978) reported Chinese data that leptospiras survive for less than 30 hours in the rich anaerobic fecal liquor of a biogas plant.

Processes with short retention times and involving aeration (for example, trickling filters, activated sludge, oxidation ditches) cannot guarantee the destruction of leptospiras. However, if sewage is held for a week or more, as in waste stabilization lagoons, leptospiras will not survive. Any anaerobic process, such as a septic tank, will also rapidly destroy leptospiras.

## Inactivation by Night Soil and Sludge Treatment Processes

Most night soil and sludge treatment processes are anaerobic and will rapidly destroy leptospiras. Diesch (1971) reported a 4-day survival for serotype *pomona* in a cattle manure sludge. However, under many anaerobic conditions survival times are unlikely to exceed 2 days, and there may well be differences among the survival characteristics of the different serotypes (for

instance, serotype *pomona* may survive for longer than *icterohaemorrhagiae*).

Leptospiras are sensitive to heat and will be rapidly inactivated by thermophilic sludge treatment processes. Chang, Buckingham and Taylor (1948) recorded the following thermal death points in distilled water: 30 minutes at 45°C, 10 minutes at 50°C, 10 seconds at 60°C, and <10 seconds at 70°C.

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# 15

# *Salmonella*, Enteric Fevers, and Salmonelloses

SALMONELLA bacteria are a cause of diarrhea, and less commonly enteric fever, throughout the world. They are distinct from most of the other major bacterial and viral agents of diarrhea (see chapters 11, 13, 16, and 17) in that, with the exception of the typhoid and paratyphoid bacteria, they commonly infect many species of mammals, birds, reptiles, and other animals. Human infections are frequently associated with contact with animal feces or ingestion of contaminated animal products.

## Description of Pathogens and Diseases

*Salmonella* bacteria, and the infections they cause, are well described in a voluminous medical and veterinary literature. Only a brief summary is given here.

### *Identification*

Salmonellosis is any infection with bacteria of the genus *Salmonella*. In man, most of the many serotypes of *Salmonella* give rise to a transient intestinal infection manifested as acute gastroenteritis with diarrhea and abdominal cramps. Some of the bacteria may transiently be found in the blood, and there may be fever, nausea, and vomiting as additional symptoms. But in infections with some serotypes, particularly *S. typhi*[1] (the causative organism of typhoid fever), *S. paratyphi* A, and *S. paratyphi* B (also designated *S. schottmuelleri*), the bacteria invade the tissues and produce a septicemia with a high temperature rather than diarrhea. This is known as enteric fever. Other salmonellae may sometimes give rise to a disease resembling typhoid, and a third syndrome—again associated mainly with particular serotypes, but especially in people with impaired resistance to infection—includes pyogenic lesions of internal organs. The salmonellae involved include *S. paratyphi* C (also designated *S. hirschfeldii*), *S. enteritidis* var. *chaco*, *S. sendai*, *S. dublin*, *S. typhimurium*, and *S. cholerae-suis*. Most other serotypes predominantly give rise to acute gastroenteritis, but many *Salmonella* infections are symptomless.

For convenience in this chapter, the septicemic syndrome seen most typically in typhoid will be referred to as enteric fever, and the primarily gastrointestinal pattern of infection as salmonellosis.

Diagnosis in cases of gastroenteritis is by isolation of the bacteria from feces or rectal swab, but in the first week of enteric fever isolation of the bacteria is more likely from blood culture, and only later are the organisms regularly found in the feces. In enteric fever there is commonly a rise in agglutinating antibodies during the course of infection. This is the basis of the Widal reaction as a method of diagnosis in the absence of bacteriological facilities. A positive reaction should be based on a fourfold or higher rise in the titer of antibodies, since past exposure or vaccination may have led to preexisting antibodies, but even then the test is unreliable because as many as half of all cases may fail to demonstrate a fourfold rise in antibody titer.

### *Occurrence*

Salmonellae are found world-wide but the pattern of common serotypes varies considerably from region to region. Some serotypes, for instance *S. typhi* and *S. typhimurium*, are found in many parts of the world, whereas others have a very localized occurrence. In all countries a few serotypes are responsible for most reported human and animal infections, and many other serotypes are found rarely. In many developed countries *S. typhi* is now rare, and most of the cases that do occur are contracted elsewhere.

1. See the subsection "Infectious agents", below, for a note on taxonomic nomenclature.

Figure 15-1. Salmonella enteritidis *under scanning electronmicroscopy*. Colonization of the small intestine of a rat. Scale bar = 3 micrometers. (Photo: C. D. Garland, Department of Agricultural Science, University of Tasmania, Hobart, Australia)

*Infectious agents*

The genus *Salmonella* is a member of the family Enterobacteriaceae and is distinguished from other so-called genera of Enterobacteriaceae primarily on biochemical criteria. For example, unlike most *E. coli* (chapter 13), *Salmonella* strains typically do not ferment lactose. Salmonellae are nonsporulating, Gram-negative, motile, noncapsulate rods (0.5 by 2–4 micrometers) with peritrichous flagella (see figure 15-1). They are facultative anaerobes.

In 1934 it was agreed that each new antigenically distinct isolate of *Salmonella* should be assigned a specific Linnaean epithet, often according to the place at which it was first isolated. This led, however, to an unhelpful proliferation of names and, at the time of writing, there are around 2,000 named serotypes. Not all the serotypes listed in the 8th edition of *Bergy's Manual* (1974) have been given names, and the practice has been discontinued.

The genus *Salmonella* can be sub-divided on biochemical grounds into four subgenera; only subgenus I is common in human disease. Subgenera I, II, and IV include many named serotypes, whereas subgenus III contains only the named species. *S. arizonae*, although many unnamed serotypes have been described (table 15-1).

In an attempt to simplify the situation, Edwards and Ewing (1972) proposed that only three species of *Salmonella* should be recognized: *S. typhi* (one serotype), *S. cholerae-suis* (one serotype), and *S. enteritidis* (around 2,000 serotypes). Following this schema, all serotypes except *S. typhi* and *S. cholerae-suis* should be designated *S. enteritidis* serotype ——— (table 15-1). *S. arizonae* was considered to be a separate genus (*Arizona*) rather than a subgenus of *Salmonella*. A similar reform was proposed by Cowan and Steel (1974), save that *S.* [illegible] was the name given to the aggregate species. This move towards a reduced number of species is reflected in the approved list of bacterial names (Skerman, McGowan and Sneath 1980): *S. cholerae-suis* and *S. typhi* retain their specific status; *S. typhimurium* is retained because of the frequency of its occurrence; *S. enteritidis* is the aggregate species; and *S. arizonae* consists of members of subgenus III.

In this book we have adhered to the system of nomenclature described in the 8th edition of *Bergy's Manual*, except that we have continued to use the names *S. paratyphi* B and *S. paratyphi* C, since they are more familiar to medical microbiologists.

*Reservoirs*

Salmonellae are primarily pathogens of animals, which provide important reservoirs for the infections of man other than enteric fever. Person-to-person transmission also occurs, and the relative importance

Table 15-1. *Variations in the nomenclature of some important types of* Salmonella

| *Bergy's Manual* (8th ed., 1974) | Edwards and Ewing (1972) |
|---|---|
| *Subgenus I (numerous named serotypes)* | |
| *S. typhi* | *S. typhi* |
| *S. cholerae-suis* | *S. cholerae-suis* |
| *S. enteritidis* | *S. enteritidis* serotype *enteritidis* |
| *S.* [illegible] | *S. enteritidis* serotype [illegible] |
| *S. hirschfeldii* (*paratyphi* C) | *S. enteritidis* serotype *paratyphi* C |
| *S. paratyphi* A | *S. enteritidis* serotype *paratyphi* A |
| *S. schottmuelleri* (*paratyphi* B) | *S. enteritidis* serotype *paratyphi* B |
| *S. typhimurium* | *S. enteritidis* serotype *typhimurium* |
| *Subgenus II (numerous named serotypes)* | |
| *S. salamae* | *S. enteritidis* serotype *dar es salaam* |
| *Subgenus III (numerous unnamed serotypes)* | |
| *S. arizonae* | *Arizona hinshawii* |
| *Subgenus IV (numerous named serotypes)* | |
| *S. houtenae* | *S. enteritidis* serotype *houten* |

of the human and nonhuman reservoirs depends upon the dietary, agricultural, and sanitary situation in a particular community.

A few *Salmonella* serotypes are almost species specific. Most importantly, *S. typhi* is a pathogen of man, and therefore the source of infection is the human case or carrier. *S. paratyphi* A, B, and C and *S. sendai* also have their reservoirs primarily in man, although infections of other animals have been reported.

Salmonellae are commonly isolated from poultry, especially chickens, turkeys, and ducks (Goyal and Singh 1970), and from livestock such as pigs, cows, sheep, and horses (Baker 1970). The prevalence of infection among these poultry and farm animals is especially high under some systems of intensive indoor farming or where contaminated foodstuffs (especially fish meal and bone meal) are used (Al-Hindawi and Taha 1979; Joint Working Party 1965; Lee 1974). In the UK and the USA between 15 and 50 percent of dressed poultry in retail stores may be contaminated by *Salmonella*. Other domestic animals and pets (for instance, cats, dogs, mice, guinea pigs, and hamsters) are frequently infected. Both rats and mice that live in close proximity to human communities may be infected. Their carrier rate may sometimes rise above 10 percent, which is in contrast to the very low prevalence observed in surveys of other wild mammals that do not appear to constitute a reservoir of infection for man or domestic animals (Jones and Twigg 1976).

Gulls, pigeons, and doves have been implicated as major reservoirs of salmonellae, but other wild birds are more rarely infected. Jones, Smith and Watson (1978) isolated salmonellae from 62 percent of samples of gull droppings from a large gullery in northwestern England, whereas Plant (1978) isolated *Salmonella* from only 0.17 percent (1/599) of wild birds examined at two sewage treatment works in southeastern England. Hussong and others (1979) failed to isolate *Salmonella* from forty-four migratory waterfowl wintering at Chesapeake Bay (USA). The infection of gulls may be due to their habit of scavenging for food at rubbish tips and sewage treatment works. Gulls have been implicated in the contamination of water reservoirs by salmonellae, and in the spread of salmonellae from refuse tips to cattle on nearby pastures (Williams and others 1977).

Many species of reptiles and other cold-blooded animals are carriers of *Salmonella* (Goyal and Singh 1970). This is of epidemiological importance in cases where these animals have close contact with man. Particular concern has been expressed over the high prevalence of *Salmonella* excretion among pet turtles, terrapins, tortoises, frogs, and snails (Bartlett and Trust 1976; Bartlett, Trust and Lior 1977; Lamm and others 1972). It has been suggested that the decrease in human infection with *S. java*, *S. litchfield*, and *S. urbana* in Canada since 1975 has been due to the government prohibition of the importation of turtles, which were popular as pets (D'Aoust and Lior 1978). In 1975 the interstate transport of pet turtles became illegal in the USA (MMWR 1975). Among the arthropods, fleas, ticks, lice, cockroaches, and houseflies may harbor salmonellae.

Nonhuman isolations of *Salmonella* in England and Wales during 1968–74 were reviewed by Sojka and others (1977). Of a total of 23,609 incidents of *Salmonella* infection, 86 percent were in cattle, 7.4 percent in poultry and other birds, 2.9 percent in sheep, 2.4 percent in pigs, and 1.3 percent in other species, especially horses, dogs, mink, guinea pigs, and cats. These figures do not reflect the relative incidence of salmonellosis among various species but rather the commercial importance of certain farm animals and the policies of veterinary laboratories. Although 153 serotypes were isolated, 88 percent of incidents were due to *S. dublin* and *S. typhimurium*. Other common serotypes, in decreasing order of importance, were *S. cholerae-suis* (almost entirely restricted to pigs), *S. abortus-ovis* (entirely restricted to sheep), *S. newport*, *S. agona*, *S. virchow*, *S. anatum*, *S. enteritidis*, and *S. montevideo*.

### *Transmission*

The transmission of typhoid fever was partly elucidated before the bacteriological era by William Budd (1856, 1873). He showed that it was spread by contagion, that infective material was excreted in the feces, and that the disease could be spread by the tainted hands of those who waited on the sick or by contamination of water and milk.

*Salmonella* transmission takes place when the infected feces of man or animal are ingested by a susceptible person. In the case of typhoid fever, transmission is only from human feces or urine to mouth. This fecal-oral (or occasionally urinary-oral) transmission may be direct where personal cleanliness is poor, or it may be via contaminated food or water. Large numbers of bacteria are excreted, and high infective doses are required to infect most persons and animals.

Cases of salmonellosis or enteric fever may excrete up to $10^{10}$ *Salmonella* per gram of feces. Unlike other bacterial enteric infections, asymptomatic carriers may also excrete very high concentrations. Thomson (1954, 1955) studied *Salmonella* excretion by cases and

carriers. Twenty cases of salmonellosis excreted $2.5 \times 10^5$–$1 \times 10^9$ *Salmonella* per gram of feces, and 6 *Salmonella* carriers excreted $1 \times 10^4$–$5 \times 10^7$. Seven cases of paratyphoid B excreted $1 \times 10^4$–$5 \times 10^9$ *S. paratyphi* B per gram, and 12 paratyphoid B carriers (known duration 1–20 years) excreted $5 \times 10^5$–$1.2 \times 10^{10}$ *S. paratyphi* B per gram. Half of the paratyphoid B carriers excreted more *Salmonella* per gram than *E. coli*. Eight typhoid carriers (known duration 3–12 years) excreted $5 \times 10^5$–$4.5 \times 10^7$ *S. typhi* per gram. Merselis and others (1964) found that thirteen typhoid carriers (known duration 4–41 years) excreted $10^4$–$10^{11}$ *S. typhi* per gram of feces.

The infective dose for typhoid and other salmonellae is high in healthy adults. In a major series of studies, McCullough and Eisele (1951*a*, 1951*b*, 1953*a*, 1953*b*) fed eggnog containing various doses of *S. anatum*, *S. bareilly*, *S. derby*, *S. meleagridis*, *S. newport*, and *S. pullorum* to healthy adult prisoners. The doses needed to produce clinical symptoms in about half of the volunteers were between $9 \times 10^5$ for *S. anatum* and $4 \times 10^9$ for *S. pullorum*. No clinical disease was produced with less than $1.3 \times 10^5$ organisms, but infection and a temporary carrier state was established with only $1.2 \times 10^4$ *S. anatum*.

Hornick and others (1970) reported a median infective dose ($ID_{50}$) of *S. typhi* for healthy adult male volunteers of $10^7$ organisms in 30 milliliters of milk. No disease was produced in any of 14 volunteers by $10^3$ organisms. In subsequent tests, five different *S. typhi* strains were fed to volunteers at a dose of $10^7$ and induced disease in between 21 and 56 percent and infection or disease in between 60 and 93 percent. As with all $ID_{50}$ data, it must be remembered that the attack rate in most common source outbreaks is very much less than 50 percent.

Although the $ID_{50}$s for salmonellae are high, some individuals may be infected and made ill by much smaller doses. D'Aoust and Pivnick (1976) reported outbreaks caused by 15,000 *S. cubana* in carmine dye capsules, by 1,000 *Salmonella* in ice-cream, and by chocolates containing only 100 *S. eastbourne* per gram. In children with abnormal gastrointestinal tracts due to cystic fibrosis, Lipson (1976) reported infection with *S. schwarzengrund* from probably $<100$ bacteria.

The infective dose of *Salmonella* for healthy cattle is high, although young calves can be readily infected, and sometimes killed, by a dose of only $10^4$ *S. typhimurium* (Deans Rankin and Taylor 1966; Robinson and Loken 1968). Hall and Jones (1978) detected no infection in four cows each fed 1 liter of raw sludge per day containing $10^2$–$10^5$ *Salmonella* per liter. In another group of cows fed 1 liter of sterilized sludge per day to which $10^5$ *S. dublin* per liter were added, three out of four were infected but none developed diarrhea. The authors pointed out that these exposures ($10^5$ *Salmonella* per day) are higher than would be expected under normal farming conditions in which cattle graze on pasture spread with sludge. However, some cattle, like some humans, may be infected by fewer organisms if they are for any reason especially susceptible. Aitken and others (1976) showed that cows were more susceptible to *S. dublin* if they were also infected by the liver fluke, *Fasciola hepatica* (see chapter 27).

Although fecal-oral transmission of salmonellae is the norm for man and animals, the organism may be spread by other routes. Garg and Sharma (1979) isolated salmonellae from the nasal passages of 2 calves and 2 piglets out of 395 young farm animals in India. Crozier and Woodward (1962) found that chimpanzees could be given typhoid fever by the respiratory route with a dose of $10^8$ *S. typhi*, compared with a dose of $10^{11}$ organisms needed for successful oral infection. Darlow, Bale and Carter (1961) found that 57 percent (34 of 60) of mice inhaling $5 \times 10^4$ *S. typhimurium* died in 4 weeks, and all except 1.7 percent (1 of 60) were infected. By contrast, when mice ingested the same dose only 1.7 percent (1 of 60) died in 4 weeks, and 75 percent (45 of 60) were not infected. Morse, Myhrom and Greenwood (1976) cited studies from the USSR showing that *Salmonella* infection of sheep and cows by aerosols was possible with 27 percent of the dose required for infection by the oral route. Moore (1957) reported that the $ID_{50}$ of *S. enteritidis* for guinea pigs was $10^9$ when fed by mouth and only $10^2$ when placed on the eye (see also Duguid, Darekar and Wheater 1976). Thus, although fecal-oral transmission is the norm, fecal-nasal, fecal-ocular, and nasal-nasal routes are also theoretical possibilities (see also Wray and Sojka 1977).

### *Incubation period*

In typhoid fever the incubation period is usually between 5 and 21 days, whereas that of paratyphoid tends to be less. Typically, the first symptom to appear is fever due to bacteremia; only later, usually in the second and third weeks of the illness, do bacteria invade the intestine from the blood stream in increasing numbers to be detected in fecal specimens. The typical symptoms of enteric fever may be preceded by acute gastroenteritis. Most other salmonelloses have considerably shorter incubation periods, generally between 8 and 36 hours, typically followed by headache, nausea, vomiting, abdominal pain, fever,

and diarrhea. Incubation periods are inversely proportional to infecting dose.

*Period of communicability*

In typhoid and other enteric fevers, the *Salmonella* species responsible appears in the excreta late in the first week of illness or thereafter. During the first few weeks of convalescence the proportion of patients excreting typhoid bacilli falls, until about 3 months after the onset of illness less than 10 percent of clinical cases continue to excrete *S. typhi* in their feces. Two to five percent may become chronic carriers and excrete the pathogen for over 1 year. A small proportion remain carriers for 10, 20, 30 years or a lifetime. As noted above, chronic typhoid carriers may excrete $10^4$–$10^{11}$ *S. typhi* per gram of feces and therefore constitute a major source of infection within a community. The gall bladder is the organ that typically remains infected, and gall stones may predispose towards the carrier state.

Some typhoid and paratyphoid cases pass *Salmonella* in their urine. Chronic urinary carriers of typhoid and paratyphoid are, however, much less common than fecal carriers, except in countries where urinary schistosomiasis (see chapter 32) is common. In such areas many urinary carriers of enteric fever bacilli are found, and the prevalence may be as high as 3 percent of typhoid convalescents. Hathout and others (1966) found that, among forty-nine males in Egypt with *Schistosoma haematobium* infection who contracted typhoid or paratyphoid A, 47 percent were still excreting *Salmonella* in their urine 1 year after the diagnosis of enteric fever. (Among those with *Schistosoma mansoni* infection, typhoid and some other *Salmonella* infections give rise to severe illness over many months.)

In other salmonelloses, the bacterium appears in the feces concurrently with the diarrhea and for a few days to a few weeks thereafter. A temporary carrier state for around 2 months occurs in 5–10 percent of convalescents, but chronic carriers (over 1 year) are rare (less than 1 percent of cases).

In many poor communities the incidence of *Salmonella* infection is high, and many infections do not produce disease. Therefore, at any time 1–5 percent of the healthy population will be excreting *Salmonella* bacteria. Infection prevalences are higher among workers who come into daily contact with potentially infected people, animals, or animal products. The carrier rates for salmonellae among healthy adults in Baghdad (Iraq) were 1.9 percent of random persons, 2.9 percent of buffalo owners, 3.2 percent of hospital personnel, 4.2 percent of slaughterhouse workers, 6.2 percent of poultry processers, and 8.6 percent of food handlers (Al-Ani and Saadallah 1979). No salmonellae were isolated from the urine of 905 people. Al-Dulaimy and Al-Allaf (1979) reported a 1.2 percent (23 of 2,000) prevalence of *Salmonella* excretion in Mosul (Iraq). Becerril, Bessudo and González Cortés (1979) found that 13 percent (110 of 850) of food handlers, and 5 percent (73 of 1,527) of the general population, were *Salmonella* carriers in Mexico City (Mexico). Various studies in South Africa showed *Salmonella* excretion in 2–6 percent of hospitalized children 0–2 years old without diarrhea and in 0–12 percent of healthy schoolchildren (Koornhof and others 1979).

In contrast, Gordon and others (1961) reported an average prevalence of *Salmonella* excretion among about 10,000 healthy children under 10 years old in highland and lowland villages in Guatemala of only 0.2 percent. Other data showed the *Salmonella* was not an important cause of diarrhea in that age group in those villages (Pierce and others 1961). Similarly, studies in the USA in the 1950s showed that *Salmonella* was rarely excreted by healthy children from lower socioeconomic groups. Among preschool children in Kentucky the prevalence of *Salmonella* excretion was 0.2 percent, whereas that for *Shigella* was 3.1 percent (Schliessmann and others 1958), and among children under 10 years old in California the prevalence of *Salmonella* excretion was 0.4 percent, whereas 4.6 percent excreted *Shigella* (Watt and others 1953).

*Resistance*

For the enteric fevers and the salmonelloses, individuals who have gastric hypochlorhydria, who are sick or who are on antibiotic therapy are especially susceptible. Indeed, *Salmonella* infections frequently select debilitated individuals, and hospital outbreaks are often especially serious.

An attack of typhoid fever confers some immunity to reinfection, usually life-long, but second and third attacks have been reported. Although antibodies to several *Salmonella* antigens are readily detectable in serum, they correlate poorly with resistance. In some areas where typhoid is endemic, about 5 percent of cases occur in those under 5 years old and perhaps 4 percent are fatal. About 45 percent of cases occur in the 5–19 age group, and fatalities range from 2 to 6 percent. Thereafter, from ages 20 to above 60 years, the incidence rate falls progressively, but the case fatality rate increases to over 30 percent in cases over 60 years old. It may be inferred that many in the community become immune during childhood and adolescent

exposure, though proof is lacking. The outcome of challenge depends also on dose size and the virulence of the strain of *S. typhi*.

Among the other salmonelloses, attack rates are highest in the age group under 5 years and especially high in infants under 1 year. Significant immunity is not conferred; in any case, the next challenge is likely to be from a different serotype.

### *Epidemiology*

The salmonelloses have a markedly seasonal incidence, peaking in the warmest months along with all diarrheal diseases. This may be in the warm-wet season, as in Lesotho and India, or in the warm-dry season, as in such countries as the UK, El Salvador, Guatemala, and the USA. In those countries where it is endemic, typhoid may peak at the same time or a few weeks later. A late summer or autumn high incidence of typhoid, for example, follows the summer peak salmonellosis incidence in Lesotho, India, and Tunisia.

Although salmonelloses constitute an important cause of diarrhea, especially foodborne diarrhea, in many developed countries and are also of major veterinary importance in some areas, *Salmonella* are responsible for only a very small proportion of infant and childhood diarrhea in some developing countries. In these latter places *Salmonella* are completely overshadowed as a cause of childhood diarrhea by enterotoxigenic *E. coli* (chapter 13), rotavirus (chapter 11), and sometimes also *Shigella* (chapter 16) and *Campylobacter* (chapter 12). For example, *Salmonella* was associated with only 1 percent of childhood (1–5 years) diarrhea in Guatemala (Pierce and others 1961), 0–1.7 percent of childhood diarrhea in Panama (Kourany and Vasquez 1969), less than 1 percent of childhood (under 5 years) diarrhea in Bangladesh (Black and others 1979), and 1.4 percent of diarrhea cases among young children in a Gambian village (Barrell and Rowland 1979). In contrast, studies on both black and white children (0–2 years) in South Africa showed *Salmonella* excretion in between 6 and 17 percent of diarrhea cases and 2–6 percent of healthy controls (Koornhof and others 1979). This contrast might suggest that *Salmonella* diarrhea becomes more prominent as economic conditions improve, since the South African children studied were probably more urban and wealthier than the Guatemalan or Bangladeshi groups. However, this economic distinction falls down in the case of Panama, and it was shown in the 1940s and 1950s that *Salmonella* were not a major cause of childhood diarrhea in California (Watt and others 1953), Kentucky (Schliessmann and others 1958), and Texas (Watt and Lindsay 1948) in the USA. It may be concluded that there are major unexplained differences in the prominence of *Salmonella* diarrheas in children (as there are with all individual agents of diarrhea), but that in many poor communities salmonellae do not make a major contribution to overall pediatric diarrheal morbidity and mortality.

Typhoid is commonly endemic in Africa and is especially prominent in some upland areas. Characteristic features of endemic highland typhoid are illustrated by the situation in Lesotho described by Feachem and others (1978). At one major hospital (St. Joseph's Hospital, Roma) during 1971–74, typhoid accounted for 1 percent of all attendances (inpatients and outpatients) and 5 percent of all admissions (inpatients). The highest age-specific incidence occurred in the 15–24 age group. The fatality rate among all hospitalized typhoid cases was about 6 percent, whereas in patients over 35 years it was 13 percent. Typhoid in Lesotho had a pronounced seasonal pattern, with major outbreaks occurring every 2 or 3 years during March–April; temperature and rainfall peaked during November–March, and all diarrhea reportings peaked during December–February.

The maximum typhoid incidence among the 10–30 age group is typical of endemic typhoid and has been reported from, for instance, Antigua (Uttley 1960), Dominica (Grell 1979), India (Mathur and Sharma 1971), Jamaica (Miller, Grant and Irvine 1961), and Tunisia (Miled, Zribi and Ben Rachid 1973). Ashcroft (1962) reported a peak incidence of typhoid in Guyana during 1956–60 among slightly younger children (5–14 years) and suggested that the age of peak incidence might rise as sanitation improved and children were less frequently immunized by mild or asymptomatic infections.

Typhoid fever in the UK has been reviewed (Public Health Laboratory Service Standing Sub-Committee on the Bacteriological Examination of Water Supplies 1978). A major waterborne outbreak (341 cases and 43 deaths) occurred in Croydon in 1937 (Holden 1939). Between 1941 and 1970, several small outbreaks were traced to the ingestion of well or stream waters contaminated by chronic typhoid carriers. Other outbreaks were caused by contaminated milk, oysters, and imported corned beef. A major typhoid outbreak in Aberdeen (Scotland) in 1964, with 507 cases and 3 deaths, was caused by a single tin of contaminated corned beef from South America. The meat was sliced and sold at a supermarket, and about 50 people were infected from this corned beef. The remainder were

infected by eating other cold meats that had been sliced by the same machine in the same supermarket (Howie 1968; Walker 1965). Since about 1955 the annual incidence of typhoid in Britain has been very low. During 1975 and 1976, 419 cases were reported in England, Wales, and Northern Ireland, and 85 percent of these were infected abroad. By contrast, only 33 percent of 1,418 *S. typhi* infections in the USA during 1967–72 were associated with foreign travel, although there was a markedly rising trend in this percentage (17 percent in 1967, 46 percent in 1972) (Rice, Baine and Gangarosa 1977). Over this period, indigenous cases of *S. typhi* infection were mainly between 5 and 30 years old, cases associated with travel were mainly in those aged 10–30 years, and known typhoid carriers were predominantly elderly women.

Human salmonelloses in the USA during 1963–67 were reviewed by Aserkoff, Schroeder and Brachman (1970). About 20,000 cases were bacteriologically confirmed and reported annually. The annual peaks occurred in July–October, and the age-specific incidence was by far the highest in those under 4 years old. The reported incidence in Hawaii was nearly 10 times the national average in each year, and this was attributed to the use of pig intestines in soups and stews by certain ethnic groups. There were 180 epidemics, comprising 16,772 cases, during 1963–67 , and 156 of these were due to a common vehicle of infection—especially eggs, egg products, and turkey. Less commonly chicken, beef, pork, milk, pet chickens, pet ducks, and pet turtles were implicated. The largest number of epidemics were domestic, but the largest epidemics were those associated with banquets and schools. During the period *S. typhimurium* was by far the most commonly identified serotype, constituting 30 percent of all human isolations and 17 percent of all nonhuman isolations. Poultry and poultry products accounted for half of all nonhuman isolations of *Salmonella*. Some serotypes exhibited a considerable degree of species specificity: *S. pullorum* and *S. gallinarum* in fowl, *S. abortus-ovis* in sheep, and, to a lesser degree of specificity, *S. cholerae-suis* in pigs and *S. dublin* in cattle.

Food-borne outbreaks of typhoid and other salmonelloses are commonly reported. Sometimes they are due to contaminated raw materials, such as poultry, tinned meat, or eggs, while in other cases the food is contaminated by a food handler. Carrier rates for *Salmonella* are typically higher among food handlers than among the general population and there is a close relationship between contaminated food and infected food handlers which places the consumer of the food at risk.

"Food poisoning" of bacterial origin may be due to several different organisms, including *Staphylococcus aureus*, *Clostridium botulinum* and *Cl. perfringens*, and *Vibrio parahaemolyticus*. The bacteria multiply in the contaminated food and may produce exotoxins in the case of *Staphylococcus* and *Clostridium*, when symptoms result from ingestion of the preformed toxin. In infective food poisoning the gastrointestinal symptoms follow proliferation of the bacteria in the human intestine. When the pattern of food preparation provides opportunities for salmonellae to multiply, infection is naturally more likely.

In developed countries the primary vehicles of foodborne salmonelloses depend on dietary habits and particularly on the main source of animal protein. Poultry and egg products are the most frequently implicated vehicles in many countries. In developing countries, where less animal protein is eaten and animals are not typically raised by "factory farming" methods, the contamination of food by infected food handlers may be more important than the ingestion of naturally contaminated animal products.

Food contamination is believed to be an important factor contributing to the high prevalences of *Salmonella* excretion among African schoolchildren in South Africa (table 15-2). Surveys of food in Pretoria and Soweto showed that 48 percent of tripe samples, 29 percent of intestines, 40 percent of pork sausage, 64 percent of minced beef samples, 20 percent of chicken carcasses, and 16 percent of biltong samples (dried meat eaten raw) were contaminated by salmonellae (Prior and Badenhorst 1974; Richardson, Burnett and Koorhof 1968). Bokkenheuser and Richardson (1959) found that 4.3 percent (67 of 1,565) of food handlers employed at nineteen gold mines in the Transvaal were infected by *Salmonella*.

An outbreak of sixty-nine typhoid cases after a party in Cape Town (South Africa) in 1978 was traced to the main caterer who had prepared and stored chickens for the party under grossly unhygienic conditions (Popkiss 1980). The main caterer was found to excrete *S. typhi* of the same phage type as those isolated from the cases. On May 5–7, 1977, 545 university students at Trujillo (Peru) were hospitalized with acute gastrointestinal symptoms due to *S. thompson* (Gunn and Loarte 1979). The outbreak was traced to the university dining hall, and 93 percent of the students who regularly ate there became ill. The implicated vehicle was sardine-mayonnaise salad. Barrell and Rowland (1979) found that *Salmonella* were associated with 1.4 percent of diarrhea cases among young children in a Gambian village and that 4.7 percent of infant food samples were contaminated by salmonellae.

Waterborne outbreaks have been chiefly associated

with *S. typhi* and much less frequently with *S. paratyphi* or other *Salmonella* serotypes. The evidence implicating a water supply is usually circumstantial, and the causative organism is rarely isolated because the pollution that gives rise to an epidemic is often temporary or intermittent. Waterborne outbreaks due to gross contamination are usually characterized by an explosive onset. The majority of cases develop over a period of a few days, and these may be followed by a secondary crop of contact cases. Sometimes the outbreaks may happen as a series of scattered cases occurring over a considerable period of time, and this may be due to a lower and intermittent contamination of the supply.

Waterborne disease outbreaks in the USA during 1971–73 have been reviewed (Hughes and others 1975; Merson and others 1974). Typhoid fever was the cause of 2.5 percent (217 of 8,537) of known cases and 4.2 percent (3 of 71) of known outbreaks, while other salmonelloses were the cause of 0.04 percent (3 of 8,537) of cases and 1.4 percent (1 of 71) of outbreaks. Large, and probably waterborne, outbreaks of diarrhea due to *S. typhimurium* occurred in Riverside (California) in 1965 (Greenberg and Ongerth 1966) and in Suffolk County (New York State) in 1976 (Zaki and others 1979).

Feldman and others (1974) described an outbreak of typhoid (225 cases and no deaths) at a migrant farm labor camp in Florida (USA) in 1973. A case-control study linked the infection to the camp water supply, which was pumped from two wells. The water supply was chlorinated but with inadequate contact time during peak water demand, and the chlorinator had a history of malfunction. High coliform counts were obtained from the camp water on several occasions, and it was discovered that surface drainage and sewage might gain access to the wells. It was estimated that the average infecting dose in the outbreak was $10^3$–$10^5$ *S. typhi* and that such doses could have been administered by the water supply if a case or carrier with heavily contaminated stools (say $10^{11}$ *S. typhi* per gram) had defecated in the pump house, around the well, or into the water storage tank.

In the notorious typhoid outbreak in Zermatt (Switzerland) in March 1963 (Bernard 1965) there were 437 cases and 3 deaths. Some evidence implicated the inadequately chlorinated water supply, which may have become contaminated by a carrier working on the catchment or by leakage of sewage into the water system.

Mendis and others (1976) described a very persistent outbreak of diarrhea due to *S. bareilly* in the maternity hospital in Colombo (Sri Lanka). Initial person-to-person spread led to the contamination of the hospital water system as a result of the suction of wastewater into water mains. *S. bareilly* became established in the water system and appeared to grow in the interior of taps and on the walls of water tanks at the water level. The contaminated water spread the infection throughout the hospital during 1968–69 so that. during January–March 1969, 48 percent (20 of 42) of hospital staff were infected. The rectification of certain plumbing defects, and the chlorination of the mains and tanks, led to the disappearance of *S. bareilly* from the water system, and the outbreak subsided.

A report from Trinidad (West Indies) described forty-eight cases of acute diarrhea among eighty-eight children and adults attending a church camp (Koplan and others 1978). *S. arechevalata* (a rare serotype) was isolated from cases, asymptomatic persons, two food items (a fish dish and stewed peas that had been prepared with roof-collected rain water), and from roof-collected rain water. The roof used for rainwater catchment was overhung by trees in which mocking birds, wrens, and doves nested and rested, and the roof was covered with dried and fresh bird feces. This may be the only account of a salmonellosis outbreak due to contaminated roof-collected rain water, but small outbreaks, in individual households, having roofs contaminated by bird droppings, may be commonplace where such rain water is a widely used water source.

The complex interactions possible between human and nonhuman reservoirs, and waterborne and foodborne transmission are illustrated by an outbreak of *S. paratyphi* B infection in five cows and ninety people in North Yorkshire (England) in 1970. The probable chain of events was that a chronic human carrier contaminated a stream, which infected a herd of cows, which infected farm workers—one of whom contaminated the water supply serving several villages (George and others 1972; Harbourne and others 1972). The failure of two types of sewage treatment to remove *S. paratyphi* B from sewage is also suggested by this study. The original chronic carrier contaminated the stream by way of sewage effluent from a "conventional" treatment plant (probably trickling filters), and the farm worker contaminated the village water supply by way of septic tank effluent from his cottage.

There is extensive evidence of direct person-to-person spread of salmonelloses and enteric fevers under conditions of poor hygiene, in crowded institutions, or simply in a normal family setting. Budd (1856) described an outbreak of typhoid at a military school in France in 1826 and reported that among twenty-nine cases nursed at home, eight were known to

have transmitted the disease to persons attending them. Rosenstein (1967) studied the family contacts of twenty-eight sporadic index cases of *Salmonella* diarrhea in Rhode Island (USA). Thirty-five percent (42 of 121) of family contacts were found to be infected by *Salmonella* of the same serotype as the index case, and 55 percent (23 of 42) of infected contacts had diarrhea. Sixty-one percent (17 of 28) of the families of the index cases had at least one infected contact. Among all family contacts, children were more likely to be infected and, if infected, were more likely to be ill. Baine and others (1973) reviewed *Salmonella* outbreaks in institutions in the USA between 1963 and 1972. Person-to-person transmission (as opposed to common vehicle outbreaks associated with contaminated food, water, or pharmaceutical products) was the major mode of transmisson in nurseries and pediatric wards and was also important in outbreaks among hospitalized adults. Person-to-person spread was implicated in 61 percent (46 of 76) of institutional outbreaks in which the mode of transmission was elucidated.

*S. anatum* could be recovered from artificially contaminated fingertips for over 180 minutes when the initial inoculation was 530 organisms per fingertip, and for 90 minutes when the initial inoculum was only 36 organisms (Pether and Gilbert 1971). In the same study, $10^6$ *S. anatum* per fingertip were not removed completely by hand washing (a 15-second wash with soap and running warm water and drying on paper towels), and unwashed contaminated fingertips could readily contaminate corned beef and cooked ham.

## Control Measures

The control of enteric fevers, which are primarily infections of man, has been achieved in many wealthy countries. The control of other salmonelloses has proved impossible due to their large and widespread animal reservoirs.

### *Individual*

The widespread resistance of *Salmonella* species, including *S. typhi*, to many antibiotics is a serious problem brought about by excessive curative and prophylactic use of antibiotics in both human and veterinary medicine. Antibiotic therapy in man is not beneficial in uncomplicated *Salmonella* diarrhea, since it does not accelerate recovery and may prolong the period of *Salmonella* excretion during convalescence. Chemotherapy, usually with chloramphenicol, ampicillin, or cotrimoxazole, is required for the treatment of enteric fevers. Prophylactic use of antibiotics for man and farm animals (a very common practice, although now illegal in some countries) is generally condemned. Antibiotic resistance in *Salmonella* is usually caused by transmissible plasmids conferring multiple antibiotic resistance. Thus, misuse of a single antibiotic can give rise to multiresistant strains. Some major epidemics of enteric fever and salmonellosis, caused by multiresistant *Salmonella* strains (especially of *S. typhi*, *S. typhimurium*, and *S. wien*), have occurred recently in man and animals.

Typhoid fever is the only disease considered in this book, except poliomyelitis, for which there is a widely used and moderately effective vaccine, and vaccination can play a role in typhoid control. Inactivated whole-cell vaccines against typhoid are given in two intradermal or intramuscular doses, with an interval of 2–4 weeks, and provide 70–85 percent protection for a period of 3–4 years. To maintain immunity at a high level, revaccination is required every 2–3 years. It has been common practice to use a TAB vaccine, which combines *S. typhi* (T) with *S. paratyphi* A and B (AB) organisms and purports to protect against all three enteric fevers. The use of TAB vaccines is, however, officially discouraged (WHO 1979) because the addition of the *S. paratyphi* organisms requires a reduction in the number of *S. typhi* present below the $10^9$ organisms necessary to produce significant antityphoid immunity. It is therefore recommended that antityphoid vaccine alone should be used. A new live oral typhoid vaccine has been tested in Egypt and is currently (1982) undergoing further field trials in Chile.

### *Carrier surveillance and control*

Eradication of typhoid from the community requires the prevention or cure of the carrier state. No uniformly successful method of curing this condition has yet been devised, although a high proportion of both typhoid and paratyphoid fecal carriers can be freed from their infection by surgical removal of the gall bladder and concomitant ampicillin therapy. The names of all chronic carriers should be registered with the local health authority, occupational restrictions imposed, and careful instructions given on strict personal hygiene to avoid infecting others. Besides being prevented from handling food for others, carriers should not be employed on or admitted to water works.

Outbreaks of enteric fever can seldom be traced to the known chronic carriers, and it is the unknown carrier that is the main danger. It is valuable to

question new employees in the catering or water supply industries to establish whether they have previously suffered from enteric fever and to examine them by standard serological and cultural procedures. These tests should be repeated at regular intervals and on individuals returning to work after any enteric infection. Excreters of pathogenic organisms identified by these procedures should be legally precluded from working in food handling and water supply until it can be demonstrated that they are completely cured.

*Environmental*

Wide-ranging economic and sanitary changes in Europe and North America over the last century, combined with methods to prevent, detect and cure typhoid carriers, have caused a very great reduction in the incidence of the enteric fevers and have certainly removed them from the list of major infectious diseases. Whether simple water and sanitary improvements applied to poor communities in developing countries can have a measurable impact upon typhoid and paratyphoid incidence remains uncertain. It is probable that a combination of improved water supply, adequate excreta disposal facilities, strenuous health education programs, and national systems for identifying, controlling and treating carriers are required.

The control of salmonelloses will inevitably prove much more difficult because of the widespread animal reservoir. In the developed countries salmonelloses may have decreased since the last century (although this is not known), but they remain a major medical and veterinary problem. The trends in diet and farming practice that accompany economic development will tend to increase the salmonellosis problem. Improved water supply and excreta disposal, coupled with tighter control of the catering industry and improved food hygiene at home, will have a beneficial effect.

Cvjetanović, Grab and Uemera (1971, 1978) constructed a mathematical model of endemic typhoid to predict the impact of immunization and latrine construction. The values fed into the model were those corresponding to the demographic, medical, and economic situation in Western Samoa. As with their cholera model (see chapter 17), Cvjetanović and his coworkers overestimated the impact, and greatly underestimated the costs, of latrine construction. They excluded all labor and material costs, which were met by the householders, and deemed the cost of a latrine to be only the costs borne by the government in the provision of supervision and technical assistance. This government input was valued at US$3.15 per latrine or US$0.50 per capita served. The value of benefits was also underestimated, however, since it was taken to be the costs of hospital treatment and lost wages for typhoid only and thus excluded the cost of death (future working days forgone) and the many other health benefits that might result from latrine construction. The model showed immunization at 5-year intervals to be greatly more cost effective than was the case for cholera (chapter 17), because of the greater effectiveness (80 percent assumed) of the typhoid vaccine and its longer period of protection (5 years assumed). The costs of five-yearly vaccinations of the population (assuming 75 percent coverage), and latrine provision for the entire population over a 10-year period, were found to be very similar, and the two strategies were predicted to have a similar effect on typhoid incidence—reducing it from around 7.2 cases to 2.5 cases per 10,000 people per year over a 30-year period. However, if five-yearly vaccinations were discontinued, incidence would increase sharply—whereas the 10-year latrine program, and a low level follow up program to cope with population increase, would continue to bring the incidence down. With vaccination alone, cumulative benefits would exceed cumulative costs after 10 years; with latrine construction alone this point would be reached after 20 years, whereas with both vaccination and latrine construction benefits would not exceed costs until after 25 years. Cvjetanović and his colleagues concluded, by using both the model and actual data on typhoid incidence in England and Wales and the USA, that continuing improvements in sanitary and economic conditions would lead eventually to the eradication of typhoid (except for imported cases) without recourse to mass vaccination. Models of this type are useful in focussing attention on the dynamics of a disease and the potential impact of various interventions. The cost-benefit analyses could be greatly improved by reconsidering some of the epidemiological variables, especially the effect of sanitation on transmission, and by incorporating more rigorous economic costing and discounting techniques.

Studies in Lesotho (Feachem and others 1978) suggested that the periodic summer typhoid outbreaks, and the sporadic cases between outbreaks, were not associated with rainfall or increased water pollution and that villages with improved water supplies providing water of good quality were not protected against these outbreaks. It was also shown that the temporal and spatial patterns of two typhoid outbreaks studied in detail were not suggestive of waterborne transmission. It was concluded that much typhoid transmission in Lesotho was nonwaterborne

and that wide ranging improvements in waste disposal, cleanliness, food hygiene, and water supply were necessary to reduce typhoid incidence. Identical conclusions were reached for all diarrheal diseases in Lesotho, an unknown proportion of which are due to *Salmonella* infections.

It is illuminating to review a series of studies on salmonellosis and shigellosis among healthy African schoolchildren in the Transvaal (Republic of South Africa; Bokkenheuser and Richardson 1960; Richardson and Bokkenheuser 1963; Richardson and Koornhof 1965; Richardson, Koornof and Hayden-Smith 1966; and Richardson and others 1968). The schools took children from very different economic backgrounds and were, in rising order of economic standard and urbanization, located at Tlaseng, Komatipoort, Witkoppen, and Soweto. The results are summarized in table 15-2. The water supply and sanitation facilities were very poor, except at Tlaseng in 1966 after a new water supply had been constructed and at Soweto where high standards of water supply and excreta disposal prevailed. The authors of these studies started out believing that waterborne transmission of *Salmonella* and *Shigella* was important and ended up concluding that it was not. After the first Tlaseng study, Bokkenheuser and Richardson (1960) wrote that "water supply was probably implicated in the conveyance of the infections". After the Witkoppen study, Richardson and Bokkenheuser (1963) wrote that "the poor quality of the water, particularly that drawn from surface wells, made it highly probable that drinking water was involved in the transmission of the infections". After the Komatipoort study, Richardson and Koornhof (1965) wrote that "the suspicion that the water supply is a factor in the transmission of salmonellosis is strengthened by the results of the present survey". Then came the shock of finding prevalences in Soweto as high as, or higher than, those reported from relatively impoverished rural areas with poor sanitation. Richardson, Koornhof and Hayden-Smith (1966) then wrote that "although it is reasonable to assume that water contamination plays some part in the transmission of these organisms, it does not appear to be the most important factor", and again that "water supplied to each house by the Johannesburg municipality was of good quality, yet it did not affect the incidence of salmonellosis and shigellosis". The complete turnabout came after the resurvey of Tlaseng in 1966, when Richardson and others (1968) concluded that "in the environment of the Bantu children the provision of high quality community water as the only sanitary measure was without effect on the prevalence of intestinal *Salmonella* and *Shigella* infections".

Other work in South Africa by the same authors has strongly implicated *Salmonella* contamination of meat and meat products in the transmission of salmonellosis (Prior and Badenhorst 1974; Richardson, Burnett and Koornhof 1968). The contention is indirectly supported by parasitological data, obtained during the 1966 survey at Tlaseng, showing that 16 percent of children had *Taenia* infections (compared with only 3 percent having *Ascaris*, 2 percent having *Trichuris*, and 2 percent having *Necator*), data that indicate the common consumption of undercooked meat.

## Occurrence and Survival in the Environment

Because of their very extensive nonhuman reservoir, salmonellae can be isolated frequently from a wide variety of environmental samples. Before the role of *E. coli* and *Campylobacter* in human diarrhea was recognized in the 1970s, the bacterial enteric pathogens of prime interest were the salmonellae, the shigellae, and *Vibrio cholerae*. Of these it is the salmonellae that are by far the most common in environmental samples, and so a great deal of work was done on the occurrence and survival of salmonellae in water and soils. Indeed, *Salmonella* became the favorite pathogen of the sanitary engineers and environmental microbiologists; whenever data on more than harmless indicator organisms were required, salmonellae would be studied. This situation has generated a large literature, second only to that on fecal indicators in the environment (see chapter 13), and only a small portion of it can be reviewed here. In the absence of specific information, it may generally be assumed that the survival, and the factors affecting survival, of *Salmonella* in the environment is similar to that described for fecal coliforms and *E. coli* in chapter 13.

### *In surface water*

Salmonellae will be found in surface waters wherever there are animal populations. Those workers who have failed to find them have probably not looked hard enough. This involves sampling large volumes of water, or using suspended swabs to sample flowing water for several days. The occurrence and survival of salmonellae in surface waters has been reviewed by Wray and Sojka (1977).

Salmonellae are found in rural streams receiving run-off from agricultural land and discharges of domestic and other effluents from villages and rural industries. Harbourne (1977) and Harbourne, Thomas

and Luery (1978) readily isolated salmonellae from swabs suspended in streams in North Yorkshire (England). Smith, Jones and Watson (1978) sampled rivers and streams running through agricultural land in Cheshire and Lancashire (England). Salmonellae were detected at 71 percent (10 of 14) of the sites investigated. Sewage effluent discharges were believed to be the major source of the salmonellae, but farms, a dairy, an abattoir, and gulls were also implicated. In one stream the concentration of salmonellae was 1,100 per 100 milliliters at an effluent discharge point and 23 per 100 milliliters at a point 0.5 kilometer downstream.

Davis (1979) studied stream water quality in a rural area north of Houston (Texas, USA). During low flow, salmonellae concentrations were up to 5,800 per 100 milliliters, and during storms the maximum concentration was 2,500 per 100 milliliters. Salmonellae were sometimes detected when fecal coliforms were absent. Dondero and others (1977) isolated salmonellae from 39 percent of 322 swabs suspended in six streams in central New York state (USA). Cherry and others (1972) found that 44 percent of samples collected from unpolluted mountain streams ("judged to be completely free from human, domestic animal and industrial pollution") in Georgia (USA) contained salmonellae. One "unpolluted" stream acquired salmonellae within 100 meters of its source. The authors suggest that some salmonellae may have an aquatic reservoir and that they may be "superior to coliforms as indexes of water quality". These suggestions, apart from being contradictory, have not been supported by subsequent work.

Drainage from farms raising animals by intensive methods are responsible for major contributions of salmonellae to rural streams (see, for instance, Miner, Fina and Piatt 1967). The *Salmonella* serotypes found in a particular water may reflect the special character of the wastes contaminating that water. *S. agona*, a serotype associated with fish meal used as a poultry and animal feed, was found in all water samples from a creek receiving poultry wastes in Georgia (USA; Cook, Champion and Ahearn 1974).

Salmonellae can travel considerable distances in streams and rivers, especially at low water temperatures. Spino (1966) showed that *S. saintpaul* and *S. thompson* traveled for at least 120 kilometers under the frozen surface of the Red River of the North from their source at the Fargo-Moorhead (North Dakota-Minnesota, USA) effluent discharge sites.

Salmonellae are also found in urban streams and stormwater run-off, although in the absence of waste discharges the concentrations may be lower than found in rural catchments. Geldreich and others (1968) isolated 4,500 *S. thompson* per 100 milliliters from a sample of urban stormwater in Cincinnati (Ohio, USA). Olivieri, Kawata and Krusé (1978) reported that salmonellae were nearly always present in samples of urban stream water and stormwater in Baltimore (Maryland, USA) and that geometric mean concentrations at various sites were between 6 and 140 per 100 milliliters.

The detection of salmonellae in waters with very low fecal indicator counts is not uncommon. Dutka and Bell (1973) studied the St. Lawrence river (Canada) and detected salmonellae in 24 percent (17 of 72) of samples containing less than 9 fecal coliforms per 100 milliliters. Samples that contained more than 1,000 fecal coliforms per 100 milliliters were 86 percent (6 of 7) positive for salmonellae. Geldreich and Bordner (1971) reviewed a variety of stream pollution data from the USA and found that salmonellae had been detected in 54 percent of samples containing 1–1,000 fecal coliforms per 100 milliliters and in 96 percent of samples containing more than 1,000 fecal coliforms per 100 milliliters.

Many attempts have been made to relate the presence of salmonellae in surface waters to the concentrations of fecal coliforms or other fecal indicator bacteria (see chapter 13). Although in general the chances of isolating salmonellae increase as water pollution (and therefore indicator densities) increase, there is no precise or generally applicable relationship. Indeed, when analyzing data from a particular location several workers have found that *Salmonella* concentrations are among the most difficult bacterial pathogens to predict on the basis of indicator concentrations (Davis 1979; Olivieri, Kawata and Krusé 1978). Gallagher and Spino (1968) reviewed water pollution data from several rivers in the USA and failed to show any fecal coliform density below which *Salmonella* isolation would be particularly unlikely. Smith, Twedt and Flanigan (1973) isolated salmonellae from the Huron and Saline rivers (Michigan, USA) at sites where fecal coliform concentrations were low and found that, probably due to technical difficulties in the laboratory, "the probability of *Salmonella* isolation decreased as the fecal coliform concentration increased".

Salmonellae are more likely to be found in bottom sediments than in the overlying waters. Hendricks (1971*a*) isolated salmonellae from 0.6 percent (1 of 195) of water samples and from 4.6 percent (9 of 195) of bottom sediment samples in a stretch of river (Georgia, USA) below a sewage effluent outfall. Van Donsel and Geldreich (1971) collected simultaneous sediment and water samples at a variety of lake and river sites. Forty-six percent of sediment samples, and only 8 percent of

water samples, contained salmonellae. The highest *Salmonella* concentration in sediment was 790 per 100 milliliters. Salmonellae were isolated from 19 percent of sediment samples when the fecal coliform concentration in the overlying water was between 1 and 200 per 100 milliliters, from 50 percent when between 201 and 2,000, and from 80 percent when the fecal coliform concentration in the water was over 2,000 per 100 milliliters.

The survival of salmonellae, especially *S. typhi*, in water has been investigated by several researchers over the past 80 years (see the appendixes to Feachem and others 1980). McFeters and others (1974) studied the survival of several species of enteric bacteria in membrane chambers suspended in well water at 9–13°C. Approximate $t_{90}$ values over 2 days were 80 hours for *S. paratyphi* A and *S. typhimurium*, 32 hours for *S. typhi*, and 19 hours for *S. paratyphi* B. Geldreich and others (1968) derived $t_{90}$ values over 14 days for *S. typhimurium* in storm water of 240 hours at 10°C and 160 hours at 20°C. At both temperatures the dieoff of *S. typhimurium* was similar to that of fecal coliforms and considerably more rapid than *Str. faecalis*.

Dutka and Kwan (1980) studied the death of *S. thompson* in membrane chambers suspended in Lake Ontario and Hamilton Bay (Canada) when water temperatures were 17–19°C. Over the first 3 days, $t_{90}$ values were 19–180 hours, and over the entire 28 days of the experiments average $t_{90}$ values were 122–224 hours. Death rates were considerably greater near the surface than at greater depth and were higher in less polluted water (Lake Ontario) than in more polluted water (Hamilton Bay). *S. thompson* dieoff was similar to *Str. faecalis* and faster than *E. coli*.

The range of death rates reported is large and there is considerable interserotype, and probably also intraserotype, variation in ability to survive in water. It has been suggested that these differences in survival may partly explain why some serotypes are common and others are very rare in human and animal infection (Enkiri and Alford 1971). Few comparative studies on different serotypes in water under identical conditions have been conducted, but the data of McFeters and others (1974) showed that *S. typhimurium*, a very common serotype, survived for longer than *S. typhi* and *S. paratyphi* B.

Overall survival times reported range from 1 to over 100 days, with typical $t_{90}$ values in the range of 20–200 hours. These results are similar to the fecal coliform $t_{90}$ data reviewed in chapter 13, and most comparative studies have found *Salmonella* survival to be similar to that of fecal coliforms (see, for instance, Smith, Twedt and Flanigan 1973). The factors affecting *Salmonella* survival are the same as those affecting fecal coliforms. Survival is greatly prolonged at lower temperatures and somewhat prolonged in darkness. Experimental data show that survival is prolonged in more polluted waters, but many experiments are done in biologically inactive polluted waters (autoclaved or filtered), or in chambers suspended in polluted waters, where the salmonellae are not exposed to predation and competition. Experiments in New Zealand, however, showed that *S. typhimurium* and *S. bovis-morbificans* survived for under 2 weeks ($t_{90}$ <56 hours) in clean water and for 12–16 weeks ($t_{90}$ = 340–450 hours) in unsterilized water containing 5 percent (weight per volume) sheep feces (Tannock and Smith 1971).

Growth of salmonellae in polluted water is possible but unlikely, although concentration and perhaps growth in bottom sediments is more likely. Hendricks (1972) showed that *S. senftenberg* grew in autoclaved river water collected downstream of a sewage effluent outfall at 30°C. No growth occurred at 20°C or 5°C or in autoclaved river water collected upstream from the same outfall (see also Hendricks 1971*b* and Hendricks and Morrison 1967).

Viewed with hindsight, the interest in low levels of salmonellae in surface waters, and their relationship to the concentrations of indicator bacteria, seems excessive. From the viewpoint of human public health, the greater importance of other bacterial agents of diarrhea, the frequent transmission of salmonelloses by contaminated animal products, and the high infective dose for salmonellae make the presence of low concentrations in surface water a matter of only minor concern in developed countries. From a veterinary viewpoint, it has been suggested that the contamination of rural streams by salmonellae may help to maintain infection among farm animals (Smith, Jones and Watson 1978). There are other more likely routes of transmission among farm animals, although stream pollution has undoubtedly contributed to some outbreak (George and others 1972; Williams 1975; Wray and Sojka 1977). In developing countries, surface water contamination by salmonellae must be seen in the light of the probable minor importance of *Salmonella* in pediatric diarrhea and of the many alternate transmission routes for salmonellae in areas of poverty and poor hygiene (see table 15-2 and the discussion of salmonellosis in South Africa and typhoid in Lesotho, above).

### *In groundwater*

*Salmonellae* are unlikely to be present in groundwater unless the water table is very shallow or fissured strata allow the direct flow of surface waters into an aquifer. Salmonellae have not been isolated from dual

Table 15-2. *Period and point prevalences of* Salmonella *and* Shigella *excretion by black schoolchildren in the Transvaal, Republic of South Africa*

| *Site* | *Year* | *No. of children* | *Age of children (years)* | *No. of surveys per year* | *Water supply (E. coli concentrations are per 100 milliliters of water)* | *Excreta disposal facilities* | *1 year period prevalence[a] (percent)* | | *Mean point prevalence (percent)* | | *Source* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Salmonella *excretion* | Shigella *excretion* | Salmonella *excretion* | Shigella *excretion* | |
| Tlaseng school, rural Western Transvaal | 1958–59 | 75 | 6–16 | 7 | Polluted shallow wells (2–900 *E. coli*) | None[b] | 36[c] | 25[c] | 6.3[d] | 4.2 | Bokkenheuser and Richardson (1960) |
| | 1966 | 92 | 7–16 | 4 | Borehole with reticulation (0 *E. coli*) | Pit latrines[b] | 13[c] | 20[c] | 3.8 | 5.5 | Richardson and others (1968) |
| School near Komatipoort on Mozambique border in rural Eastern Transvaal Lowveld | 1964 | 99 | 7 17 | 2 | Piped water (0 *E. coli*) River water (50–250 *E. coli*) and salmonellae) | Pit latrines[b] | 6 | 3 | 3.5 | 2.0 | Richardson and Koornhof (1965) |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| School at Witkoppen, a periurban area 25 kilometers from Johannesburg | 1961 | 75 | 7–18 | 8 | Protected well with hand-pump (0 *E. coli*)<br>Borehole with motor pump (0–4 *E. coli*)<br>Borehole with wind pump (0–>4 *E. coli*)<br>Open well (>4 *E. coli*) | Pit latrines[b] | 30 | 2.7 | 5.8 | 0.3 | Richardson and Bokkenheuser (1963) |
| Two schools in Soweto, a large African city near Johannesburg | 1964 | 55 | 7–18 | 8 | Chlorinated piped water (0 *E. coli*) | Waterborne sewerage | 36 | 9 | 5.9 | 1.1 | Richardson, Koornhof and Hayden-Smith (1966) |

a. The recorded annual period prevalence of an acute and readily transmissible infection, such as salmonellosis, rises markedly as the number of surveys per year increases. Therefore, the figures for Komatipoort, for instance, are not comparable with those from Witkoppen.

b. This information is not given in the original source but in the summary by Koornhof and others (1979).

c. For the reasons stated in note a, it is not possible to compare the period prevalences from 1958–59 (seven surveys) with those from 1966 (four surveys).

d. This figure is elevated due to an outbreak of *S. mobeni* infection that coincided with the survey in December 1958.

purpose wells in Israel used for groundwater recharge and abstraction (Goldshmid 1974). Salmonellae will be found in many open wells due to the drainage of contaminated surface waters down unprotected well shafts.

*In drinking water*

It is uncommon to isolate salmonellae from piped water supplies, whether treated or untreated, and their presence suggests a serious fault in the design or maintenance of the system. Raman and others (1979) found that 9 percent (3 of 33) of tap water samples in Aurangabad (Maharashtra, India) contained salmonellae due to leakage of polluted water into the distribution system.

*S. typhimurium* was isolated from 7 percent (5 of 74) of water samples from taps and reservoirs of the water supply system of Riverside (California, USA) during the major outbreak of salmonellosis in May–June 1965 (Boring, Martin and Elliot 1971). One composite sample examined quantitatively revealed 1.7 *S. typhimurium* and 0.14 *E. coli* per 100 milliliters. All *S. typhimurium* isolates examined were of phage type 10, as were the clinical isolates during the outbreak. The water sources were deep wells, and the water was delivered unchlorinated.

Schubert and Scheiber (1979) reported that salmonellae could frequently be isolated from piped drinking water in Togo, even in the absence of *E. coli* and coliforms. This was attributed to faulty wellheads and broken reservoir covers allowing access to rainbow lizards (*Agama agama*), which were often found to excrete salmonellae and sometimes few or no *E. coli* and coliforms. Keeping small animals out of water systems, and thus reducing the risk of contamination by salmonellae, can prove extremely difficult, especially in arid areas.

A large proportion of the population of the developing countries drink untreated and unprotected surface waters or open well waters that are often heavily polluted by feces (table 13-1). Salmonellae may be expected in these drinking water sources, although few data on their presence exist. Salmonellae will be far more common than shigellae (chapter 16) in unprotected water sources because, unlike shigellae, they are excreted by some pigs and by donkeys, goats, cattle, camels, rats, dogs, and other village animals whose excreta commonly pollute streams, ponds, and open wells. Gracey and others (1979) isolated salmonellae from 48 percent of water samples and from 63 percent of sediment samples collected from a river and canals in Jakarta (Indonesia) used as drinking water sources by the poorer inhabitants of the city.

The contamination of clean drinking water by *E. coli* while the water is stored in the home is well documented (chapter 13). No similar data exist for salmonellae, although such pollution may be expected and has been shown for clean water used by infected turkeys (Gauger and Greaves 1946).

*In seawater*

Salmonellae are found in estuarine and marine environments where there is fecal contamination from domestic or agricultural wastewater discharges or where there are large populations of water birds. The occurrence and survival of salmonellae in seawater have been reviewed by Buttiaux (1962).

Colwell and Kaper (1978) reported that the ratios of salmonellae to fecal coliforms in Chesapeake Bay (USA) ranged from 1:100 to 1:1000 and that there were up to 240 salmonellae per 100 milliliters in Baltimore Harbor (Maryland, USA). Salmonellae could not be isolated from Chesapeake Bay in the winter, only during April–November. In contrast, Carney, Carty and Colwell (1975) failed to isolate salmonellae in an extensive survey of microbiological pollution in a subestuary of Chesapeake Bay, despite occasionally elevated concentrations of fecal coliforms (up to 5,400 per 100 milliliters)

Goyal, Gerba and Melnick (1977, 1978) studied the occurrence of fecal indicator bacteria and salmonellae in the waters (salinities 1.0–2.2 percent) and bottom sediment of canals bordering Galveston Bay (Texas, USA). The canal networks are largely man-made, and holiday homes are located along their banks. Domestic wastes are discharged into the canals through septic tanks or small treatment plants, and the canals are greatly used for bathing, boating, skiing, and diving. Salmonellae were isolated from 47 percent (17 of 36) of sediment samples but from only 3 percent (1 of 36) of water samples. Salmonellae concentrations in sediment were between 0 and 150 per 100 milliliters.

The limited information on *Salmonella* survival in seawater indicates that it may be only slightly less than survival in fresh water. Since *E. coli* survival is very much shorter in seawater than in fresh water (chapter 13), it follows that *Salmonella* are more persistent in marine environments than are *E. coli*. Therefore *E. coli* are poor indicators of salmonellae, as they are of enteroviruses, in marine environments (see Petrilli and others 1979).

Jamieson, Madri and Claus (1976) added $1.5 \times 10^7$ *S. typhi* to samples of sterilized seawater, adjusted to salinities of 0.5, 2, and 3.5 percent, and stored them at 4, 25, and 37°C. Survival was inversely proportional to salinity and temperature. Maximum survival was for 7

days ($t_{90}$ = 23 hours) at 4°C and 0.5 percent salinity, and minimum survival was for 5 days ($t_{90}$ = 17 hours) at 37°C and 3.5 percent salinity. Survival of *E. coli* was shorter than that of *S. typhi* at all temperatures and salinities. Vasconcelos and Swartz (1976) compared the survival of *S. heidelberg* and *E. coli* in sterilized seawater at 14.5°C. After 6 days, the *E. coli* concentration had declined by 6 log units ($t_{90}$ = 24 hours), whereas the concentration of *S. heidelberg* was reduced by only 1.5 log units ($t_{90}$ = 6 hours).

*In feces and night soil*

In developing countries, pooled human feces are likely to contain *Salmonella* in areas where asymptomatic excretion of the organism is fairly common (see above). Similarly, pooled animal excreta are also likely to contain *Salmonella*. Screening night soil for *S. typhi* is potentially useful in epidemiological investigations of typhoid, and screening farm wastes for specific *Salmonella* serotypes can assist livestock and poultry hygiene.

Jordan (1926) studied the bacterial content of feces from typhoid patients and carriers (Chicago, USA); the feces were stored at room temperature in sealed cans. *S. typhi* survived for between 3 and 52 days. Desiccation was not a factor contributing to death, since the moisture content of the feces stored in this manner was still over 80 percent after 77 days. No multiplication of *S. typhi* in stored feces was observed.

Tannock and Smith (1972) reported that *S. typhimurium* survived for 6–18 weeks ($t_{90}$ = 150–450 hours) in sheep feces outdoors in New Zealand. Survival was longer on shaded than exposed sites and longer in summer than winter (presumably because of the bactericidal effect of freeze-thaw cycles in winter).

Berkowitz, Kraft and Finstein (1974) studied the survival of various *Salmonella* serotypes (*typhimurium*, *saintpaul*, *thompson*, and *infantis*) inoculated into samples of wet poultry excreta (80 percent water) and stored at various temperatures. Initial concentrations were $1.6 \times 10^5$–$2.4 \times 10^6$ *Salmonella* per gram. Overall persistence was usually less than 1 month; $t_{90}$ values (averaged over the first 3 log units' decline) averaged 184 hours at 9–12°C, 112 hours at 18–20°C, and 40 hours at 30°C. Growth occurred before decline in sixteen of twenty-three tests, with concentrations of *Salmonella* rising to a maximum of 1.4 log units above initial values. No clear pattern of differential survival among the four serotypes emerged. When samples were allowed to dry for 2 days (12 percent water after 2 days) $t_{90}$ values became 21 hours at 20°C. However, the organisms that survived the 2 days drying then survived for considerably longer than in the undried samples, with overall survival times being 148 days at 11°C. It may be concluded that storing undried excreta is more effective in eliminating *Salmonella* than promoting initial rapid drying.

These and other studies (listed in the appendixes of Feachem and others 1980) suggest $t_{90}$ values of 40–100 hours for *Salmonella* in feces or night soil in tropical climates (20–30°C).

*In sewage*

The monitoring of sewage for salmonellae in general, or for *S. typhi* in particular, is of practical public health value in epidemiological surveillance, investigating outbreaks of salmonellosis or typhoid, and, especially, in tracing typhoid carriers. The favored technique is the Moore's swab, an absorbant pad suspended in the flow for 2–3 days. If a pad of calcium alginate wool is used, it can be completely dissolved in sodium hexametaphosphate on return to the laboratory, thereby liberating all the entrapped bacteria. For more rapid results, a wipe swab can be used—the sewer wall is wiped and the swab immediately returned to the laboratory (Bokkenheuser 1964; Gell and others 1945; Harvey and Phillips 1955; Kelly, Clark and Coleman 1955; Moore, Perry and Chard 1952; Robinson 1958; Shearer and others 1959). Alternatively, various filtration methods have been devised for detecting salmonellae in large volumes of sewage collected at a single time. Hirn (1980) suggested that monitoring the effluent of food-processing plants for salmonellae was a useful contribution to the microbiological quality control of the processed food.

Reported concentrations of salmonellae in sewage vary considerably. Concentrations per 100 milliliters of 7–250 in India (Phirke 1974), 2–41 in South Africa (Grabow and Nupen 1972), 500 in Baltimore (Maryland, USA; Olivieri, Kawata and Kruse 1978), 8,000 in Houston (Texas, USA; Davis 1979), up to 2.3 in Finland (Hirn 1980), up to 7,240 in northwest England (Jones 1977), and 670 in Holland (Kampelmacher and van Noorle Jansen 1970) have been reported.

Several workers have recorded concentrations of salmonellae in sewage that are difficult to explain by analyzing known inputs from patients, carriers, farm wastes, or food-processing effluents. One such study in Holland concluded that salmonellae must be multiplying in the sewerage system (Kampelmacher and van Noorle Jansen 1976).

The concentration of salmonellae in sewage may fluctuate on a regular basis. McCoy (1977) reported that raw sewage in Hull (England) contained most

salmonellae (median concentration of 150–400 per 100 milliliters) during July–September and least during January–March. Yaziz and Lloyd (1979) studied the hourly fluctuations of *Salmonella* concentrations in raw sewage at Guildford (England). Peak concentrations occurred at 0900–1000 hours, 2 hours before the peak flow of sewage at midday. Samples of raw sewage collected during the morning peak over a 9-month period contained 20– > 1,800 (median 130) *Salmonella* per 100 milliliters at Guildford and 11–1,600 (median 170) *Salmonella* per 100 milliliters at Woking, a nearby town. *Salmonella* concentrations were higher in the cooler months than in the summer.

During the 1930s, a survey of sewage in Bandung (Java, Indonesia)—which then had a population of 200,000, of whom 40,000 were sewered—demonstrated the presence of typhoid bacilli in 62 out of 80 samples (Mom and Schaeffer 1940); *S. typhi* concentrations varied from less than 1,000 to 45,000 per 100 milliliters, with an average of 5,000 per 100 milliliters. Despite individual accounts of high levels of *Salmonella* in sewage, reported concentrations from developing countries are typically lower than from developed countries. This may reflect the greater input of effluents from food-processing plants in developed countries or it may be an artifact caused by differences in laboratory technique. Daniel and Lloyd (1980) reported geometric mean concentrations of salmonellae in two refugee camps in Bangladesh of only 7.1 and 7.7 per 100 milliliters, and they noted that these results could have been caused by the difficulty of selectively isolating salmonellae from sewage with a very high solids content (17,000 milligrams per liter).

There are few studies on *Salmonella* survival in sewage (see the appendixes of Feachem and others 1980), and most attention has focussed instead on survival in sludges and slurries (see below). $t_{90}$ values of 77–108 hours may be computed from the data of Green and Beard (1938) on *S. typhi* in raw sewage at 7–20°C. *S. tennessee* inoculated at $10^9$ per 100 milliters into strong Jerusalem (Israel) sewage (BOD 800–1200 milligrams per liter), survived for 22 days ($t_{90}$ = 60 hours) in outdoor storage tanks in summer (Bergner-Rabinowitz 1956). Gallagher and Spino (1968) reported the survival of *S. typhimurium* and fecal coliforms in various process waters and effluents at two sugar beet factories in the USA. $t_{90}$ values for *S. typhimurium* were generally over 72 hours and under 168 hours, and those for fecal coliforms were similar. In the absence of other information, it is reasonable to assume that *Salmonella* survival in sewage is similar to that of fecal coliforms (chapter 13), with $t_{90}$ values in warm climates of 20–100 hours.

*In sludge and slurry*

The common practice of applying sludge and slurry[2] to arable and pasture land has stimulated interest in the occurrence and survival of salmonellae in these materials. Sludges from sewage treatment works will almost always contain salmonellae. Reported cencentrations vary greatly and may fluctuate seasonally. In England, concentrations of salmonellae per 100 milliliters of raw sludge have been reported as around 70 (median value), with 7 percent of samples containing over 2,400 near Hull (McCoy 1977, 1979), 40–11,000 in Yorkshire (Fennell 1977), and 4,000–23,000 in the northwest (Jones, F. 1977). In Switzerland, over 90 percent of raw sludge samples contained *Salmonella* with maximum and mean concentrations of $10^6$ and $10^4$ per 100 milliliters respectively (Hess and Breer 1975; Obrist 1979).

Pike (1981) reviewed the data on salmonellae in sewage sludges in England and Wales. Geometric mean counts per 100 milliliters of raw sludge reported from various regions were between 8 and 1,400. Salmonellae were more numerous and more frequently isolated from sludge at treatment works serving communities of 10,000–100,000 people than at works serving larger or smaller communities. Common *Salmonella* serotypes in sludge were those (particularly, *agona*, *typhimurium*, *heidelberg*, *virchow*, *anatum*, and *hadar*) that infect a wide range of animals and man and not those host-adapted serotypes (such as *dublin* in cattle, *abortus-ovis* in sheep, *cholerae-suis* in pigs, and *gallinarum* and *pullorum* in poultry) that are a major cause of salmonelloses in farm animals in the UK. Interestingly, of the ten most common serotypes in sludge, six were among the ten most common serotypes in human infections and only four were among the ten most common isolates from farm animal infections. This suggests that sludge and its disposal on land do not play a major role in transmitting salmonellae among farm animals in England and Wales. Hall and Jones (1978) examined eight raw sludges from sewage treatment plants in England and found between 34 and 11,000 *Salmonella* per 100 milliliters. The most commonly isolated serotypes were, in decreasing order of frequency of isolation: *newhaw*, *heidelberg*, *saintpaul*,

2. In this context, "slurry" refers to the mixture of animal feces, urine, water, and sometimes some straw or other bedding produced at farms. This material may be rich in salmonellae; it has a solids content similar to that of sewage works sludge (1–10 percent), and it presents disposal problems very similar to those of sludge produced at a sewage works receiving primarily domestic, rather than industrial, sewage. The major distinction is between a sludge of primarily human origin and a slurry that contains the wastes of farm animals.

*typhimurium*, *paratyphi* B, *oranienberg*, and *kaapstad*. Only one of these serotypes, *typhimurium*, is a prominent cause of farm animal infection in Britain.

The frequency and concentration of salmonellae in animal slurries are typically lower than those in sewage works sludges. The lower frequency is partly due to the size of the contributing population; the herd of animals on a typical farm is much smaller than the number of people in a typical town. Jones and Mathews (1975) isolated *Salmonella* from 11 percent (20 of 187) of cattle slurry samples taken randomly throughout England and Wales; concentrations were up to 180 per 100 milliliters. Jones, Bew and Gammack (1975) isolated *Salmonella* from only 3 percent (2 of 63) of dairy factory sludge samples in England. Jones and others (1976) isolated *Salmonella* from 22 percent (12 of 54) of pig slurries in southern England at concentrations up to $2 \times 10^5$ per 100 milliliters. Kraft and others (1969) isolated *Salmonella* from the wastes of 50 percent (18 of 36) of poultry farms in New Jersey (USA) at concentrations of up to $3.4 \times 10^6$ per 100 grams. Monitoring animal slurries for salmonellae can assist the detection and control of infection in the herds (Jones and Hall 1975).

The literature on *Salmonella* survival in sludges and slurries is extensive (see the appendixes of Feachem and others 1980). Jones (1978), reviewing European literature, reports the survival of salmonellae in slurry as being between 13 and 286 days. Strauch (1978) reported the survival of various *Salmonella* serotypes in animal waste slurries of up to about 1 year at 8°C and up to about 6 months at 17°C. At both temperatures and in four different slurries, *S. enteritidis* and *S. cairo* consistently survived for longer than *S. gallinarum*, *S. typhimurium*, or *S. paratyphi* B. Braga (1964) found that *S. cholerae-suis* survived for longer than *S. typhimurium*, *S. typhi*, or *S. enteritidis* in sewage sludges at 6 and 28°C. Initial concentrations were $6 \times 10^5$ per 100 milliliters, and survival times ranged from 20–38 days at 6°C and 4–9 days at 28°C. Findlay (1972) recorded the survival of *S. dublin* in cattle slurry in northeastern England for 31–33 weeks in winter and 18–19 weeks in summer. Ekesbo (1979) found that *S. dublin* and *S. zanzibar* survived for at least 13 weeks in cattle slurry (8–9 percent solids) and settled cattle slurry (5–6 percent solids) at 5°C.

Jones (1976) experimented with various *Salmonella* serotypes inoculated into cattle slurry from a dairy. When *S. dublin* was inoculated at $10^6$ per milliliter into slurry (4.7 percent solids), $t_{90}$ values were 528 hours at 5°C and 10°C, 228 hours at 20°C, and 52 hours at 30°C. Overall survival times were 132 days at 5°C and 10°C, 57 days at 29°C, and 13 days at 30°C. Survival increased as the solids content of the slurry was increased from 1 to 5 percent, but did not increase further at solids contents above 5 percent. When the survivals of eight strains (four serotypes) were compared (at 10°C and 5.5 percent solids), overall survival times ranged from 90–140 days and appeared to be related to strain not to serotype. There was no evidence of multiplication. Jones suggested that the storage of slurry for 1 month prior to spreading on pasture, followed by a further month during which the pasture is not grazed, would be a minimal treatment regime for protecting cattle from *Salmonella* infection by this route.

Salmonellae can multiply vigorously in sterilized sludge or slurry, but under natural conditions they are strongly inhibited by the activity of other microflora (Findlay 1973; Jones, Smith and Bew 1977).

*In soil*

Salmonellae are likely to be found in soils that have been treated with sludges, slurries, or effluents. Reported survival times in several countries are: *S. dublin* for up to 12 weeks in the autumn in southern England (Taylor and Burrows 1971); *S. dublin* for 24 weeks in winter and 13 weeks in summer in northeast England (Findlay 1972); *S. typhimurium* for more than 35 weeks in England (Mair and Ross 1960); *S. typhimurium* for 4–10 weeks in New Zealand (Tannock and Smith 1972); *S. typhi* for up to 17 weeks during the rainy season in California (USA; Beard 1940); and *S. typhi* for 5–19 days in Michigan (USA; Mallman and Litsky 1951).

The long reported survival times represent residual contamination by a very small proportion of the large original inoculum of bacteria. Delage (1961) found that $10^6$ *S. abortus-ovis* in 1 gram of soil were reduced by 99.9 percent after 50 days but were still detectable for over 300 days.

Watson (1980) applied digested sludge, containing 25–30 *Salmonella* per 100 milliliters, to cabbage fields at a rate of 70 cubic meters per hectare. Survival times, during spring and summer in northern England (temperatures 3–32°C), were 42–49 days, with $t_{90}$ values of 336–528 hours. Chandler and Craven (1978) also reported about 8 weeks survival for *S. typhimurium* on dry soil in Australia.

Zibilske and Weaver (1978) studied the effect of soil type, moisture, and temperature on the survival of *S. typhimurium* applied to soil in cattle slurry or saline. Survival was inversely proportional to temperature but was not obviously related to soil type or delivery medium. At 22°C survival times ranged from 3 days ($t_{90}$ = 17 hours) to 84 days ($t_{90}$ = 483 hours).

Dazzo, Smith and Hubbell (1973) studied the survival of *S. enteritidis* in fine sand (soil moisture 10 percent), which was returned to the laboratory following different regimes of cattle slurry application. Samples were stored in darkness at 22°C. Death rate was inversely related to the rate at which the sand had received slurry. When no slurry was applied, overall survival was 8 weeks (from an initial concentration of $10^6$ *Salmonella* per gram of soil), and the $t_{90}$ was 254 hours. At the maximum rate of slurry application tested (508 cubic meters per hectare per week), the $t_{90}$ was 363 hours, and over 100 *Salmonella* per gram of soil remained after 8 weeks. The effect of slurry application in enhancing *Salmonella* survival in fine sand was not due to moisture content, since this was controlled at 10 percent throughout each experiment.

Bergner-Rabinowitz (1956) studied the survival of *S. tennessee* seeded into sewage (at about $10^8$ per 100 milliliters) and applied to soil of low organic content near Jerusalem (Israel). During the winter (air temperature 2–21°C), at the soil surface (soil moisture 8–39 percent) overall survival times averaged 46 days, whereas at a depth of 100 millimeters (soil moisture 19–30 percent) survival was for 70 days. Coliforms were still detectable in low concentrations after 74 days. During the summer (temperature not given) survival at the surface (soil moisture 4–23 percent) was 15 days, and at 150 millimeters depth (soil moisture 17–29 percent) 11 days. These experiments were all on uncultivated plots using a single application of sewage seeded with *S. tennessee*. In subsequent experiments in summer, *S. tennessee* in sewage was applied to plots growing sunflowers, and the plots were reirrigated with sewage not containing *Salmonella* every 6–8 days (in line with normal practice in Israel). Survival at the surface (soil moisture 4–31 percent) was increased to 23 days, and at a depth of 150 millimeters (soil moisture 18–30 percent) to 37 days.

These and other studies show that salmonellae can survive in soil for periods of many months when conditions are ideal. The factors most affecting survival are temperature, exposure to sunlight, and the moisture content, pH, and the organic content of the soil (Gerba, Wallis and Melnick 1975; Rudolfs, Falk and Ragotzkie 1950). In hot and sunny climates, maximum survival times may be around 2 months, with almost complete elimination after 2 weeks.

### *On pasture*

The role of sludge and slurry application to pasture in transmitting *Salmonella* infections remains controversial. Considerable evidence (reviewed by Williams 1975, 1979) has been accumulated in West Germany, Holland, and Switzerland (Hess and Breer 1975; Obrist 1979) to suggest that the use of inadequately treated sludges and slurries on pasture is a major factor in spreading bovine *Salmonella* infections. Some countries, Switzerland among them, have banned the use of unpasteurized sludge on pasture in summer (Williams 1979). In the UK, a general association between sludge application to pasture and salmonellosis in grazing animals has not been demonstrated. Wray and Sojka (1977) reviewed the subject and mention that, in addition to documented outbreaks in which pasture contamination has been implicated (for instance, Jack and Hepper 1969), the salmonellosis peak in cattle in many European countries occurs in late summer when the cattle are grazing and that the incidence declines markedly when cattle are brought indoors for the winter. This is scarcely evidence; the human salmonellosis peak occurs at the same time, and people neither graze contaminated pasture nor contract most of their infection from contaminated beef and milk. Further evidence against the importance of pasture contamination in cattle salmonellosis is the high infective dose of *Salmonella* (see the subsection on transmission, above, and the studies of Taylor 1973 and Taylor and Burrows 1971).

The interim guidelines produced by the Commission of the European Communities (Kelly 1978) recommended that, ideally, slurry should be applied only to arable land. If applied to pasture, it should receive a minimum of 60 days storage before spreading, and there should be a further interval of 30 days before grazing. Only healthy adult animals should be grazed on treated pasture.

Hess and Breer (1978) reported that salmonellae on grass treated with sludge could survive for up to 16 months in Switzerland. Most reported survival times are much shorter than this. In New Zealand, Josland (1951) reported survival of *S. typhimurium* on pasture of 12–24 weeks, and Tannock and Smith (1971) reported 6–14 weeks for *S. typhimurium* and 2–8 weeks for *S. bovis-morbificans*.

Taylor and Burrows (1971) applied cattle slurry (0.2 percent solids) containing $10^9$ *S. dublin* per 100 milliliters to pasture during September–December in southern England. *S. dublin* survived for 11 days on grass 75–150 millimeters above ground, 18 days on grass at ground level, and 12 weeks in the soil. In similar experiments, with slurry impregnated with $10^8$ *E. coli* per 100 milliliter, *E. coli* survival was only 3 days at 75–100 millimeters, 7 days at ground level, and 11 days in soil. Calves, given a choice of contaminated and

uncontaminated pasture, avoided the contaminated pasture for 2 days but then grazed it. When twelve calves were grazed on pasture that had been treated with slurry containing $10^8$ avirulent *S. dublin* per milliliter, eight showed evidence of infection. None of six calves was infected when grazed on pasture treated with slurry containing $10^5$ avirulent *S. dublin* per 100 milliliters.

*Salmonella* survival on pasture is considerably shorter than in soil and is shorter high up the blades of grass than near the ground. Heat, desiccation, and sunlight are all lethal factors, and survival on pasture in the tropics is unlikely to exceed 10 days.

### *On crops*

Several outbreaks of salmonellosis and typhoid have been linked to the contamination of vegetables and fruit by sewage or sludge (Geldreich and Bordner 1971). Gayler and others (1955) linked an outbreak of gastroenteritis due to *S. miami* with sliced watermelon and showed that the act of cutting a dirty watermelon could contaminate the inner fruit and that multiplication of *S. miami* in the melon could then occur. Sixty-eight percent of lettuce and 72 percent of fennel samples marketed in Bari (southern Italy) were contaminated with salmonellae (Ercolani 1976). Twenty-two percent of vegetables purchased in Holland contained salmonellae, and contamination was especially prevalent on tropical imports (Tamminga, Beumer and Kampelmacher 1978).

In Colorado (USA) 100 percent of settled sewage samples contained salmonellae (concentrations 43–360 per 100 milliliters), and 63 percent of irrigation water samples from streams receiving raw or treated sewage contained salmonellae (concentrations 1–360 per 100 milliliters) (Dunlop and Wang 1961). Only 1 out of 97 samples of irrigated turnips, cabbage, spinach, endive, and lettuce contained *Salmonella*. This was attributed to the inadequate laboratory methods, which could only detect high levels of contamination, and to the use of furrow irrigation on sandy soil in a dry climate, which minimized the survival of salmonellae.

*Salmonella* survival data (see the appendixes of Feachem and others 1980), reviewed by Geldreich and Bordner (1971), indicate up to 53 days on root crops, up to 40 days on leafy vegetables, up to 5 days on berries, and over 2 days on orchard crops. Two early studies of *S. typhi* on radish and lettuce showed survival for 10 days in sunny sites and for 31 days in the shade (Creel 1912) and for 21–37 days (Melick 1917). The latter study also showed that *S. typhi* became attached to leaves lying on contaminated soil and were not readily removed by washing. Lovett and Francis (1976) concluded that fecal coliforms were good indicators of *Salmonella* survival on vegetables, but that coliforms and fecal streptococci were not.

The use of waters containing sewage effluents to irrigate crops is commonplace in developing countries and will increase as growing urban populations produce more wastewater and create an increasing market for intensively cultivated crops. The risks of crop contamination are reduced by adopting drip, furrow, or subsurface irrigation methods instead of using spray or flood irrigation. A further safeguard is to discontinue irrigation or fertilization with fecal materials 2 weeks prior to harvesting; the survival of salmonellae on crop surfaces will be very much reduced by heat, sunlight, and low humidity. If these precautions are taken *Salmonella* survival on crops is unlikely to exceed 10 days in hot and arid climates.

### *In fish and shellfish*

Fish and shellfish from unpolluted waters, such as the open sea or mountain streams, do not contain salmonellae. Fish and shellfish living in waters polluted by waste discharges are commonly found to harbor *Salmonella* (Buttiaux 1962). Salmonellae are not known to cause disease in fish or shellfish, but they do cause temporary infection when the fish or shellfish are residing in waters containing salmonellae. Fish or shellfish can be decontaminated by placing them in clean water, but salmonellae seem to be eliminated rather more slowly than enteroviruses or *E. coli*.

Fish caught in deep-sea areas are free of *Salmonella* but may become contaminated prior to sale. Likely means of contamination are from ice made from polluted water, from storage in contaminated boxes or baskets, and from handling by infected packers or process workers. When salmonellae are isolated from fish intestines, contamination in polluted water is indicated; when salmonellae are isolated only from external surfaces, contamination during transport and handling is more probable. The contamination of fish meal used as a high protein food for poultry and pigs has been of great importance in the epidemiology of salmonelloses in Europe in recent years.

Geldreich and Clark (1966) studied the survival of various enteric bacteria in the sterilized intestinal contents of fish. In carp intestinal contents (pH = 7.3) at 10°C, *S. typhi*, *S. typhimurium*, *Shigella flexneri*, and fecal coliforms declined, whereas fecal streptococci grew slowly. At 20°C, fecal streptococci grew rapidly, fecal coliforms and *S. typhimurium* grew slowly, and *S. typhi* and *Sh. flexneri* declined. In bluegill intestinal

material (pH = 8.0) at 20°C, all species of bacteria tested grew rapidly except *S. typhi*.

In order to clarify the risks of salmonellosis associated with fish culture in sewage effluents, Heuschmann-Brunner (1974) experimented with carp and tench kept in water heavily contaminated by *S. enteritidis* and *S. typhimurium*. A few hours residence in heavily polluted water caused infection, and *Salmonella* spread rapidly along blood and lymph vessels throughout the body, including the musculature. Salmonellae were found most often, and for the longest time, in the digestive tract. At 9–12°C, *Salmonella* infection persisted in the tench gut for 60 days and in the carp gut for 68 days. In warmer water, infection persisted for longer—the reverse of what occurs with depurating shellfish, which cleanse themselves faster in warmer water because their rate of filter feeding increases. Lesions in the gut were produced by massive intralymphatic injection of salmonellae. Salmonellae isolated from fish several weeks after exposure were still pathogenic to mice. A great deal more research is required on the uptake and elimination of *Salmonella* by fish grown in sewage in developing and tropical countries.

Shellfish are often harvested from estuaries where polluted or potentially polluted waters flow into the sea. Salmonellae may be concentrated in the flesh of filter-feeding molluscs in the same manner as enteroviruses (chapter 9) and *E. coli* (chapter 13). Salmonellae are frequently isolated from shellfish harvested from contaminated waters and have given rise to major and many minor outbreaks of salmonelloses and enteric fevers (Buttiaux 1962). Depuration of shellfish, by placing them in clean water, seems to be less effective in removing *Salmonella* than in removing enteroviruses (chapter 9) and *E. coli* (chapter 13).

Janssen (1974) took oysters (*Crassostrea virginica*) from the Chesapeake Bay (USA) and kept them in an aquarium with salinity of 1.5 percent and water temperature of 20°C. Oysters were exposed to artificial seawater containing $2 \times 10^7$ *S. typhimurium* per 100 milliliters for 48 hours and then kept in clean water continually decontaminated by ultraviolet light. Oysters accumulated *S. typhimurium* up to a concentration of $2.8 \times 10^4$ per oyster and still contained 170 per oyster after 42 days in sterilized water. In other experiments in which the depuration water was only intermittently sterilized by ultraviolet light, oysters excreted *S. typhimurium* for 14 days and after 49 days still contained 6,000 per mollusc. These rates of *Salmonella* elimination by oysters in clean water are far slower than the reported rates for enteroviruses and *E. coli* elimination (DiGirolamo, Liston and Matches 1975; Hedstrom and Lycke 1964; Hoff and Becker 1969; Mitchell and others 1966).

Slanetz, Bartley and Stanley (1968) studied salmonellae in water and oysters in an estuary at Portsmouth (New Hampshire, USA). Water salinities were 1.1–2.5 percent and temperatures were 8–26°C. Salmonellae were readily isolated from water in which the coliform count was below the limit recommended for shellfish-growing waters (70 per 100 milliliters) and were on two occasions isolated from shellfish that met the coliform standard (less than 230 per 100 grams). On three occasions salmonellae were isolated from estuarine waters and shellfish containing no fecal coliforms per 100 milliliters and per 100 grams, respectively. Jegathesan and others (1976) studied 38 shellfish bought at markets in Malaysia. Three species were included: cockles (*Anadura granosa*) cultivated on muddy shores of the Malaysian west coast and mussels (*Modiolus senhaussi* and *M. metcalfi*) harvested from muddy and sandy shores, respectively. Cockles are commonly eaten half-boiled, whereas mussels are normally fried or baked. Two specimens contained *Salmonella*

### *In the air*

Salmonellae are likely to be aerosolized and dispersed from flush toilets, spray irrigation devices, and activated sludge plants in the same manner as *E. coli* (see chapter 13). Comparatively few data on airborne *Salmonella* are available because they have been seldom studied and because they are present sporadically or in low concentrations in feces and sewage and so are far more difficult to detect in the air than *E. coli*.

Newson (1972) added $10^{10}$–$10^{11}$ *S. typhimurium* to toilet bowls and flushed, producing an average aerosol of 132 *Salmonella* per cubic meter of air. The survival ability of *Salmonella* in splashes produced by toilet flushing was tested by studying 0.1 milliliter droplets of water and feces, containing $10^8$ *S. typhimurium*, on a laboratory bench. *Salmonella* survived for 12 days in both aqueous and fecal droplets, compared with 2–11 days for *E. coli* and 5 days for *Shigella sonnei* in parallel experiments. Katzenelson, Teltch and Shuval (1977) detected *S. infantis* 60 meters downwind from spray irrigators delivering raw sewage in Israel (see also Katzenelson and Teltch 1976). Hickey and Reist (1975) and Pereira and Benjaminson (1975) failed to detect *Salmonella* downwind of activated sludge tanks and grit chambers in the USA.

## Inactivation by Sewage Treatment Processes

The literature on *Salmonella* in sewage treatment plants is limited, both in coverage of the various technologies and in the quality of the experimental procedures. Some studies are reviewed below, and others are listed in the appendixes of Feachem and others (1980). Unless otherwise stated, it may be assumed that removal of *Salmonella* during sewage treatment processes is similar in nature and degree to removal of *E. coli* (chapter 13).

### *By primary and secondary sedimentation*

Mom and Schaeffer (1940) recorded that an Imhoff tank at Bandung (Indonesia) reduced an influent *S. typhi* concentration of 5,100 per 100 milliliters to an effluent concentration of 800 per 100 milliliters (an 84 percent reduction). Data on the removal of salmonellae by primary and secondary sedimentation at two treatment plants in southern England are given in tables 15-3 and 15-4, below, and are discussed in the section below on conventional treatment. Further literature is cited in the appendixes of Feachem and others (1980).

Although the number of studies reported is small, it may be concluded that *Salmonella* removal during sedimentation is similar to fecal coliform removal (chapter 13) and that salmonellae become concentrated in the sludge, as do all excreted viruses and bacteria undergoing sedimentation.

### *By septic tanks*

Salmonellae, like all enteric pathogens, are not normal residents of the healthy gut and are found only sporadically, and in widely varying concentrations, in sewage treatment systems serving individual buildings or small clusters of buildings. Surprisingly, a survey of seven septic tanks in the USA detected *Salmonella* in 15 percent (4 of 27) of effluent samples even though the average number of people per septic tank was only 3.9 (Small Scale Waste Management Project 1978).

Green and Beard (1938) simulated a septic tank in the laboratory and found that *S. typhi* in the supernatant liquor at 15–21°C declined at a $t_{90}$ rate of 52 hours. Howard, Lloyd and Webber (1975) installed an Oxfam sanitation unit (two large flexible septic tanks in series) in the yard of the cholera hospital in Dacca (Bangladesh). At a mean retention time of 8 days (4 days in each tank), and sewage temperatures of 22–32°C, the 2,900 *Salmonella* per 100 milliliters of influent were reduced by 98.8 percent ($t_{90}$ = 100 hours).

From the data on survival in sewage reviewed above and in the appendixes of Feachem and others (1980), it may be concluded that *Salmonella* removal in septic tanks is unlikely to exceed 95 percent and will be very much less in an overloaded or sludge-filled unit.

### *By conventional treatment*

Perhaps the most detailed study on *Salmonella* removal in conventional treatment plants is that on the trickling filter plant at Woking and the activated sludge plant at Guildford (both in the county of Surrey in southern England) reported by Yaziz and Lloyd (1979). The results are summarized in tables 15-3 and 15-4. Whereas the efficiencies of the two primary sedimentation systems are similar (79 percent removal at Woking, 73 percent at Guildford), the performance of the activated sludge plant is very much better in terms of *Salmonella* removal than that of the trickling filter plant. This difference results in a total removal of only 93 percent (1.2 log units) of *Salmonella* at Woking compared with 99.86 percent (2.9 log units) at Guildford. Judging from the extremely good $BOD_5$ removal performance of the two plants, they were in good operating order and were not overloaded. It is likely that the *Salmonella* removal rates reported are close to the maximum achievable by full scale plants employing conventional trickling filter and activated sludge processes. Many plants that are poorly designed or maintained, or that are overloaded, will achieve considerably lower removal rates.

Studies on four treatment plants in Holland showed overall *Salmonella* removals of 90 percent, 94 percent and 79 percent in three trickling filter plants and 99.4 percent in an activated sludge plant (Kampelmacher, Fonds and van Noorle Jansen 1977 and Kampelmacher and van Noorle Jansen 1970). Early laboratory studies on *S. typhi* showed a 90–99 percent reduction after 6 hours aeration in activated sludge and 95–99 percent reduction by trickling filters (Green and Beard 1938). Green and Beard (1938), like other early workers, were impressed by removal efficiencies of 95 percent and wrote that conventional treatment "may be expected to reduce greatly typhoid organisms present in sewage, and are, therefore, effective barriers for the protection of public health." This view, although widely held, was and is erroneous. It is only more recently that researchers have emphasized that, with high influent concentrations, 90–99 percent removal rates are poor and that, in any case, most of those *Salmonella* removed from the liquor are

Table 15-3. Salmonella *removal at the Woking trickling filter plant, UK*

| | Salmonella *per 100 milliliters* | | | *Percentage reduction* | | |
|---|---|---|---|---|---|---|
| *Sewage and process* | *Minimum* | *Maximum* | *Median* | *Minimum* | *Maximum* | *Mean* |
| Raw sewage[a] | 11 | 1600 | 170 | | | |
| Primary sedimentation[b] | | | | 29 | 99.00 | 79[c] |
| Settled sewage | 1 | 160 | 20 | | | |
| Trickling filters | | | | −92.3[d] | 91.7 | 68 |
| Trickling filters plus secondary sedimentation | | | | 86 | 99.6 | 92.3 |
| Final effluent[e] | 0 | 250 | 3 | | | |
| Total plant | | | | 64 | 100 | 93.0 |

*Source:* Adapted from Yaziz and Lloyd (1979).
a. $BOD_5$ = 194 milligrams per liter. Total flow = 7,600–12,100 cubic meters per day.
b. 6–7 hours' detention.
c. *Salmonella* removal correlated with suspended solids removals (r = 0.82).
d. Increase of 92.3 percent.
e. $BOD_5$ = 7 milligrams per liter.

concentrated in the sludge, which then presents its own treatment and disposal problems.

Kabler (1959) reviewed several studies on *S. typhi* removal by conventional treatment. These, plus other studies mentioned above and in the appendixes of Feachem and others (1980), indicate removal by trickling filter plants of 75–95 percent and by activated sludge plants of 90–99.9 percent. In other words, *Salmonella* removal by these processes is similar to the removal of fecal coliforms (chapter 13).

### *By oxidation ditch*

Will, Diesch and Pomeroy (1973) studied *S. typhimurium* in a 1:10 scale model oxidation ditch treating cattle slurry (0.5–1.0 percent solids). Maximum survival times were 17 days under simulated summer conditions (20°C) and 47 days under simulated winter conditions (2°C). When oxidation ditch effluent was held in a settling chamber, *S. typhimurium* survived for 66 days in the liquid layer and 87 days in the sludge layer at 2–3°C.

Oxidation ditches are commonly used in Holland to treat the sewage of small communities, and two were studied by Kampelmacher and van Noorle Jansen (1973). *Salmonella* concentrations per 100 milliliters ranged from 23 to 2,400 in the influent and from 0 to 350 in the effluent. *Salmonella* reductions of 90–99 percent were recorded. Kampelmacher and van Noorle Jansen (1971) also studied an oxidation ditch treating pig wastes in Holland. Concentrations of *Salmonella* per

Table 15-4. Salmonella *removal at the Guildford activated sludge plant, UK*

| | Salmonella *per 100 milliliters* | | | *Percentage removal* | | |
|---|---|---|---|---|---|---|
| *Sewage and process* | *Minimum* | *Maximum* | *Median* | *Minimum* | *Maximum* | *Mean* |
| Raw sewage[a] | 20 | >1,800 | 130 | | | |
| Primary sedimentation[b] | | | | 35 | 96.9 | 73[c] |
| Settled sewage | 7 | 250 | 35 | | | |
| Activated sludge[d] plus secondary sedimentation | | | | 93.6 | 100 | 98.7 |
| Final effluent[e] | 0 | 1.7 | 0.1 | | | |
| Total plant | | | | 98.7 | 100 | 99.86 |

*Source:* Adapted from Yaziz and Lloyd (1979).
a. $BOD_5$ = 259 milligrams per liter. Total flow = 7,600–22,800 cubic meters per day.
b. 6–7 hours' detention.
c. *Salmonella* removal correlated with suspended solids removal (r = 0.65).
d. 6–9 hours' detention in the activated sludge tanks.
e. $BOD_5$ = 8 milligrams per liter.

100 milliliters were 33–1,600 in the influent, 3–75 in the ditch, and 1–12 in the effluent.

### *By waste stabilization ponds*

Joshi, Parhad and Rao (1972) studied bacterial removal in a series of three ponds, with total retention time of 7 days, near Nagpur (India). *Salmonella* concentrations in the influent were 4–540 per 100 milliliters, and none was detected in the effluent. The reductions of coliforms, *E. coli*, and fecal streptococci in the same ponds were in the range 99–99.9999 percent. In a subsequent study (Joshi, Parhad and Rao 1973) on two ponds with 12 days total retention in Nagpur, *Salmonella* were present in the influent (3–100 per 100 milliliters) and in all effluent samples (qualitative determinations only). Reductions of indicator bacteria were only 43–98 percent. The striking difference in performance between the two sets of ponds was attributed to short-circuiting and poorly designed interpond connections in the second set of ponds. Two ponds in series in Lima (Peru), with total retention of 37 days and temperatures of 18–27°C, yielded salmonellae (mainly *paratyphi* B, *derby* and *newport*) in all influent and effluent samples (Yánez 1980). This must reflect poor pond design and major short-circuiting.

As far as is known, the processes affecting salmonellae removal in ponds are the same as those determining the removal of indicator bacteria (chapter 13). One study suggested that *Salmonella* death rates in ponds were similar to those of *E. coli* (Davis and Gloyna 1972). More commonly it has been found that *Salmonella* reduction is significantly less than that of coliforms, *E. coli*, or fecal streptococci in the same ponds (table 13-3; Coetzee and Fourie 1965; Walker, Carbonnelle and Leclerc 1977). The removal rate for salmonellae in ponds is very temperature dependent: higher removal is obtained in summer than in winter (for instance, Slanetz and others 1970), and ponds in the tropics remove salmonellae more effectively than those in temperate climates.

### *By tertiary treatment*

The realization that salmonellae, like all excreted viruses and bacteria, may be present in moderately high concentrations in the secondary effluents of conventional sewage treatment plants has stimulated some research into *Salmonella* removal by tertiary processes. In the absence of other information it may be assumed that *Salmonella* behave like fecal coliforms during tertiary treatment (chapter 13).

LAGOONING. Lagooning of secondary effluents will remove salmonellae if retention times are long enough. Removal will be greatly enhanced at warmer temperatures. The secondary effluent from an activated sludge plant in London (England) was held in three lagoons in series with a total retention time of 17 days (Metropolitan Water Board 1963–64). The lagoon influent contained 0.2–29 salmonellae per 100 milliliters, while the effluent contained 0–0.2 per 100 milliliters.

DISINFECTION. Brezenski, Russomanno and DeFalco (1965) studied the effluents and receiving waters of four sewage treatment plants discharging into Raritan Bay (New York–New Jersey, USA). The treatment plants incorporated primary treatment (sedimentation) and chlorination (0.25–2.5 milligrams per liter of combined chlorine residual). When effluents were chlorinated, no salmonellae were isolated from the effluents or receiving waters. When chlorination was suspended for 1 week, salmonellae were recovered from two of the effluents and from the receiving waters.

Kampelmacher, Fonds and van Noorle Jansen (1977) studied three sewage treatment plants in Holland discharging effluents into lakes used for recreation during the summer. Plant 1 had trickling filters followed by chlorination (6 milligrams per liter chlorine added); plant 2 had trickling filters, aeration, $FeCl_3$ addition for phosphate removal, and chlorination (2 milligrams per liter chlorine added); and plant 3 had activated sludge and chlorination (2 milligrams per liter of chlorine added). Reductions of *Salmonella* by biological treatment (influent compared with effluent prior to chlorination) were 94 percent in plant 1, 79 percent in plant 2, and 99.4 percent in plant 3. *Salmonella* concentrations per 100 milliliters were $2->10^4$ in influents, $0–10^4$ before chlorination, $0–10^3$ after chlorination, and $0–10^3$ in the receiving lake. Ninety-two percent (68 of 74) of samples contained *Salmonella* before chlorination, whereas only 17 percent (9 of 52) were positive afterward.

Schiemann, Brodsky and Ciebin (1978) compared three methods of disinfection of an activated sludge plant effluent in Ontario (Canada) with respect to their ability to remove wild *Salmonella*. *Salmonella* were recovered from 89 percent of primary effluent samples, 73 percent of secondary effluent samples, 13 percent of chlorine dioxide treated effluent samples, 8 percent of ozonated effluent samples, and 0 percent of chlorinated effluent samples.

Oliver and Carey (1976) reviewed data showing that *S. typhi* was inactivated by chlorine and ultraviolet light to the same degree as *E. coli* but was considerably

more resistant to ozone. This finding was not supported by Burleson, Murray and Pollard (1975), who found that *S. typhimurium* and *E. coli* had a similar response to ozone, both in saline and secondary effluent.

LAND TREATMENT. Near Phoenix (Arizona, USA) secondary effluent from an activated sludge plant was treated by intermittent flooding onto soil basins (Gilbert and others 1976). Secondary effluent contained, on average, 21 *Salmonella* per 100 milliliters, and no *Salmonella* were detected from ground water 9 meters under the site. The *Salmonella* had been removed by percolation of wastewater through 1 meter of fine loamy sand and 8 meters of sand and gravel.

## Inactivation by Night Soil and Sludge Treatment Processes

The realization that 70–99 percent of salmonellae entering a sewage treatment plant are concentrated in the sludge, and the widespread use of sludge in agriculture, have stimulated an increasing amount of research on the fate of salmonellae during sludge treatment. More research is urgently needed on salmonellae in simple sludge treatment processes (storing, drying, and composting) in warm climates and on the fate of salmonellae in night soil treatment and disposal systems in developing countries.

### *By pit latrines*

Galvagno and Calderini (1908) reported *S. typhi* survival in pit latrines for 15–30 days. Survival may be estimated from reports on survival in feces, night soil, sludge, and slurry (see above and the appendixes of Feachem and others 1980).

### *By storage*

Sludges stored for long periods will normally be free from *Salmonella*. In Yorkshire (England) lagooned raw sludges over 1 year old were negative or gave low counts commensurate with possible recontamination (Fennell 1977). Jones, P. (1977) found *Salmonella* in 50 percent of lagooned sludge samples less than 2 years old, but in no samples more than 2 years old. Jones (1975) reported that *S. dublin* underwent no loss of virulence when stored in cattle slurry for 1 month at 10°C.

### *By anaerobic digestion*

Pike (1981) reviewed data on *Salmonella* removal by sludge digestion at various sewage treatment plants in England and Wales. Reductions in *Salmonella* concentrations were between 16 and 98 percent. Two mesophilic digesters in Yorkshire (England), with mean retention times of 30 days, were receiving on average 5,900 and 2,500 *Salmonella* per 100 milliliters and putting out on average 3,600 and 37 *Salmonella* per 100 milliliters—thus achieving very different removal efficiencies of 39 and 98.5 percent, respectively (Fennell 1977). Stokes and others (1945) found that *S. typhimurium* could be detected in sludge after 45 days anaerobic digestion at 26°C.

Obrist (1979) reported that approximately 70 percent of the 2 million cubic meters of municipal sewage sludge produced annually in Switzerland is applied to land, and of this 52 percent is applied to pasture and forage crops. Raw sludge contained *Salmonella* in 91 percent of samples, with maximum and mean concentrations being $10^6$ and $10^4$ per 100 milliliters respectively. Digested sludge contained *Salmonella* in 81 percent of samples, with maximum and mean concentrations being $10^5$ and $10^2$ per 100 milliliters respectively.

Dudley and others (1980) studied the bacterial content of digested sludges from three sewage treatment plants in the southern USA. *Salmonella* concentrations were 200–2,400 per 100 milliliters. Cooke, Thackston and Malaney (1978) studied *Salmonella* removal by three anaerobic mesophilic digesters at sewage treatment plants near Nashville (Tennessee, USA). Digester 1 had a mean retention time of 9 days, operated at 34°C, and reduced *Salmonella* concentration by 98 percent. Digester 2 had a retention time of 50 days, operated at 37°C, and reduced *Salmonella* concentrations by 99.4 percent. Digester 3 had a retention time of 38 days, operated at 36°C, and removed 99.9 percent of salmonellae.

Mom and Schaeffer (1940) studied sludge digestion in an Imhoff tank in Bandung (Indonesia). Raw sludge contained 0–2,400 *S. typhi* per 100 milliliters; after 30 days digestion at 27°C, the concentration was 0–2,200 per 100 milliliters.

Clearly, mesophilic digestion produces a sludge that may still retain a considerable population of *Salmonella*. Thermophilic anaerobic digestion at around 50°C will certainly eliminate *Salmonella* if operated as a batch process with a retention time of 5 days or more. Under continuous feed *Salmonella* elimination can never be guaranteed, although concentrations in the digested sludge should be very low.

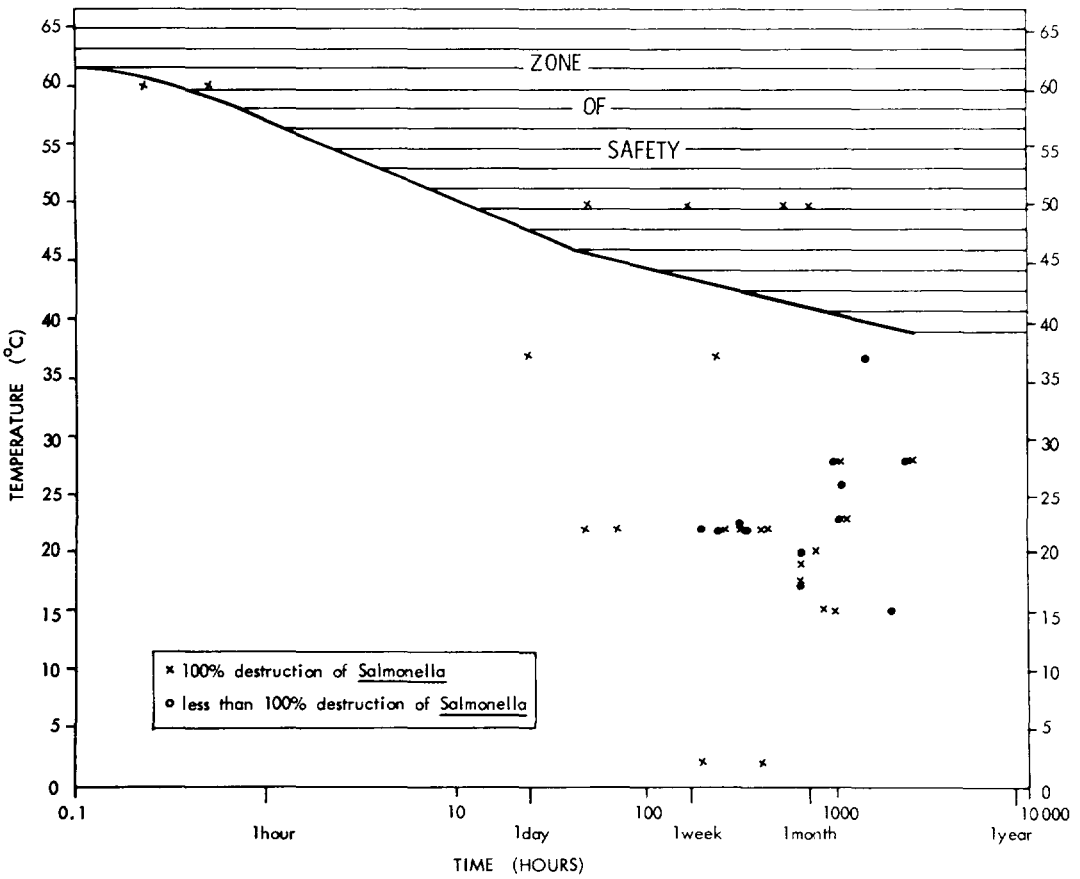


Figure 15-2. *The influence of time and temperature on salmonellae.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

*By aerobic digestion*

Thermophilic aerobic digestion (wet composting) is similar to composting in its effect upon enteric bacteria. If temperatures are maintained at the required level for the required time (figure 15-2) throughout the sludge mass, total destruction of *Salmonella* will occur. Smith, Young and Dean (1975) reported reductions in *Salmonella* of 99.8 percent in one aerobic digester (temperature 56°C, retention 4 days) and 100 percent in another (temperature 49°C, retention 4 days).

Aeration of stored slurry is used on some farms to promote bacterial dieoff. Willinger and Thiemann (1978) found that *S. typhimurium* survived for 6–12 days in continuously aerated cattle slurry in Austria. Average ambient temperature was 3–24°C, whereas the temperature in the aerated slurry was 29–35°C. *E. coli* survival in the same experiments was 9–12 days.

*By drying*

Pike (1981) reported that raw and aerobically digested sludges on drying beds in England maintained their levels of *Salmonella* contamination for up to 85 days. Stokes and others (1945) inoculated sludge (5.8 percent solids) with $2.5 \times 10^9$ *S. paratyphi* B per 100 milliliters and found that they were detectable after 27 days (solids content 36 percent), but not after 41 days (solids content 43 percent), on sludge drying beds in England during the summer. In similar experiments conducted during December–June with *S. typhimurium*, the bacteria could be detected after 180 days on the drying beds, by which time the solids content had risen to 86 percent.

*By heating*

All high temperature processes, such as pasteurization, wet oxidation, and the Porteous process, will eliminate *Salmonella* from sludge. Pasteurization is commonly used in Switzerland to treat digested or aerobically stabilized sludges prior to land application (Obrist 1979). Sludge is heated to 70°C for 30 minutes by steam injection. Hess and Breer (1975) reported that pasteurized sludges from five treatment plants

contained less than 10 Enterobacteriaceae per gram in 98–100 percent of samples.

In many situations, and especially in developing countries, the more practical and appropriate technologies for harnessing heat to destroy pathogens in sludge are aerobic thermophilic digestion (see above) and composting (see below). The time-temperature requirements for the destruction of salmonellae are shown in figure 15-2, and it may be seen that 1 hour at 60°C, 1 day at 50°C, and 1 week at 45°C are lethal combinations.

*By composting*

In laboratory experiments on composting poultry excreta, *S. typhimurium* were eliminated after 19 hours of composting during which the temperature rose to 64°C at hour 10 (Platz 1978). Salmonellae were more readily eliminated by composting when inoculated into the compost than when held in sealed glass ampoules within the compost, and this indicates that factors other than heat were contributing to bacterial death. In both cases *Salmonella* could not be detected after temperatures had risen to about 62°C.

Wiley and Westerberg (1969) determined that *S. newport* in nutrient broth was destroyed in 40 minutes at 60°C and in 30 minutes at 65°C. Dewatered primary sludge, containing $2.4 \times 10^6$ *S. newport* per gram, was then treated for 5 days in a continuously mixed, forced air, laboratory composter containing 1.1 cubic meters of sludge. The temperature within the composter was 60–76°C. *S. newport* was not detectable in the sludge after 25 hours of composting.

Savage, Chase and MacMillan (1973) experimented with various regimes for composting pig wastes (a combination of uneaten garbage and pig feces) in New Jersey (USA). In a windrow comprising 36 tons of pig waste that was turned twice per week, *Salmonella* concentrations rose from day 0 (temperature within windrow 36°C) to day 40 (temperature 48°C) and then declined to negligible levels by day 187 (temperature 68°C). In another windrow, comprising 36 tons of pig waste plus 1.4 tons of straw, turned 20 times per week, temperatures rose to 72°C within 10 days and remained above 60°C for 20 days. Coliforms, and presumably also salmonellae, were eliminated within 14 days.

The effect on enteroviruses and coliforms of the composting experiments at Beltsville (Maryland, USA) are described in chapters 9 and 13. In windrows of sludge and woodchips turned daily, salmonellae grew at first but were eliminated after 14 days. In piles of sludge and woodchips subjected to forced aeration, salmonellae again increased initially but were undetectable after 10 days (Burge, Cramer and Epstein 1978; Kawata, Cramer and Burge 1977). The importance of management and process control are illustrated by the poor *Salmonella* removal properties of a digested sludge plus sawdust composting plant at El Paso (Texas, USA) reported by Reeves (1959).

Samples of compost from Vietnamese double-vault composting toilets (retention times 6–7 weeks) have been found not to contain *Salmonella* (Nimpuno, personal communication). These results must be treated with caution, however, because they may result from studies of toilets operating under controlled, experimental conditions.

To eliminate salmonellae, all parts of the composting mass have to be brought to a warm enough temperature for a long enough time (figure 15-2). This generally requires the presence of a carbon source (refuse, straw, or woodchips), careful moisture control, and a supply of oxygen throughout the mass provided by turning or forced aeration.

*By coagulation and vacuum filtration*

Sludge coagulation followed by vacuum filtration is commonly used to dewater sludges in some developed countries. Kampelmacher and van Noorle Jansen (1972) studied three treatment plants in Holland; two plants added lime and ferrous sulfate prior to vacuum filtration, whereas the third used lime and ferric chloride. The solids contents were 4–10 percent and 25–30 percent, respectively, before and after dewatering. At the three plants raw sludge contained salmonellae in 59, 65, and 100 percent of samples, whereas dewatered sludge contained salmonellae in only 5, 5, and 14 percent of samples. The bactericidal properties of this process are due to the addition of coagulants and especially to their action of raising the pH.

*By lime treatment*

Any sludge treatment process involving the addition of lime is likely to produce a sludge free from salmonellae. Pike (1981) reviewed sludge treatment data for England and Wales and found that lime treatment was highly effective in removing *Salmonella*.

*By irradiation*

Experiments in the Federal Republic of Germany showed that $10^2$–$10^3$ wild *Salmonella* in 100 milliliters of raw sludge could be reduced to zero by the

application of 3 kilogray (Lessel and Suess 1978). Hess and Breer (1975) concluded that a dose of 3 kilogray has an effect on Enterobacteriaceae in sludge similar to heating to 70°C for 30 minutes. *Salmonella* in sludge and in composted sludge (60 percent solids) were reduced by approximately 1 log unit for each 300 gray of ionizing radiation applied (Brandon, Burge and Enkiri 1977). Similarly, White (1979) reported inactivation of coliforms at a rate of 1 log unit per 200 gray at 20°C and inactivation of *Salmonella* at a rate of 1 log unit per 300 gray at 23°C.

It is clear that the radiation doses necessary to eliminate *Salmonella* are similar to those required for other Gram-negative enteric bacteria (Osborn and Hattingh 1978) and are about one-tenth of the doses required to inactivate enteroviruses (2.5–5 kilogray for a 1 log unit reduction—see chapter 9). Therefore, ionizing radiation treatment designed to inactivate viruses will certainly eliminate *Salmonella*.

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# 16

# *Shigella* and Shigellosis

DYSENTERY, the frequent passing of bloody stools, has been recognized and feared throughout history as a serious and sometimes fatal condition. One major cause of dysentery is infection by members of the bacterial genus *Shigella* (the other major cause is infection by the protozoon *Entamoeba histolytica*—described in chapter 20).

## Description of Pathogen and Disease

Shigellae, and the infections they cause in man, are well documented in many countries. Together with *Salmonella* species (chapter 15) and *Vibrio cholerae* (chapter 17), they are the classic bacterial agents of intestinal infection.

### *Identification*

Shigellosis (bacillary dysentery) is an acute diarrheal disease caused by bacteria of the genus *Shigella*. The disease, which primarily involves the large intestine, may be asymptomatic or may have symptoms ranging from mild diarrhea to a severe disease accompanied by fever, vomiting, cramps, and tenesmus, with blood, mucus, and pus in the stools. The typical case is of short duration (about 4 days), but in exceptional cases the symptoms may last for up to 2 weeks. The severity of the illness and the mortality rate depend on the nutritional state and age of the patient, on the serotype of the organism, and on the infecting dose.

The severe form of the disease, bacillary dysentery, is often due to *Shigella dysenteriae*. Disease due to type I (Shiga's bacillus) is particularly serious. Mortality of untreated cases of bacillary dysentery may be as high as 25 percent but is usually much lower. Diagnosis is by isolation of the bacteria from feces or rectal swabs. The presence of many pus cells in the stool is highly suggestive of this diagnosis.

### *Occurrence*

Shigellosis has a worldwide distribution, with the highest incidence in communities where hygiene is poor. Children aged 1–4 years are the most affected, and 60 percent of cases and most fatalities are children under 10 years of age.

The different species of *Shigella* vary in their relative importance in different parts of the world. In Asia, South America, and Africa, *Shigella dysenteriae* is often responsible for severe disease, but all species are common. Shigellosis in the developed countries is most commonly caused by *Shigella sonnei*. In England and Wales 43,285 cases were reported in 1960 and 10,765 cases in 1970. At present some 98 percent of the infections in England and Wales are caused by *Sh. sonnei*, and the majority of known cases of infection with *Sh. flexneri*, *Sh. boydii*, or *Sh. dysenteriae* are persons recently returned from developing countries.

All types of shigellae are found in areas of inadequate sanitation and poverty; *Sh. dysenteriae*, *Sh. flexneri*, and *Sh. boydii* are the most frequently identified, and *Sh. sonnei* infections are relatively rare. As hygiene improves, *Sh. sonnei* becomes the dominant species, *Sh. flexneri* the next most common, and *Sh. boydii* and *Sh. dysenteriae* become rare. When conditions of hygiene degenerate, such as with an army in the field, infections with species other than *Sh. sonnei* again become common.

### *Infectious agent*

Shigellae are Gram-negative, nonmotile rods belonging to the family Enterobacteriaceae and closely resembling *Escherichia coli* and *Salmonella* (figures 13-1a and 15-1). Four major serological groups have been described, with some forty serotypes making up these

groups. The group and type antigens are all cell-wall antigens, but group antigens are difficult to demonstrate.

- group A, *Sh. dysenteriae,* includes at least ten serotypes, with three others provisionally recognized. Phage-typing systems are also used.
- group B, *Sh. flexneri,* includes nine serotypes that are related not only by a common group antigen but also by various shared type antigens. Phage-typing systems are also used.
- group C, *Sh. boydii,* includes fifteen serotypes and two additional, provisional serotypes. Phage-typing systems are also used.
- group D, *Sh. sonnei,* includes only one serotype, and colicin-typing and phage-typing are used to subdivide the group.

*Reservoir*

Shigellae have no natural hosts other than the higher primates. Although experimental infections can be produced in other primates, man is the only effective source of infection. Unlike *Salmonella* and *Escherichia* infections, animal feces are not a source of inocula, though animals may become contaminated by the ingestion of human feces.

*Transmission*

Shigellae are transmitted from man to man, from ill persons, healthy convalescents, or symptomless carriers to susceptible persons. The organisms, which are excreted in the feces, are usually transmitted by the direct fecal-oral route. Infected persons with diarrhea typically excrete $10^5$–$10^9$ shigellae per gram of wet feces, while symptomless carriers may excrete $10^2$–$10^6$ per gram (Dale and Mata 1968; Thomson 1955). Food may be contaminated through the contaminated fingers of patients or carriers. Foodborne and waterborne outbreaks occur. However, contamination of the environment (such as seats, door handles, and water-flushing devices in toilets) by infected feces and their transfer to the mouth seems to be the usual mode of infection. Transfer of shigellae by flies breeding on feces has been of crucial importance in some outbreaks.

The infective dose for *Shigella* is reported to be lower than for the other main diarrhea-causing bacterial pathogens (*Salmonella, Vibrio cholerae,* and *E. coli*). The median infective dose ($ID_{50}$) for *Shigella* may be around $10^4$ in healthy adults; for the other bacterial pathogens listed above, it is $10^7$ or higher. Dupont and others (1972) found that a dose of $10^4$ *Sh. flexneri* produced disease (oral temperature $\geqslant 37.8°C$, with four or more watery stools per 24 hours) in 59 percent of eighty-eight adult male volunteers, whereas a dose of 180 produced disease in 22 percent of thirty-six volunteers. Levine and others (1973) induced disease (oral temperature $\geqslant 37.8°C$, with three or more watery stools per 24 hours) in one out of ten adult male volunteers with only 10 virulent *Sh. dysenteriae* type 1. By contrast, Shaughnessy and others (1946) required massive doses of *Sh. flexneri* ($10^8$ organisms) to induce disease in volunteers who had previously ingested 2 grams of sodium bicarbonate to lower their gastric acidity.

*Incubation period*

The incubation period ranges from 36–72 hours; frank dysentery usually appears within 2 days.

*Period of communicability*

Patients recovering from an acute attack of shigellosis may continue to excrete bacilli in their stools. In general, this excretion lasts for only a week or so, but a small proportion become persistent carriers. DuPont and others (1970) found that 75 percent of 542 children who contracted shigellosis at an institution for the mentally retarded in New York (USA) excreted *Shigella* for less than 1 month, but that 7 percent continued to excrete the organism for over a year. There is also evidence of the presence of shigellae in completely symptomless carriers, and carriage for periods of years may be common under conditions of poor hygiene. In this context the carriage of shigellae for long periods by breast-fed infants should be noted. In such infants overt disease is seldom seen until breast feeding ceases.

*Resistance*

Children are especially susceptible, and natural resistance to *Shigella* infection has not been reported. Individuals in closed communities may have numerous repeated infections with a single serological type of *Shigella.* Such immunity as does occur is probably group specific and may involve the local production of short-lived antibodies in the colonic mucosa.

*Epidemiology*

Shigellosis is endemic and common in almost all communities where living standards are low and water and excreta disposal facilities inadequate. It may also be endemic in some institutions, such as schools and

geriatric wards, where poor hygiene occurs. Children, especially weanlings, suffer the highest incidence of infection and mortality, and malnourished children are especially vulnerable.

*Shigella* infections among poor people in developing countries are often very common. Over a 1-year period in an Egyptian village, for instance, 51 percent of eighty-two children between 6 months and 5 years old had one episode, 17 percent had two episodes, and 5 percent had three or more episodes (Higgins and Floyd 1955). Several studies have shown that up to about 18 percent of young children in poor communities may be excreting *Shigella* at any time. Beck, Munoz and Scrimshaw (1957) reported that the point prevalence of *Shigella* excretion among children under 10 years old in twelve communities in Guatemala varied between 3.7 and 16.2 percent. Richardson and others (1968) recorded that 13 percent of African school children (7–16 years old) in the Western Transvaal (South Africa) were excreting *Shigella* (mainly *Sh. flexneri*) in summer. In the USA, *Shigella* excretion prevalences of up to 10 percent among children have been recorded in some samples of farm labor families in California and mining families in Kentucky (Hollister and others 1955; Schliessmann and others 1958; Watt and others 1953). It has been estimated that the prevalence of *Shigella* excretion among the whole population is 0.46 percent in the USA, 0.33 percent in England and Wales, and 2.4 percent in rural Sri Lanka (Geldreich 1972).

Khan and Mosley (1968) studied shigellosis in Rayer Bazar, a village on the outskirts of Dacca (Bangladesh). Shigellosis occurred throughout the year with a peak during the monsoon (June–October). Seventy-one percent of shigellosis cases identified were children under 5 years old. The proportion of all recorded diarrheas that were associated with *Shigella* was 4.4 percent. Khan, Curlin and Huq (1979) studied the families of forty-seven index cases with diarrhea due to *Sh. dysenteriae* type 1 in Dacca. Twenty percent (49 of 240) of family contacts of index cases were infected with *Shigella* at some time during the 10 days following the reporting of the index case. The proportion of infected family contacts was highest (31 percent) among children 0–4 years old. Of the forty-nine infected family contacts, five (10 percent) required hospitalization and twenty-seven (55 percent) developed mild to moderate diarrhea. The equivalent proportions among the 0–4 age group were 27 percent and 73 percent—in other words, there were no asymptomatic infections in this age group. Poor families living in one-room houses with "open latrines" and unprotected water sources had higher secondary infection rates than other families. In another study in Dacca, Khan and Shahidullah (1980) found similar infection and diarrhea rates among family contacts of index cases having *Sh. dysenteriae* type 1 infection. Among family contacts of index cases having *Sh. flexneri* infection, 20 percent were infected, but the proportion of those infected who experienced diarrhea was only 20 percent.

Reller, Gangarosa and Brachman (1969) reviewed shigellosis in the USA over the period 1964–68. Approximately 10,000 cases per year were reported (very many will have gone unreported), and the highest attack rate occurred in the 0–4 age group. Peak incidence was in the late summer of each year. Residents of mental institutions, Indian reservations, and urban slums were identified as being especially at risk from shigellosis. Fifty-four percent of isolations were *Sh. sonnei* and 40 percent were *Sh. flexneri*. Only 6.6 percent of reported cases of shigellosis were associated with either foodborne or waterborne outbreaks (Black, Craun and Blake 1978).

The transmission of *Sh. sonnei* among school children in England was studied by Hutchinson (1956), who isolated the organism from toilet seats, toilet floors, chamber pots, clothes, bedding, toys, and floors. Eleven out of thirty-four toilet seats were found to be contaminated in a school during an epidemic. When heavily infected, loose, bulky stools were flushed away it was found that contamination of the seat could occur, but this did not appear to happen with solid stools. As many as 50 percent of children were found to be hand carriers after visiting a toilet at the peak of an outbreak. Observations at a nursery school showed that, of thirty-seven children, half handled the seat when settling themselves on it, and one-third of these then either handled their face or mouth or sucked their fingers. On the skin of the fingers *Sh. sonnei* remained alive for over 3 hours. Tests with five types of toilet paper showed that, when double thicknesses of paper were used, organisms from fluid or semisolid feces passed through the paper to the fingers each time. With solid feces the organisms passed through four out of five of the papers.

There is general agreement in the literature that the maintenance of endemic shigellosis has little or no relationship to water quality, but that it is strongly related to water availability and associated hygienic behavior. However, there will always be specific exceptions to this; for instance, Sultanov and Solodovnikov (1977) considered that the maintenance of dysentery in Dagestan (USSR) during 1959–73 was due to the widespread use of polluted surface water for domestic purposes.

Shigellosis occurs in epidemics in addition to being endemic. In 1969 and 1970 an epidemic due to Shiga's

bacillus (*Sh. dysenteriae* type 1) occurred in Central America and Mexico in which there were an estimated 112,000 cases and 8,300 deaths in Guatemala alone in the first 10 months of 1969 (Gangarosa and others 1970). Major outbreaks of Shiga dysentery also occurred in Bangladesh in 1973 (Rahaman and others 1975) and in Somalia in 1963–64 (Cahill, Davies and Johnson 1966). *Shigella* strains with plasmid-mediated multiple drug resistance were involved in Central America and Bangladesh.

Some epidemics are waterborne. An outbreak of some 2,000 cases of shigellosis due to *Sh. sonnei* occurred in 1966 in Montrose (Scotland) when the chlorination plant on the town's water supply broke down (Green and others 1968). During 1961–75, thirty-eight waterborne outbreaks (comprising 5,893 cases) of shigellosis were reported in the USA (Black, Craun and Blake 1978). Most of these outbreaks involved semipublic or individual water systems and were usually the result of inadequate or interrupted chlorination of water contaminated by feces. An outbreak due to *Sh. sonnei* and linked with bathing in a polluted section of the Mississippi River in Iowa (USA) has been reported (Rosenberg and others 1976).

Some epidemics are foodborne. An outbreak of at least 600 cases occurred on the island of Maui (Hawaii, USA) in 1970. *Shigella sonnei* was the causative bacterium, and the outbreak was spread by the contamination of poi (ground taro root) produced at a single factory and distributed around the island (Lewis and others 1972). It was shown that *Sh. sonnei* in poi could undergo a tenfold increase in concentration in 1 day at room temperature. An outbreak of at least 140 cases (culture-positive for *Sh. sonnei*) occurred in 1979 among hospital staff in the USA and was linked to the contamination of tuna salad by an infected cafeteria employee (Bowen 1980). During 1961–75, seventy-two foodborne outbreaks (comprising 10,648 cases) of shigellosis were reported in the USA (Black, Craun and Blake 1978). Most of these outbreaks were caused by contaminated salads associated with poor hygiene among food handlers.

## Control Measures

*Shigella* bacteria are transmitted from person to person, especially where hygiene is poor and the domestic environment is fecally contaminated. Effective control depends upon personal hygiene and the sanitary disposal of excreta.

### *Individual*

Antimicrobial prophylaxis has little to recommend it, although individual agents have been used for the control of institutional outbreaks. Sulphonamide and tetracycline have been used widely in some communities, but their use has a dubious effect. It must be remembered that resistance transfer factors were first described in *Shigella* and that the worldwide resistance of these bacteria to sulphonamides (and often also to other antibiotics including tetracycline, streptomycin, chloramphenicol, and ampicillin) is the result of the spread of such factors.

Killed vaccines are ineffective. Live oral vaccines are still at an experimental stage. They are type specific and protect for short periods against the disease. They are difficult to prepare and administer and are therefore of limited use.

Scrupulous personal hygiene is the most effective means of individual protection. Breast feeding considerably reduces the risk of disease in infants.

### *Environmental*

There is probably more information available on the effect of environmental improvements in reducing shigellosis than on any other infection described in this book. Studies have been conducted into the spread and control of shigellosis in institutions in developed countries (such as the work of Hutchinson, summarized above), and other investigations have examined the role of environmental modifications in controlling shigellosis in poor communities (see table 2-1). These later studies have either used diarrhea rates or the prevalence of *Shigella* isolations from rectal swabs from a sample of the community, often children.

In towns in Georgia (USA) Stewart and others (1955) found that *Shigella* infection was related to poor water supply, poor excreta disposal facilities, high fly counts, and to poor housing in general. More specifically it was found that, among otherwise similar households, those with water close to the house had a lower incidence than those who fetched water from further away, but that the type of water source (well or tap) did not affect shigellosis incidence. A subsequent study in Georgia (McCabe and Haines 1957) recorded that a latrine program (bored hole latrines, 2.5 meters deep) in the town of Boston was associated with a reduction in the detection of *Shigella* from 4.7 percent to 2.8 percent of rectal swabs. Rates in the control towns did not fall over the period. After completion of the latrine program, the rate of reported diarrhea was half that in the control towns. Although the housefly

population was not reduced, the breeding of flies in excreta was much reduced.

Schliessmann and others (1958) investigated environmental influences on shigellosis in 11 mining camps in the eastern coalfields of Kentucky (USA) during 1954 to 1957. Reported diarrheal disease rates ranged from 9.4 to 53.6 per 100 persons per year in the different study areas. More than half the total cases were children between 0 and 4 years old, and more than one-quarter were under 2 years old. The highest incidence occurred in August and September. *Shigella* isolation rates, obtained by rectal swabbing of preschool children, ranged between 0.7 percent and 10 percent in individual study areas. Seventy-six percent of *Shigella* isolates were *Sh. flexneri*. Shigellosis was the major cause of acute diarrhea in the areas with poor sanitation but was not a primary cause in the areas with the best sanitation. Housefly populations were generally low and were not associated with *Shigella* prevalence. Water quality was not related to the incidence of diarrhea or to the prevalence of *Shigella* excretion. Those having flush toilets and inside water had an incidence of diarrhea of 14 per 100 persons per year and a *Shigella* excretion prevalence of 1.1 percent; those having inside water but an outside latrine had figures of 24 per 100 per year and 2.4 percent; and those having outside water and an outside latrine had an incidence of 36 per 100 per year and a *Shigella* prevalence of 5.9 percent. Where water was not piped inside the house, persons having access to water in their yard had a diarrhea incidence one-third less than individuals obtaining water away from their premises (see also Schliessmann 1959).

In the San Joaquin Valley of California (USA), Watt and others (1953) reported that the *Shigella* excretion prevalences among children under 10 years old were higher in migrant worker camps (6.1 percent) than in poor but permanent housing on urban fringe areas (3.9 percent). A follow-up study on the camps (Hollister and others 1955) recorded the following *Shigella* excretion prevalences among children under 10 years old: in cabins with inside water, shower, and toilet, 1.6 percent; in cabins with inside water but with communal shower and toilet, 3.0 percent; in cabins with no internal facilities, 5.8 percent. Studies in the Lower Rio Grande Valley in Texas (USA) showed that fly control with DDT reduced the rates of both diarrheal disease and *Shigella* isolation (Watt and Lindsay 1948).

Gordon, Behar and Scrimshaw (1964) reported a comparison of acute diarrheal disease rates between families having a latrine and those having no latrine in rural Guatemala. The authors concluded that "the data give no indication that privies as used in the villages had any influence on the diarrheas of children in the first two years of life, the important part of the problem." A summary of surveys of diarrhea among preschool children in Bangladesh, Egypt, Iran, Mauritius, Sri Lanka, Sudan, and Venezuela (van Zijl 1966) concluded that water supplies and excreta disposal facilities were important determinants of shigellosis (see also Wolff, van Zijl and Roy 1969).

Rajasekaran, Dutt and Pisharoti (1977) studied 1,041 children under 5 years old for 1 year in five villages in Tamil Nadu (India). Thirty-two percent of all diarrheal episodes were associated with *Shigella* excretion. Those who used an open well (98 percent of water samples contained $>10$ coliforms per 100 milliliters) had a significantly *lower* incidence of diarrhea and shigellosis than those who used a street tap (25 percent of water samples contained $>10$ coliforms per 100 milliliters). Those who used tap water in the house had a lower incidence than both well users and street tap users. Preliminary findings from Teknaf (Bangladesh) suggested that diarrhea and shigellosis incidences were inversely related to the daily per capita usage of tubewell water (Rahaman 1979).

These and other studies (see table 2-1) indicate that a plentiful water supply located close to or in the home (to allow good personal cleanliness) and an adequate latrine that is properly used are key elements in the control of shigellosis. Good personal and domestic hygiene and restricting the access of flies to human excreta are also important. Water quality is not of particular importance in communities where shigellosis is highly endemic.

It must be emphasized that those who most commonly experience shigellosis, those who most commonly excrete *Shigella*, and those for whom the consequences of infection are potentially the most serious are small children. Small children are not only the major sufferers but also the major source of the bacteria, which will contaminate the domestic environment and subsequently infect other children and adults. The personal hygiene of small children, and mother-child behavior patterns, are therefore of great importance in controlling shigellosis.

## Occurrence and Survival in the Environment

Although the shigellae are among the most important pathogenic excreted bacteria, their presence and persistence in the environment have been studied far less than is the case for *E. coli* and the salmonellae.

*In water*

Shigellae will be found in low concentrations in most surface waters contaminated by human feces. They will therefore be present in many contaminated drinking water sources in developing countries, although their presence is almost never sought in routine water testing. Unlike the salmonellae, *E. coli*, and fecal streptococci, the shigellae are excreted only by man, and because much contamination of village water supplies derives from animals, the concentrations of shigellae will in general be much lower than the concentrations of the fecal indicator bacteria or the salmonellae.

Tap water will only contain shigellae if it is untreated and drawn from a contaminated source, or if the treatment plant has broken down—as in the Montrose (Scotland) outbreak (Green and others 1968).

Some studies on the survival of shigellae in water are listed in the appendixes of Feachem and others (1980). Survival depends upon factors such as the concentrations of other bacteria, nutrients, and oxygen and on the temperature. In clean waters, survival times are typically less than 14 days at warm temperatures (>20°C), whereas the bacteria may survive for a few weeks below 10°C. At warm temperatures, 99 percent reduction in *Shigella* numbers is likely to occur in less than 5 days. McFeters and others (1974) found that shigellae died more slowly in well water at 9–12°C than the fecal indicators, salmonellae, or *Vibrio cholerae* (the half-life of the shigellae was about 24 hours).

Survival is most prolonged in very clean waters (such as unchlorinated tap water) or in polluted water containing nutrients but having a minimum of other bacteria present. In these latter conditions, *Shigella* may grow. Talayeva (1960) recorded survival of *Shigella flexneri* at 19–24°C for up to 21 days in clean river water, up to 47 days in autoclaved river water, up to 9 days in well water, up to 44 days in autoclaved tap water, and for up to 6 days in polluted well water. McGarry and Stainforth (1978) reported experiments in China that showed the survival of *Sh. dysenteriae* for up to 93 days in sterilized water at 11–28°C. Shrewsbury and Barson (1957) made up sterile synthetic well water of the same general composition as that obtained from Hagar's Well in Mecca (Saudi Arabia) at the time of the 1883 cholera outbreak. Shigellae could survive for between 2.5 and 29 months in this sterile but fecally contaminated water at 21°C. Hendricks (1972) reported that *Sh. flexneri* multiplied in sterilized river water collected downstream from a sewage outfall. Growth occurred at 30°C but not at 20°C or 5°C, and no growth at any temperature was recorded in water collected upstream from the sewage outfall (see also Hendricks 1971).

Limited tests on *Shigella* in seawater (Nakamura and others 1964) suggest that survival times (15 to more than 70 days at 15°C) may be somewhat longer than those in freshwater—in contrast to the fecal indicator bacteria (chapter 13), which die more rapidly in seawater than in freshwater. Tests in sterile saline waters (salinities = 0.5, 2.5, and 3.5 percent) at various temperatures (4, 25, 37°C), however, showed that *Sh. dysenteria* survived for less than 6 days, even at 4°C (Jamieson, Madri and Claus 1976).

*In feces and sewage*

Between perhaps 0.2 percent and 4 percent of a community will be excreting *Shigella* depending on the levels of hygiene that prevail. Sewage may therefore contain between about 10 and $10^4$ *Shigella* per liter.

Few data are available, but it is likely that survival in feces and sewage is curtailed by the activity of the large populations of other bacteria present. Survival is enhanced at low temperatures, by sterilizing the feces or sewage prior to introducing the shigellae, or by raising the pH. Hutchinson (1956) studied the survival time of *Sh. sonnei* in naturally infected feces. At room temperature, survival times varied between 2 and 26 days depending on the initial concentration of shigellae, which varied from $7 \times 10^3$ to $3.2 \times 10^7$ per gram. With an initial concentration of $1.5 \times 10^6$ per gram, none could be detected after 7 days storage at 37°C, whereas an 82 percent reduction occurred at 20°C. Kligler (1921) reported that *Sh. dysenteriae* survived for less than 6 days in septic tank effluent. Experiments in China (McGarry and Stainforth 1978) showed that *Sh. dysenteriae* survived for up to 17 days in biogas plant effluent (11–28°C) but for less than 30 hours in the biogas plant itself (14–24°C).

*On surfaces*

The transmission of shigellosis depends substantially on the contamination of clothes, hands, and household surfaces; the bacteria are transferred from these surfaces to the mouth. Hutchinson (1956) recorded that *Sh. sonnei* could survive for over 3 hours on the fingers and for up to 17 days on a wooden toilet seat. Survival was prolonged by low temperature, high humidity, and poor lighting. Spicer (1959) found that *Sh. sonnei* survived for 7–10 days on cotton threads at cool temperatures and high or low humidities. Nakamura (1962) studied *Sh. sonnei* survival at various temperatures on metal, wood, cotton, paper, and glass in a laboratory with relative humidities between 17 and

33 percent. Survival times were 10–57 days at −20°C, 4–40 days at 4°C, 2–28 days at 15°C, 0–13 days at 37°C and 0–2 days at 45°C. At 15°C, survival was longer on cotton, wood, and paper than on glass and metal. It is noteworthy that toilets and latrines are often relatively cool, humid, and poorly lit—conditions ideal for the optimal survival of shigellae on interior surfaces.

*In food*

The contamination of food with shigellae is probably an important route of transmission in many communities (see, for instance, Barrell and Rowland 1979). Taylor and Nakamura (1964) reported that *Sh. sonnei* and *Sh. flexneri* survived for considerable periods (80 days or more) in foods such as flour, eggs, oysters, clams, and milk. At warm temperatures (25°C) some growth was noted. Acidic foods, such as orange juice, were more hostile environments for shigellae; even so, the organisms remained detectable in these foods for up to 10 days.

*On crops*

The few studies on *Shigella* survival on crops irrigated with night soil or sewage are listed in the appendixes of Feachem and others (1980). Shigellae on crop surfaces will typically be exposed to warm temperatures, bright sunlight, and rapid drying. These factors are all hostile to shigellae, and reported survival times are nearly always less than 7 days (Geldreich and Bordner 1971; Rudolfs, Falk and Ragotzkie 1951). It is probable that, in arid hot climates, only a very small fraction of shigellae on crops would survive beyond 2 days. Babov, Nadvornyi and Keimakh (1967) reported that a variety of vegetables grown on sewage farms at Odessa (USSR) were contaminated by *Sh. flexneri*. Contamination was eliminated when irrigation was stopped 2 weeks before harvest, but harvested vegetables could be recontaminated by being laid on the soil.

*In the air*

As with other enteric bacteria and viruses (see chapters 9 and 13), shigellae may be spread in aerosol droplets produced by flush toilets, activated sludge plants, and spray irrigation systems. Hutchinson (1956) found that toilet seats were contaminated by droplets containing shigellae when a loose stool was flushed away but not when a solid stool was flushed. Newson (1972) found that flushing a fluid suspension of $10^{10}$ *Sh. sonnei* produced an aerosol of about thirty-nine bacteria per cubic meter of air and that shigellae dispersed in splashes from the toilet could survive for up to 4 days.

Hickey and Reist (1975) failed to isolate any airborne shigellae downwind from two activated sludge tanks in the USA, and they attribute this to the very low concentrations of shigellae in the sewage. Katzenelson, Buium and Shuval (1976) found that the incidence of shigellosis on 77 kibbutzim practicing wastewater spray irrigation was 10 cases per 1,000 persons per year, whereas the incidence on 130 kibbutzim practising no form of wastewater irrigation was 4.5 cases per 1,000 per year.

## Inactivation by Sewage Treatment Processes

Few studies have been conducted on the inactivation of shigellae by sewage treatment plants—in part because they are difficult organisms to enumerate in sewage and in part because it is quite common to fail to find any in sewage, even where the community is known to be infected (for instance Brezenski, Russomanno and DeFalco 1965; Daniel and Lloyd 1980; Dixon and McCabe 1964; Olivieri, Kawata and Krusé 1978; Wang, Dunlop and De Boer 1956). The data that are available suggest that removal of shigellae is very similar to *E. coli* removal (chapter 13). Conventional treatment plants, without tertiary processes, will remove between 90 and 99 percent (Kabler 1959), whereas waste stabilization ponds can remove a far higher proportion. It is likely that the survival of shigellae in sewage and sewage effluents is considerably shorter than the survival of *E. coli*. Slijkhuis, Betzer and Kott (1976) reported that *Sh. flexneri* were not detectable after 2 days in a waste stabilization pond in Israel.

## Inactivation by Night Soil and Sludge Treatment Processes

There are no data available on *Shigella* destruction in most sludge treatment processes. However, the conditions of sludge treatment will be highly antagonistic to shigellae, and high rates of destruction may be expected. It is probable that *Shigella* destruction will proceed considerably more rapidly than that of the fecal indicator bacteria (chapter 13).

Processes about which data are available are composting and heating. Studies on aerobic thermophilic composting of night soil and garbage in Beijing

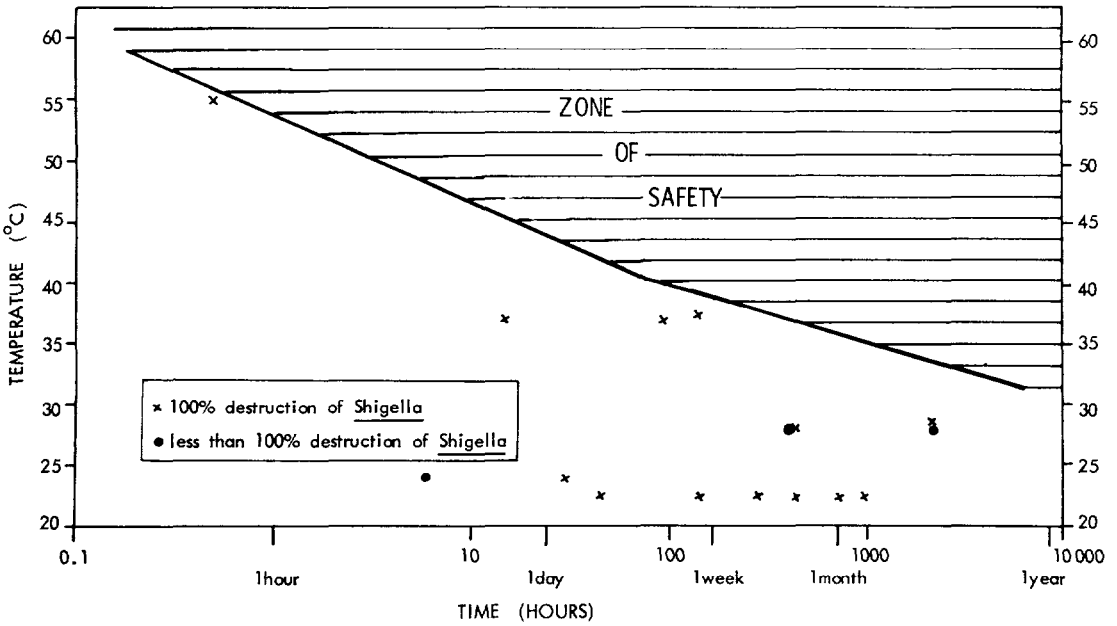


Figure 16-1. *The influence of time and temperature on* Shigella. The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

(China) showed that seeded *Sh. dysenteriae* were destroyed within 5 days in piles with a temperature of up to 50°C (Chinese Academy of Medical Sciences 1975). Other studies and reviews (for instance Bhaskaran and others 1957; Petrick 1954; Wiley 1962) confirm the elimination of shigellae from well-managed thermophilic composting systems but warn that shigellae, and other enteric bacteria, may survive on the edge of a pile where temperatures remain low (Reeves 1959). Various data on *Shigella* destruction by time-temperature effects have been compiled in figure 16-1, and it may be seen that 1 hour at 55°C, 1 day at 45°C, and 1 week at 40°C are lethal combinations.

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# 17

# *Vibrio cholerae* and Cholera

CHOLERA is probably the best known and most feared of the diarrheal diseases discussed in this book. Although it is by no means the most important cause of diarrhea in terms of total morbidity or mortality, it has caused, and in some parts of the world continues to cause, dramatic outbreaks of acute disease accompanied by considerable loss of life. In other areas cholera is a part of the overall spectrum of endemic diarrhea, and in these situations it often occurs with a regular seasonal periodicity. Cholera has a long history of scientific investigation, with some features of its epidemiology being clarified in London (England) by John Snow in the 1850s; the first full accounts of its clinical, bacteriological, and epidemiological aspects were published in the 1880s as a result of work done in Egypt (Koch 1884).

## Description of Pathogens and Disease

Despite the long history of study referred to above, cholera is attracting renewed scientific interest, and some traditional understandings are being considerably modified. New information is being gained not only on the mechanisms of pathogenesis and immunity but also on certain aspects of epidemiology and transmission. The information summarized in this chapter must therefore be considered as somewhat provisional.

### *Identification*

Cholera is caused by bacterial infection of the small intestine. The causative organism, *Vibrio cholerae*, exists in two biotypes—classical and El Tor. Both can cause an acute intestinal disease characterized by profuse rectal loss of water and electrolytes. The disease begins with sudden painless evacuation from the bowel; as it progresses, (acidotic) vomiting may start, together with muscle cramps due to lowered blood potassium levels (hypokalemia). If untreated, some patients become rapidly dehydrated, pass into shock, and die. Other patients experience much milder diarrheal illness. Sixty percent or more of untreated classical chloera cases die, whereas El Tor is generally regarded as a milder infection with a lower fatality rate and a higher proportion of asymptomatic infections. Recent evidence from Bangladesh suggests, however, that El Tor virulence may be increasing (Khan and Shahidullah 1980). It is not possible to distinguish classical from El Tor cholera clinically by reference to any particular case.

The effects of cholera are due to the action of an exotoxin, produced by the vibrios, which affects the epithelial cells of the gastrointestinal mucosa and leads to massive secretion of water into the lumen of the gut. Diagnosis is by isolation of the bacteria either from stool samples early in the clinical phase of watery diarrhea or by rectal swab from convalescents. It is usual to attempt direct plating on selective media as well as enrichment in alkaline peptone water before plating. To confirm suspected isolates, agglutination tests with anticholera O-group 1 serum are carried out together with microscopic investigation for vibrio morphology and biochemical charactrerization for isolates failing to agglutinate. The El Tor biotype differs from the classical vibrio in very few of its laboratory properties.

Fatality rates can be reduced to under 1 percent in well-managed treatment centers. The treatment of cholera primarily consists of preventing the patient from dying from loss of salts and water. The infection is then self-limited, but its duration is shortened by appropriate antibiotic therapy. Rehydration may be by mouth in patients that are not vomiting and is by giving clean water containing appropriate quantities of salt, potassium chloride, alkali such as sodium bicarbonate, and glucose to promote the absorption of the electrolytes. Patients, particularly children, in a state of shock or vomiting require appropriate intravenous

fluids rapidly. Normal hydration and acid-base balance should be achieved for adults within 2 hours of admission to a treatment center but is achieved more slowly for children weighing less than 20 kilograms.

*Occurrence*

The classical cholera vibrio is the historic cause of cholera. From its homeland in Bengal and the Ganges Valley, six classical cholera pandemics have spread. The El Tor biotype, first identified in Sinai in 1905, has only comparatively recently been accepted as *V. cholerae*. A focus of El Tor cholera was known to exist in the Indonesian island of Sulawesi in the 1930s. In 1961 this focus exploded and began to spread, thereby initiating the seventh known pandemic of cholera. It spread eastward to the Philippines, northward to Taiwan and Korea, and westward into India, where it replaced the classical biotype, and then on to Pakistan, the Middle East, and Europe. It also spread into East and West Africa and the Pacific islands (figure 17-1).

Where it is endemic, cholera develops a regular periodicity, and epidemic waves occur at one or two seasons of the year. These seasonal patterns are not the same in various places, and there is no good explanation of how the cholera infection cycle correlates with climatic conditions.

Endemic cholera prior to 1960/1961 was confined to India, especially the Ganges system, Bangladesh, and Sulawesi. Since then it has invaded many parts of the world and is, at the time of writing, considered to be endemic in several areas of Africa and Asia. Many national health authorities are very reluctant to admit or report endemic cholera because of the possible effect on tourism and international travel (for instance, the pilgrimage to Mecca). For this reason endemic El Tor cholera exists in a number of countries that officially deny it. The present pandemic has not yet spread to the

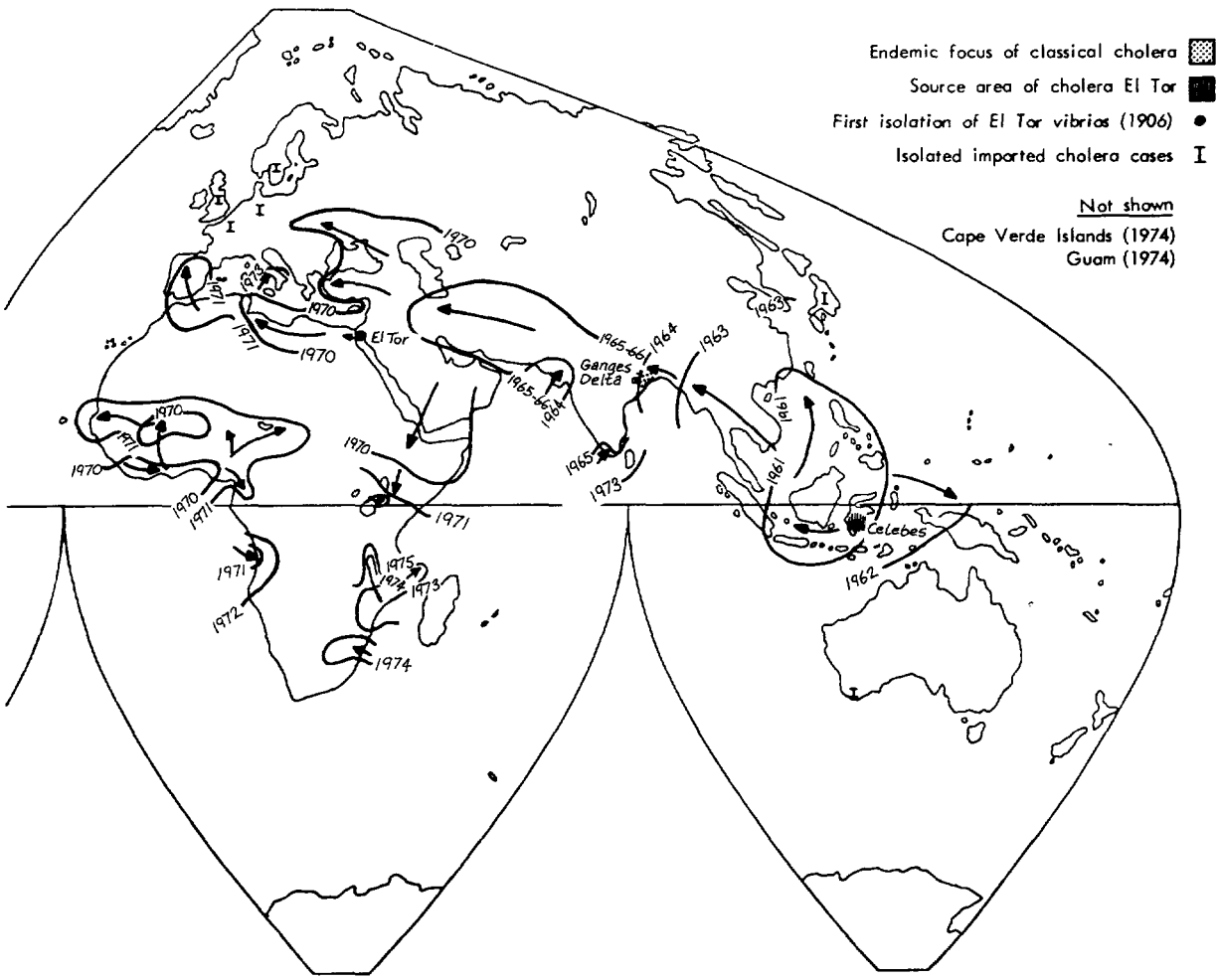


Figure 17-1. *The global spread of cholera, 1961–75*

Americas, although the risk of its introduction is very great.

*Infectious agents*

The family Vibrionaceae includes several human enteric pathogens of the genus *Vibrio*, and the taxonomic status of some of them remains uncertain and controversial. They are all Gram-negative, motile rods (0.5 by 1.5–3 micrometers) usually having a curved or comma shape. They are nonsporulating, noncapsulated, facultative anaerobes and possess a single polar flagellum (figure 17-2). The terminology for the various pathogenic and closely related vibrios used here is the one most commonly used at the present time, although it is not ideal and may be revised (WHO Scientific Working Group 1980).

Of greatest public health importance, and the main topic of this chapter, are organisms that have traditionally been called *Vibrio cholerae* or cholera vibrio, but which are now strictly known as *V. cholerae* O-group 1 or O1. They will be called *V. cholera* in this chapter. *V. cholerae* is the cause of epidemic cholera and exists in two biotypes (classical and El Tor) and three serotypes (Inaba, Ogawa, and the much less common Hikojima). *V. cholerae* produces an enterotoxin that has been extensively studied and is similar to *Escherichia coli* heat-labile enterotoxin (see chapter 13). Adherence to the intestinal mucosa is also an important virulence factor but is poorly understood.

A second group of *V. cholerae*, which agglutinate O1 antiserum but which do not produce enterotoxin, have been recently recognized. These are known as atypical *V. cholerae* O1 (in this chapter atypical *V. cholerae*), and some of them have biochemical properties that differ from those of *V. cholerae*. Atypical *V. cholerae* have been isolated from water both in areas where endemic clinical cholera is known to occur and in areas—such as Brazil, England, and the USA—where it does not occur. Atypical *V. cholerae* are thought not to be enteric pathogens.

The third group of *V. cholerae* strains are those which do not agglutinate O1 antisera but which are biochemically and genetically similar to *V. cholerae* O1. These are now called non-O1 *V. cholerae*, but until very recently were called non-agglutinating vibrios (NAGs) or non-cholera vibrios (NCVs). They are currently classified into seventy-two O-group serotypes, but this typing scheme is tentative and provisional. Non-O1 *V. cholerae* have been associated with many individual cases of cholera-like diarrhea and with some small outbreaks. Some non-O1 *V. cholerae* produce a cholera-like enterotoxin.

Finally, there are other potentially pathogenic vibrios that are clearly not *V. cholerae*. *V. parahaemolyticus* is a halophilic marine organism responsible for numerous outbreaks and attacks of food poisoning associated with seafood. It has a marine rather than an enteric reservoir and so is not considered in this chapter, although it is briefly discussed in chapter 7. The Group F (or Group EF6) vibrios (often mistakenly identified as *Aeromonas*) have been isolated from the stools of patients with diarrhea in many countries, but it is uncertain whether they are toxin-producing or pathogenic. Other vibrio species


Figure 17-2. V ibrio cholerae *under scanning electronmicroscopy*. The single polar flagellum of the organism is prominent. Scale bar = 1 micrometer. (Photo: J. Gallut, Institut Pasteur, Paris, France. Reproduced by courtesy of *Bulletin of the World Health Organization*)

occasionally isolated from man—*V. alginolyticus*, *V. metschnikovii*, *V. vulnificus*, and L+ *Vibrio*—are not believed to cause diarrhea.

*Reservoir*

The primary source of infection that has been clearly documented is the human case or carrier. There is speculation over the role of environmental isolates of atypical *V. cholerae* and non-O1 *V. cholerae* in cholera epidemiology and the possibility of an environmental reservoir (see below, the section "Occurrence and Survival in the Environment"). There is also speculation about the role of animal reservoirs, especially for non-O1 *V. cholerae* or for *V. cholerae* were isolated interepidemic periods. Sanyal and others (1974) examined 1,287 fecal samples from 195 domestic animals following an outbreak of cholera in Varanasi (India) during 1972. The proportions of animals from which *V. cholerae* or non-O1 *V. cholerae* were isolated were: dogs, 27 percent; chickens, 18 percent; cows and goats, each 11 percent. There were no isolations from buffalo, donkeys, or horses. Out of a total of fifty-four strains of *V. cholerae* isolated, eight were *V. cholerae* O1 (El Tor, Ogawa). Neither this nor other studies have clearly shown that animal infections with *V. cholerae* or non-O1 *V. cholerae* play any role in the epidemiology of human infection and disease.

*Transmission*

Cholera is transmitted by the fecal-oral route from person to person, and transmission is encouraged by inadequate water supply and excreta disposal facilities and, more generally, by poverty and overcrowding. Convalescent and asymptomatic individuals may excrete $10^2$–$10^5$ *V. cholerae* per gram of feces, whereas an active case excretes $10^6$–$10^9$ per milliliter of rice-water stool (Dizon and others 1967; Greig 1914; Smith, Freter and Sweeney 1961).

Infective doses are high in healthy adult males. Hornick and others (1971) required $10^8$ classical *V. cholerae* in water to produce diarrhea in 50 percent of adult volunteers (the median infective dose, or $ID_{50}$), and $10^{11}$ organisms to produce cholera-like diarrhea. With the prior administration of 2 grams of sodium bicarbonate, the $ID_{50}$ was lowered to $10^4$ for diarrhea and $10^8$ for cholera-like diarrhea. No diarrhea or infection was produced by $<10^8$ organisms without $NaHCO_3$ or by $<10^3$ organisms with $NaHCO_3$ (see also Cash and others 1974).

Gastric acidity is an important barrier to cholera infection, and those with lowered acidity (hypochlorhydria) may be infected by lower doses than others. More recent volunteer studies with El Tor strains have shown that infective doses are lower when the organisms are administered in food than in small volumes of water (WHO Scientific Working Group 1980). This could be due to more rapid gastric emptying, neutralization of gastric acid by food, or protection of vibrios that are adsorbed to, or embedded within, food particles. Nothing is known about the dose needed to cause acute diarrhea in 1 percent of malnourished children, but it may be $10^2$ or even less.

If it is assumed that the environmental reservoirs of *V. cholerae* described below are epidemiologically unimportant, then cholera transmission must take place by direct person-to-person contact or by the fecal contamination of water or food. Waterborne and foodborne transmission have both been clearly demonstrated on specific occasions. Cholera has classically been regarded as a waterborne disease, and there are some experts who believe that this is its dominant and normal mode of transmission. Others maintain that this may be true in Bangladesh but not elsewhere, while a third opinion holds that cholera transmission among poor people in developing countries is primarily nonwaterborne. This subject has attracted recent debate (for instance Feachem 1976; Levine and Nalin 1976) and is of considerable importance in designing control strategies. The topic has been comprehensively reviewed by Feachem (1981, 1982).

*Incubation period*

The incubation period is generally short and clinical symptoms occur within 0.5 to 5 days (usually 1–3 days) of ingesting the bacteria. Incubation periods may be inversely related to the dose of organisms ingested.

*Period of communicability*

Convalescents generally excrete *V. cholerae* intermittently and only for short periods. Thus, 50 percent of cholera cases will be found to excrete the pathogen for up to 5 days, 30 percent continue to excrete for up to 15 days, and 10 percent for up to 25 days. By 1 month usually less than 5 percent of cases are still excreting *V. cholerae*, and it is very uncommon to find carriage persisting beyond 2 months. The truly chronic carrier—such as Cholera Dolores from the Philippines (Azurin and others 1967)—is a very rare phenomenon. Asymptomatic infection is common, and the El Tor biotype produces a higher infection to case ratio than classical cholera.

*Resistance*

In endemic areas, it appears that repeated reinfection by *V. cholerae* leads to a gradual build-up of immunity with increasing age (Gangarosa and Mosley 1974). This may be one reason why the attack rates in children in endemic areas are considerably higher than in adults, whereas in epidemic situations where cholera has been recently introduced the reverse is often true. However, among those infected overt disease is more common in adults than in children.

A previous attack of cholera diarrhea confers solid immunity against reinfection with the same serotype of *V. cholerae* for about 1 year. An investigation in Bahrain showed that infants who were principally bottle-fed had a significantly higher risk of cholera than infants who were breast-fed, although it was not clear whether this arose from contaminated milk and bottles or from protective ingredients in maternal milk (Gunn and others 1979).

Cholera is a disease of the lower socioeconomic groups. Fishermen and boatmen, living along polluted water courses, are specially at risk. So also are people with hypochlorhydria, either due to malnutrition or other natural causes, or following gastric surgery (Sack and others 1972). Although the El Tor biotype may be less virulent than the classical, causing more mild cases of cholera, the host is probably equally susceptible to colonization by either.

*Epidemiology*

Studies on *V. cholerae* El Tor infection, in both epidemic and endemic situations, have repeatedly emphasized that the severe cases that reach the attention of treatment centers and physicians are the tip of an iceberg of widespread asymptomatic and mild clinical infection in the community. Estimates of a case to infection ratio of 1:30, or less, are commonly quoted. The asymptomatic infections are generally short lived but can be of crucial epidemiological importance in transmitting and geographically spreading cholera. Attempts to reconstruct the modes of transmission and spread of cholera that concentrate on known clinical cases are unlikely to be successful. To understand cholera epidemiology, it is necessary to take full account of the transient carrier, and to document the occurrence of transient carriage it may be necessary to undertake multiple fecal examinations and use serological techniques to determine whether an asymptomatic individual has been infected. These difficulties are one reason why so many investigations of cholera outbreaks are inconclusive or fall back on plausible but usually unproven explanations of waterborne transmission.

One of the most characteristic features of endemic cholera is its very pronounced seasonal pattern. For instance, in Dacca (Bangladesh) cholera used to peak dramatically during November–January, whereas 200 kilometers away in Calcutta (India) the peak was April–June. Recently these peaks have shifted and now occur during September–November in both areas. The reasons for these and other seasonal patterns of cholera remain entirely unexplained.

Non-O1 *V. cholerae* has been isolated from stools of persons with diarrhea in many countries in Asia, Africa, Europe, and, significantly, North and South America. Large epidemics have not been reported. In the USA most infections occur during the warmer summer months, while in Bangladesh there appears to be a peak in spring and summer before the annual cholera peak. Small foodborne outbreaks are common in the developed countries, but little is known of transmission and epidemiology in developing countries.

The epidemiology of cholera remains in many ways uncertain and controversial. The importance of waterborne transmission, the maintenance of cholera during interepidemic months of the year, the explanation of seasonality, the failure of tubewells in Bangladesh to reduce incidence, and the role of a possible aquatic reservoir for *V. cholerae* are all topics of current debate. Space does not permit a full review of these issues here. For a conventional account of cholera epidemiology, the reader should consult Gangarosa and Mosley (1974); Feachem (1981, 1982) provides a review of the more recent literature and debates.

## Control Measures

The most cost-effective control measures to deal with either endemic or epidemic cholera remain uncertain. Understanding of control will increase as more information is gathered on the epidemiological issues discussed above. Cholera control among people who are poor has so far proved to be extremely difficult. The course of a cholera epidemic is often dramatic and short-lived, and by the time control measures are applied the epidemic may be waning naturally. This can give a false impression of the efficacy of the control measures and lead to unjustified claims—as was the case when John Snow removed the handle from the Broad Street pump in London (England) in 1855.

*Individual*

Prophylactic antibiotics have been used to control some cholera outbreaks and to limit their spread. There is no evidence that this practice is effective, and there is mounting concern over the rising prevalence of antibiotic-resistant strains of *V. cholerae* in some countries. Large amounts of tetracycline (1,788 kilograms in the first 6 months) were used therapeutically and prophylactically following the outbreak of cholera in Tanzania in October 1977. Initially, all strains of *V. cholerae* tested were fully sensitive to tetracycline, but after 6 months 76 percent of isolates were resistant (Mhalu, Mmari and Ijumba 1979). Subsequent work showed that this antibiotic resistance was mediated by transferable plasmids that confer multiple antibiotic resistance (Towner and others 1980; Towner, Pearson and O'Grady 1979). Multiple antibiotic resistance has also been reported from 5–36 percent of *V. cholerae* isolates from Bangladesh (Threlfall, Rowe and Huq 1980).

Immunological prevention by vaccines is at present disappointing. Killed vaccines do afford a measure of protection but are usually less than 70 percent effective, and such immunity as is produced does not last at reasonable levels for more than about 4 months. A study in Bangladesh showed that mass vaccination was costly and ineffective (Sommer and Mosley 1973). Current research is directed at further characterizing pathogenesis and virulence factors and at developing and testing a variety of alternate vaccines based on live mutant strains or nonviable antigens such as the B subunit of the cholera enterotoxin.

Rigorous personal cleanliness and care in eating and drinking habits are probably the surest ways by which an individual can reduce the risk of cholera in an endemic or epidemic situation.

*Environmental*

There is no doubt that some combination of improved water supplies, excreta disposal facilities, better housing, and all the various improvements in daily life that come with increased wealth and education have been responsible for the elimination of cholera from the developed countries and from many middle-class communities in developing countries. Cholera was and remains a disease of poverty and the living conditions that are associated with poverty. Countries that experience the problem of endemic or epidemic cholera today are faced with the question of how to control the disease among poor communities in the short-term while poverty persists. Many claims have been made for the efficacy of various environmental control methods, but few of these have been justified, and most programs have been unsuccessful. Indeed, the experience with environmental control among the rural and urban poor has been so bad that some experts feel that the priority allocation of resources should be toward the establishment of networks of treatment centers for providing simple but highly effective rehydration therapy to reduce mortality (Greenough 1979).

The impact of water supply and sanitation schemes on endemic or epidemic cholera in poor communities is uncertain. Six studies in Bangladesh showed no impact (Briscoe 1978; Feachem 1982), whereas a study in the Philippines showed a very considerable impact (Azurin and Alvero 1974). The interpretation of these findings is controversial and has been recently reviewed in detail by Feachem (1982).

In some outbreaks—for instance, in Tanzania from 1977 to 1980—the geographical spread of cholera was due to the movement of infected individuals and gave rise to the characteristic pattern of spread along major railway and road routes. In such circumstances the limitation of movement of people in or out of areas known to be affected may reduce the risk of spreading the disease. Travel restrictions are difficult to enforce, however, and may seriously disrupt the movement of foodstuffs. If travel restrictions are combined with issuing prophylactic tetracycline to those who must travel, as was done in Tanzania, the problems of increased antibiotic resistance described above may occur.

Cvjetanović (1979) and Cvjetanović, Grab and Uemura (1978) used a mathematical model to compute the relative economic merits of sanitation, chemoprophylaxis, and immunization as methods of cholera control. Unfortunately, the cost of sanitation was set far too low (US$0.15 per capita at 1971 prices), and the effectiveness of sanitation was overestimated. Not surprisingly, this analysis showed sanitation to be highly cost-beneficial (with benefits taken only as the medical treatment costs saved), whereas immunization was shown to have costs far exceeding benefits because the currently available vaccine would have to have been given annually to have had any major impact on disease. Nonetheless, the analysis highlighted the benefits of sanitation as a measure having potential effects on a range of enteric and other diseases, as compared with vaccination, which, even if a more protective vaccine were available, is difficult to administer to most children, probably requires repeated readministration, and only protects against a single pathogen.

*Carrier surveillance and international regulations*

Since the chronic carrier is extremely rare, surveillance to identify carriers is not of significance in the control of this disease. This is in marked contrast with typhoid. The principal types of cholera carriage are incubatory, convalescent, and contact.

Up to December 31, 1970 International Sanitary Regulations were in force. They stipulated a 5-day quarantine period for travelers from areas where cholera was established. The regulations were abandoned when it was recognized that they were not preventing the spread of the current pandemic. Among the reasons for this failure were the concealment or denial of the existence of the disease in a country, together with the unknown importation of cases across unpatrolled borders. Current surveillance at national and international levels has been ineffective in preventing the spread of cholera into receptive countries—those with poor sanitation, hygiene, and health services. Nonetheless, surveillance to identify clinical cases (and, hence, the geographical advance of the disease) provides valuable epidemiological information and allows the organization of treatment in the absence of effective control measures.

## Occurrence and Survival in the Environment

The study of *V. cholerae*, atypical *V. cholerae*, and non-O1 *V cholerae* in the environment is attracting increasing attention at the present time. The conventional view that *V. cholerae* is an organism only found in the environment in close association with human cases or infections, and only surviving for a few days at most, is now being revised.

*In water*

The relationship between *V. cholerae* and water has been the focus of many investigations and is crucial to an understanding of the epidemiology of cholera. The traditional view of this subject—as stated by Felsenfeld (1974):

> some authors claimed that cholera vibrios may survive in water, particularly, seawater, for as long as 2 months. This is, however, scarcely possible under natural conditions if reinfection of the water does not take place

—is now known to be incorrect.

Data on the occurrence of *V. cholerae* in water are of two types. First, there are the numerous reports of *V. cholerae* isolations from rivers, tanks, ponds, wells, and household water jars in or near communities where cholera cases or infections are known to be occurring. Some of these reports are reviewed in a separate publication (Feachem 1981). Second, there are the more recent findings of *V. cholerae*, especially but not exclusively atypical O1 and non-O1 strains, in water and wastewater at sites distant from any known human *V. cholerae* infection. These findings are reviewed below in the section on possible aquatic reservoirs.

The reason that the view expressed by Felsenfeld was so strongly held for nearly 100 years is, first, that researchers had failed to find *V. cholerae* in the aquatic environment except in close association with human infection (due to a combination of not looking, looking in the wrong manner and looking in the wrong place), and, second, that survival experiments conducted in the laboratory had shown *V. cholerae* to be an organism with only limited survival ability in certain aquatic environments.

Some of the considerable accumulation of data on *V. cholerae* survival in water is summarized in tables 17-1 to 17-5. In clean water (for instance, dechlorinated tap water), survival times are up to 1 month at 4°C and 2–14 days at 20–30°C. In raw well water, survival times are over a month at 4°C and generally between 1 and 20 days at 20–30°C, although reports from India and Tanzania suggest survival of the El Tor biotype in raw well water of up to 55 days. A single report of *V. cholerae* survival in refrigerated raw surface water gives a survival time of 48 days, while survival at 20–30°C is generally 1–6 days, with occasional reportings of longer survival and one exceptional report from Tanzania of 48 days. As would be expected, survival in seawater is prolonged, with durations of 2 months at 4°C and 6–60 days at 20–30°C. Finally, a single report from the USSR (table 17-4) and epidemiological evidence from Portugal (Blake and others 1977) suggest the ability of *V. cholerae* to survive for prolonged periods in certain mineral waters.

It is clear from the tables that survival can be greatly prolonged in nutrient-rich waters and seawaters that have been boiled or autoclaved prior to contamination with *V. cholerae*, thus eliminating competing microorganisms and possibly also making the chemical composition of the water more favorable for *V. cholerae* survival. Although the nature and extent of *V. cholerae* inhibition by a mixed microflora in a natural surface water are not known, one study showed a failure of *E. coli*, *Pseudomonas* spp., and *Aerobacter* spp. to suppress *V. cholerae* El Tor survival in artificial sterile well water (Pandit and others 1967). Sunlight considerably curtails *V. cholerae* survival.

Table 17-1. *Survival of* Vibrio cholerae *in surface waters*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Cheng (1963) | El Tor $1.5 \times 10^5$ | River water | 21–31°C | 3 days |
| | | Drain water | | 2 days |
| | | Pond water (all taken in or near Taipei) | | 6 hours |
| Gohar and Makkawi (1948) | Classical | Nile water | Room temp. (Egypt) | |
| | from feces | | | 5 days |
| | from culture | | | 10 days |
| Khan and Agarwal (1929) | Classical (clinical isolate) | Jumna and Ganges river waters | Room temp. (Allahabad) | |
| | | Raw | | 8 days |
| | | Filtered | | 18 days |
| | | Boiled | | 29 days |
| | | Boiled & filtered | | 14 days |
| | Non-O1 (water isolate) | Raw | | 20 days |
| | | Filtered | | 20 days |
| | | Boiled | | 18 days |
| | | Boiled & filtered | | 20 days |
| Konchady and others (1969) | Classical $10^4$ | Calcutta | 25°C | |
| | | River Hooghly | | 6 days |
| | | Canal water | | 6 days |
| | | Pond water | | 6 days |
| Lahiri, Das and Malik (1939) | Classical (Inaba) $10^6$ | Spring water | Room temp. (Calcutta) | |
| | | Raw | | 1 hour |
| | | Autoclaved | | 18 hours |
| | | River Hooghly (Calcutta) | | |
| | | Raw | | 18 hours |
| | | Autoclaved | | 3 days |
| | | Filtered | | 2 days |
| | | Autoclaved & filtered | | 2 days |
| | | Tank waters (Calcutta) | | |
| | | Raw | | 2–3 days |
| | | Autoclaved | | 3–12 days |
| | | Filtered | | 7 days |
| | | Autoclaved & filtered | | 15–18 days |
| Lema, Ogwa and Mhalu (1979) | El Tor $10^5$ | Swamp water in Dar es Salaam | 4°C | 48 days |
| | | | 30°C | 48 days |
| | | | 32°C in sunlight | 3 days |
| Mukerjee, Rudra and Roy (1961) | Classical $2 \times 10^6$ | River Hooghly (Calcutta) | Room temp. (Calcutta) | |
| | | Raw | | 1–6 days |
| | | Autoclaved | | 4–22 days |
| | | Filtered | | 3–12 days |

Table 17-1 (*continued*)

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| | | Tank water (Calcutta) | | |
| | | Raw | | 1–6 days |
| | | Autoclaved | | 4–23 days |
| | | Filtered | | 3–7 days |
| | El Tor (clinical isolate) $2 \times 10^6$ | River Hooghly (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 11 days |
| | | Tank water (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 13 days |
| | El Tor (water isolate) $2 \times 10^6$ | River Hooghly (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 11 days |
| | | Tank water (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 16 days |
| | Non-O1 (clinical isolate) $2 \times 10^6$ | River Hooghly (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 9 days |
| | | Tank water (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 12 days |
| | Non-O1 (water isolate) $2 \times 10^6$ | River Hooghly (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 11 days |
| | | Tank water (Calcutta) | | |
| | | Raw | | 2 days |
| | | Autoclaved | | 13 days |
| Neogy (1965) | Classical | Pond water | Room temp. (India) | 1–2 days |
| | El Tor | | | 8 days |
| Read and others (1939) | Classical | Autoclaved tank waters (Calcutta) | Room temp. (Calcutta) | >30 days |

*Note:* Older literature is reviewed by Pollitzer (1959).

a. Times given, for instance, as 22 days are durations at which viable organisms could no longer be detected. Times given as >30 days indicate that organisms were still viable at that time but that sampling was discontinued.

Some experiments have included direct comparisons of the survival of classical and El Tor biotypes, and occasionally also non-O1 strains (tables 17-1, 17-2 and 17-4). Two studies showed markedly longer survival of El Tor than classical *V. cholerae* (Felsenfeld 1965; Neogy 1965); one study showed similar survival between the two biotypes (Sayamov and Zaidenov 1978); one study showed non-O1 *V. cholerae* surviving for longer than classical *V. cholerae* O1 (Khan and Agarwal 1929); and one study showed no difference in survival between classical O1, El Tor O1 and non-O1 *V. cholerae* (Mukerjee, Rudra and Roy 1961). It would appear from this literature review that the widely held belief that El Tor *V. cholerae* survives for considerably longer periods in water than the classical biotype is not firmly based. This is especially true in view of the major

Table 17-2. *Survival of* V. cholerae *in well water*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Cheng (1963) | El Tor $1.5 \times 10^5$ | Well water (village near Taipei) | 21–31°C | 1 day |
| Felsenfeld (1965) | Classical | Shallow well water | ? | 8 days |
| | El Tor | | ? | 19 days |
| Khan and Agarwal (1929) | Classical (clinical isolate) | Well water (Allahabad) | Room temp. (Allahabad) | |
| | | Raw | | 1 day |
| | | Filtered | | 6 days |
| | | Boiled | | 9 days |
| | | Boiled & filtered | | 8 days |
| | Non-O1 (water isolate) | Raw | | 12 days |
| | | Filtered | | 6 days |
| | | Boiled | | 18 days |
| | | Boiled & filtered | | 26 days |
| Konchady and others (1969) | Classical $10^4$ | Well water (Calcutta slum) | 25°C | 6 days |
| Lema, Ogwa and Mhalu (1979) | El Tor $10^5$ | Well water (Tanzania) | 4°C | 55 days |
| | | | 30°C | 55 days |
| | | | 32°C in sunlight | 1 day |
| McFeters and others (1974) | ? $10^5$ | Sterile well water | 9.5–12.5°C | >2 days ($t_{90}$ = 1.3 days)[b] |
| Pandit and others (1967) | El Tor (Ogawa) $10^3$ | Well water (Punjab) | 21°C | 18 days |
| | | | 37°C | 4 days |
| | | Well water (Uttar Pradesh) | 21°C | 51 days |
| | | | 25°C | Fourfold growth after 1 day Survival for >7 days |
| | | | 37°C | 4 days |
| | | Experiments with well water simulating actual removal and replacement of water in well following single contamination with $10^3$ *V. cholerae* per milliliter | 25°C | 10–12 days |
| Pesigan, Plantilla and Rolda (1967) | El Tor $10^6$ | Deep well water (Manila) | | |
| | | Raw | 5–10°C | 18 days |
| | | | 30–32°C | 13 days |
| | | | Sunlight | 4 days |
| | | Autoclaved | 5–10°C | 42 days |
| | | | 30–32°C | 17 days |
| | | | Sunlight | 8 days |

Table 17-2 (*continued*)

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| | | Raw well water stored in clay jar | 30–32°C ambient, but jar storage may have cooled water | 32 days |
| Shrewsbury and Barson (1957) | Classical | Sterile, synthetic well water of same composition (pH = 5.6) as Hagar's Well (Mecca, Saudi Arabia) during the cholera epidemic of 1883 | 5°C | 1 day |
| | | | 21°C | 1 day |
| | | | 25°C | 1 day |
| | | Same water with: | | |
| | | pH 7 | 5°C | 3 days |
| | | | 21°C | 3 days |
| | | pH 8 | 5°C | 3 days |
| | | | 21°C | 77 days |
| | | pH 9 | 5°C | 3 days |
| | | | 21°C | 3 days |

*Note:* Older literature is reviewed by Pollitzer (1959).

a. Times given, for instance, as, 18 days are durations at which viable organisms could no longer be detected. Times given as > 7 days indicate that organisms were still viable at that time but that sampling was discontinued.

b. $t_{90}$ Time for 90 percent reduction.

probable strain-by-strain differences within each biotype and the differences between laboratory cultures, fresh clinical isolates, and water isolates. On the basis of the literature reviewed here it remains unproven than El Tor is a more persistent organism in water than the classical biotype, and the true interbiotypic and intrabiotypic variabilities in survival remain to be documented. It follows that explanations of the differences in epidemiology between El Tor and classical cholera—for instance, the greater "endemic tendency" of the former—cannot, at the present time, make use of putative differences in environmental persistence between the two biotypes.

Laboratory experiments on *V. cholerae* survival in water may accurately reflect conditions in manmade containers of clean water (such as reservoirs, cisterns, jars, and glasses), but they cannot replicate conditions in natural water bodies such as rivers, ponds, or even open wells. In these latter waters there may be abundant flora and fauna, and many varied surfaces, not reproduced or simulated in the laboratory experiments. There is increasing evidence (reviewed below) that *V. cholerae* in natural waters are frequently in close association with bottom sediments, chitinous fauna, and plant surfaces; therefore, laboratory data must be interpreted with extreme caution.

### *In feces and night soil*

Except for the atypical *V cholerae* and non-O1 *V. cholerae* which may maintain an environmental reservoir, the primary source of *V. cholerae* in the environment is the feces of man. Persons infected by *V. cholerae*, though not sick, may excrete $10^2$–$10^5$ per gram of feces, while those with active and severe disease may excrete $10^6$–$10^9$ per milliliter of rice-water stool (Dizon and others 1967; Greig 1914; Smith, Freter and Sweeney 1961). Unlike most other enteric bacterial infections, the prevalence of excretion of *V. cholerae* by the general healthy population is very low—typically well under 1 percent, even in endemic areas.

In areas of endemic cholera, or during a cholera outbreak, it is to be expected that *V. cholerae* will occur in the night soil produced by the affected communities. Forbes, Lockhart and Bowman (1967) and van de Linde and Forbes (1965) reported numerous isolations of *V. cholerae* from night soil in Hong Kong, both when cholera cases were and were not occurring in the city.

Table 17-3 *Survival of* V. cholerae *in tap water*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Chlorine residual milligrams per liter* | *Survival*[a] |
|---|---|---|---|---|---|
| Cheng (1963) | El Tor $1.5 \times 10^5$ | Taipei tap water | 21–31°C | 0.5 | 2 hours |
| Konchady and others (1969) | Classical $10^4$ | Tap water from deep tubewell (Calcutta) | 25°C | 0 | 6 days |
| Lahiri, Das and Malik (1939) | Classical (Inaba) $10^6$ | Calcutta tap water | Room temp. (Calcutta) | ? | |
| | | Raw | | | 18 hours |
| | | Autoclaved | | | 24 hours |
| | | Filtered | | | 2 days |
| | | Filtered & autoclaved | | | 12 days |
| Lema, Ogwa and Mhalu (1979) | El Tor $10^5$ | Dar es Salaam tap water | 4°C | Chlorinated at treatment works but probably no residual chlorine remaining at tap | 34 days |
| | | | 30°C | | 14 days |
| | | | 32°C in sunlight | | 3 days |
| Mukerjee, Rudra and Roy (1961) | Classical $2 \times 10^6$ | Calcutta tap water | Room temp. (Calcutta) | ? | |
| | | Raw | | | 2–8 days |
| | | Autoclaved | | | 4–18 days |
| | | Filtered | | | 2–6 days |
| Pandit and others (1967) | El Tor (Ogawa) $10^3$ | Delhi tap water | 21°C | De-chlorinated | 12 days |
| | | | 37°C | | 1 day |
| Pesigan, Plantilla and Rolda (1967) | El Tor $10^b$ | Manilla tap water | | | |
| | | Raw | 5–10°C | 0.6 | 1 hour |
| | | Raw | 30–32°C | 0.6 | 1 hour |
| | | Raw | Sunlight | 0.6 | 1 hour |
| | | Autoclaved | 5–10°C | 0 | 10 days |
| | | Autoclaved | 30–32°C | 0 | 1.6 days |
| | | Autoclaved | Sunlight | 0 | 12 hours |

*Note:* Older literature is reviewed by Pollitzer (1959).

a. Times given are durations at which viable organisms could no longer be detected.

During a 10-month sampling period, 46 percent (200 of 433) of bucket latrines in the slums of eastern Calcutta (India) were positive for *V. cholerae* on one or more occasions (Sinha and others 1967). *V. cholerae* isolations from latrines were obtained during months when no cholera cases were reported. In contrast, during 1968 in Dacca and Chittagong (Bangladesh) a total of 72,494 night soil samples yielded only 56 isolations of *V. cholerae*. all of which occurred at times when cholera cases were being reported (Bart, Khan and Mosley 1970).

Some reported data on *V. cholerae* survival in feces are summarized in table 17-6. Clearly survival is inversely related to temperature. Cheng (1963) and

Table 17-4. *Survival of* V. cholerae *in mineral water*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Sayamov and Zaidenov (1978) | Classical | Spring water from spa (Matsesta, USSR) | | |
| | $9 \times 10^5$ | Raw | 20–24°C | 22 days |
| | $1.5 \times 10^3$ | Diluted | | 15–65 days |
| | $9.5 \times 10^5$ | Boiled | | >1429 days |
| | $1.6 \times 10^3$ | Diluted | 37°C | >289 days |
| | El Tor | | | |
| | $1.2 \times 10^6$ | Raw | 20–24°C | 22 days |
| | $10^3$ | Diluted | | 18–39 days |
| | $9 \times 10^5$ | Boiled | | >1429 days |
| | $1.6 \times 10^3$ | Diluted | 37°C | >413 days |

*Note:* Further evidence of prolonged survival of *V. cholerae* in mineral water is provided by the investigation of the cholera outbreak in Portugal in 1974 (Blake and others 1977).

a. Times given, for instance, as 22 days are durations at which viable organisms could no longer be detected. Times given as >289 days indicate that organisms were still viable at that time but that sampling was discontinued.

Shoda, Koreyeda and Otomo (1934) found that survival was longer in liquid stools than in soft or solid stools. In summary, at ambient temperatures in tropical and subtropical countries, *V. cholerae* is unlikely to survive beyond 5 days in feces.

### *In sewage*

There are very few reports of *V. cholerae* in sewage. This is primarily because, in most developing countries, the section of the population that experiences the highest attack rates of cholera produces no sewage because their houses do not have flush toilets. Instead, they produce night soil (where *V. cholerae* has been found) or they defecate beside or into open water bodies (where *V. cholerae* has also been found).

Kott and Betzer (1972) reported estimates that Jerusalem sewage contained between 10 and $10^4$ *V. cholerae* per 100 milliliters during the 1970 cholera epidemic in Israel. Daniel and Lloyd (1980*a*) reported geometric mean concentrations of 2,600 and 160 non-O1 *V. cholerae* per 100 milliliters of very strong sewage (suspended solids 17,000 and 7,400 milligrams per liter, respectively) in two refugee camps near Dacca (Bangladesh). Isaacson and others (1974) reported the use of Moore pads to detect *V. cholerae* in sewage at mines in the Transvaal (South Africa) during 1973–74, when the spread of cholera from Malawi, Mozambique, and Angola was feared. *V. cholerae* (El Tor, Inaba) was isolated from the sewage prior to and during cholera outbreaks at the mines and acted as an effective early warning system for the outbreaks.

Survival of *V. cholerae* in sewage is summarized in table 17-7. Three studies (Altukhov and others 1975; Daniel and Lloyd 1980*b*; Zaidenov and others 1976) suggested that some sewages provide a permanent culture medium for some strains of classical, El Tor, and non-O1 *V. cholerae*. The other studies found that survival times were 1–24 days in sewage at 20–30°C. Survival times are shorter at warmer temperatures and longer in sterilized sewage than in raw sewage.

Direct comparisons of different biotypes and serotypes showed no differences in survival among classical O1, El Tor O1, and non-O1 strains (Mukerjee, Rudra and Roy 1961). Altukhov and others (1975) found an El Tor, Ogawa strain better able to multiply in bath house sewage at 37°C than a classical, Ogawa strain, although even the classical strain had not fallen below its initial concentration after 10 days. Daniel and Lloyd (1980*b*) found a sewage-derived non-01 strain better able to multiply in sewage than a laboratory reference strain of El Tor O1, although even the El Tor strain showed no reduction in concentration between 6 hours and 48 hours at 22–25°C. As with water, therefore, there is little evidence at present to suggest that the El Tor biotype is necessarily better able to survive in sewage than the classical biotype.

### *Summary of survival in water and wastewater*

In some survival studies the initial concentration of organisms present was reported, and it is therefore possible to estimate a death rate expressed as a $t_{90}$ value—the time in hours for a 90 percent or 1 log unit decline in concentration. In only a few studies were death curves plotted from which accurate $t_{90}$ values might be taken. For other studies the $t_{90}$ value can only be estimated from the initial concentration and the

Table 17-5. *Survival of* V. cholerae *in seawater*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Cheng (1963) | El Tor $1.5 \times 10^5$ | Coastal water near a fresh-water source | 21–31°C | 6 days |
| Jamieson, Madri and Claus (1976) | El Tor $1.5 \times 10^7$ | Sterilized seawater with adjusted salinity (percent) | | |
| | | 0.5 | 4°C | 5 days |
| | | | 25°C | 3 days |
| | | | 37°C | 2 days |
| | | 2.0 | 4°C | 4 days |
| | | | 25°C | 3 days |
| | | | 37°C | 1 day |
| | | 3.5 | 4°C | 4 days |
| | | | 25°C | 1 day |
| | | | 37°C | 1 day |
| Lema, Ogwa and Mhalu (1979) | El Tor $10^5$ | Seawater (Dar es Salaam) | 4°C | >58 days |
| | | | 30°C | >58 days |
| | | | 32°C in sunlight | 5 days |
| Pesigan, Plantilla and Rolda (1967) | El Tor $10^6$ | Seawater (Manilla) | 5–10°C | 58–60 days |
| | | | 30–32°C | 10–13 days |
| | | | Sunlight | 10–11 days |
| Various studies between 1885 and 1920 reviewed by Pollitzer (1959) | Classical | Sterilized seawater (Marseilles) | ? | 81 days |
| | | Seawater (Copenhagen) | Summer | 7–17 days |
| | | | Winter | 47 days |
| | | Seawater (New York) | | |
| | | Raw | ? | 7–47 days |
| | | Sterilized | ? | >285 days |
| | | Seawater (Japan) | | |
| | | Raw | 4°C | 9–27 days |
| | | Raw | Room temp. | 7–41 days |
| | | Raw | 37°C | 3–12 days |
| | | Sterilized | 4°C | 53–230 days |
| | | Sterilized | Room temp. | 152–209 days |
| | | Sterilized | 37°C | 30–83 days |
| Yasukawa (1933) | Classical $3 \times 10^4$ | Artificial seawater Top of tank | 18°C | 23 days |
| | | Bottom of tank | 18°C | 30 days |
| | $3 \times 10^5$ | In sunlight | 19–40°C | 2 hours |

a. Times given, for instance, as 6 days are durations at which viable organisms could no longer be detected. Times given as >58 days indicate that organisms were still viable at that time but that sampling was discontinued.

Table 17-6. *Survival of* V. cholerae *in feces*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Abel and Claussen (1895); cited by Pollitzer (1959) | Classical | Naturally infected cholera stools | 13–16°C | 10 days for over half the samples with a maximum of 29 days |
| Cheng (1963) | El Tor | Naturally infected stools | 29–31°C | 1–4 days |
| | | Artificially infected stools | 29–31°C | 2–4 days |
| Gildemeister and Baerthlein (1915); cited by Pollitzer (1959) | Classical | Naturally infected stools | 12–21°C | 10 days for half the samples; with a maximum of 51 days |
| Greig (1914) | Classical $1.5 \times 10^8$–$2 \times 10^9$ | Naturally infected ricewater stools | 22°C | Min. 1–3 days<br>Max. 10–17 days<br>Av. 3–8 days |
| | | | 29°C | Min. 1 day<br>Max. 2–13 days<br>Av. 1–7 days |
| Shoda, Koreyeda and Otomo (1934) | Classical | Naturally and artificially infected stools | 4°C | 1–5 days |
| | | | Room temp. (Japan) | 0.5–2 days |
| | | | 37°C | 6 hours |

a. Times given are durations at which viable organisms could no longer be detected. Max. = maximum, Min. = minimum, Av. = average.

overall survival time, without knowing the shape of the intervening death curve or whether the number of organisms fell below detectable levels considerably prior to the stated survival time.

Bearing in mind these limitations, $t_{90}$ values have been derived where possible. The few studies that showed prolonged maintenance of concentrations equal to or greater than initial values have been excluded and are discussed separately in the next section. Derived $t_{90}$ values are presented in table 17-8. The mean figures in table 17-8 suggest maximum survival in well water and seawater. The mean figures for the El Tor biotype are greater than for the classical biotype, but this comparison is invalid since each experiment used very different techniques and a wide variety of strains of various origins. It remains uncertain whether the interbiotypic variability of survival is greater than the intrabiotypic variability.

These $t_{90}$ values may be compared with typical $t_{90}$ values for coliforms of 20 to 115 hours (median 60 hours) in surface waters and with 0.6 to 8 hours (mean 2 hours) in seawater (chapter 13). For shigellae, in surface waters at temperatures of over 20°C, $t_{90}$ values generally fall well below 60 hours (chapter 16). Thus, even discounting the prolonged survival findings reviewed below, the $t_{90}$ values for *V. cholerae* are not greatly lower than those reported for coliforms and may be similar to those reported for other bacterial enteric pathogens. In a direct comparison of various bacteria in sterile well water, McFeters and others (1974) found the following $t_{50}$ values: shigellae, 22–27 hours; coliforms, 17 hours; salmonellae, 2–19 hours;

Table 17-7. *Survival of* V. cholerae *in sewage*

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Altukhov and others (1975)[b] | Classical (Ogawa) $10^3$ | Sewage of a bath house (USSR; BOD = 320 milligrams per liter) | 37°C | > 10 days |
| | El Tor (Ogawa) $10^5$ | | | > 10 days |
| Daniel and Lloyd (1980*b*) | El Tor $2 \times 10^6$ | Strong sewage at refugee camp (Bangladesh) | 22–25°C | Concentration fell by 1 log in 6 hours and remained steady for further 42 hours |
| | Non-O1 (sewage isolate) $2 \times 10^5$ | | | Concentration rose to $4 \times 10^6$ in 6 hours and remained steady for a further 42 hours |
| Flu (1921) | Classical | Sewage in septic tanks | Ambient temperature (Batavia) | 2 days |
| Gerichter and others (1975) | El Tor | Sewage (Jerusalem) | 20–28°C | Two phase decline: $t_{90}$ = 1.8 days for first 5 logs and $t_{90}$ = 8 days subsequently. *V. cholerae* not detected after 24 days[c] |
| Howard and Lloyd (1979) | El Tor $10^6$ | Raw sludge 1 percent solids | 25°C | $t_{90}$ = 2 days max survival = 14 days |
| | | 5 percent solids | | $t_{90}$ = 3 days max survival = > 14 days |
| Kott and Betzer (1972) | El Tor 10 | Diluted sewage (Haifa; BOD = 200 milligrams per liter) | Room temp. (Israel) | 1 day |
| Mukerjee, Rudra and Roy (1961) | Classical $2 \times 10^6$ | Sewage (Calcutta) Raw | Room temp. (Calcutta) | 1–5 days |
| | | Autoclaved | | 4–24 days |
| | | Filtered | | 2–7 days |
| | El Tor (clinical isolate) $2 \times 10^6$ | Raw | | 2 days |
| | | Autoclaved | | 9 days |

Table 17-7 (*continued*)

| *Source* | *Biotype and initial concentration per milliliter* | *Type of sample* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| | El Tor (water isolate) $2 \times 10^6$ | Raw | | 2 days |
| | | Autoclaved | | 10 days |
| | Non-O1 (clinical isolate) $2 \times 10^6$ | Raw | | 2 days |
| | | Autoclaved | | 8 days |
| | Non-O1 (water isolate) $2 \times 10^6$ | Raw | | 2 days |
| | | Autoclaved | | 8 days |
| Ohwada (1924); cited by Pollitzer (1959) | Classical | Sewage | 4°C | 12 days |
| | | | Room temp. (Japan) | 4 days |
| | | | 37°C | 1 day |
| Zaidenov and others (1976) | El tor (Ogawa) $10^4$ | Locomotive depot wastewater | 18–24°C | > 39 days |
| | | Domestic sewage | | 3 days |
| | | Dairy effluent | | 14 days |
| | 10 | Oil and water | | > 14 months |
| | | Diesel fuel and water | | > 14 months |

*Note:* Older literature is reviewed by Pollitzer (1959).

a. Times given, for instance, as 6 days are durations at which viable organisms could not be detected. Times given as > 10 days indicate that organisms were still viable at that time but that sampling was discontinued.

b. These experiments were discontinued after 10 days, at which time the concentration of classical *V. cholerae* was $5 \times 10^2$ while that of El Tor had risen to over $10^8$ per milliliter. Data from the bath house suggested that *V. cholerae* (El Tor, Ogawa) survived for at least 13 months in the sewerage system (temperature 20–25°C) despite repeated disinfection and no known external recontamination.

c. $t_{90}$: time for 90 percent reduction.

and *V. cholerae*, 7 hours. Pandit and others (1967) found that *V. cholerae* (El Tor) survived 2 to 5 times longer than *E. coli*, *Pseudomonas* spp., and *Aerobacter* spp. when they were added to artificial well water and stored at 25°C.

### *Prolonged survival in water and wastewater*

Pollitzer (1959) cited several early studies that reported prolonged survival of *V. cholerae* in various waters. Examples are up to a year in sterilized spring or well water, up to a year in sterilized river water, and over 9 months in sterilized seawater.

Sayamov and Zaidenov (1978) studied the survival of classical and El Tor *V. cholerae* in mineral waters from a spa at Matsesta (USSR). In raw mineral water, survival did not exceed 22 days for either biotype. In boiled mineral water at 20–24°C, initial concentrations of $9 \times 10^5$ per milliliter remained steady for 4 years for both biotypes. Other results from these experiments are given in table 17-4.

More remarkable are reports of prolonged survival in raw sewage. Altukhov and others (1975) studied a bath house in the USSR. *V. cholerae* (El Tor, Ogawa) was isolated from 49 percent of samples of wastewater from the bath house over a 13-month period. Repeated attempts to disinfect the wastewater system had no effect on *V. cholerae* isolation. There was no known cholera infection in the community. *V. cholerae* was not isolated from the incoming water supply, nor from large numbers of samples of human feces, water, fish, and frogs that were examined. Serological surveillance also failed to detect evidence of *V. cholerae* infection. *V. cholerae* was isolated from river water contaminated by the discharge from the bath house. In laboratory experiments, wastewater from the bath house

($BOD_5$ = 320 milligrams per liter, pH = 7.6) was inoculated with an El Tor (Ogawa) strain previously isolated from the bath house and with a reference strain of classical *V. cholerae* (Ogawa), and stored at 37°C. The concentration of El Tor organisms was $10^5$ per milliliter at the start, rose to over $10^8$ per milliliter after 3 days, and maintained this concentration up until 10 days when sampling was discontinued. The concentration of classical organisms was $10^3$ per milliliter at the start, rose to $10^5$ after 3 days, and fell back to $5 \times 10^2$ after 10 days. The investigation failed to discover how the bath house sewerage system became infected, but it was clear that, once infection had taken place, *V. cholerae* (El Tor, Ogawa) maintained itself in the warm sewage (20–25°C) and was remarkably resistant to disinfection.

A very similar experience was reported by Zaidenov and others (1976). A sewerage system serving a locomotive depot and a housing estate was investigated. Wastewater from the locomotive depot (450 cubic meters per day) was rich in oil products and passed through oil traps and a flotation chamber before being mixed with domestic sewage (150–250 cubic meters per day). The mixed sewage was then pumped to treatment fields. Because hot water was used in the locomotive depot, the sewage was warm, even in winter, and temperatures of 19–24°C were recorded throughout the year. The pH of the sewage was 7.1 to 9.3. Over a 17-month period 1,454 samples of sewage from various points in the system were examined, and 17 percent were positive for *V. cholerae* (El Tor, Ogawa). The wastewater from the locomotive depot was far more frequently infected (18–42 percent) than the domestic sewage (5 percent). The oil traps and flotation chamber were most frequently infected. The *V. cholerae* strain isolated was always the same and was nontoxigenic. Fecal examination of 2,708 people in the depot and the housing estate revealed only three infections with non-O1 *V. cholerae*. When one oil trap was isolated from the system, *V. cholerae* were shown to survive in it for 36 days (the temperature in this oil trap fell to 10°C after isolation from the sewerage system). In laboratory experiments, the El Tor strain isolated from the locomotive depot was inoculated into various wastewaters and stored at 18–24°C. In mixtures of oil plus water and diesel fuel plus water, survival was for over 14 months, with an initial concentration of 10 per milliliter. In domestic sewage, survival was less than 3 days; in locomotive depot wastewater, survival was over 39 days; and in dairy effluent (included for comparison), survival was less than 14 days. All experiments were performed with initial inocula of $10^4$ *V. cholerae* per milliliter. The source of infection of the sewerage system was not discovered. Repeated disinfection failed to clear *V. cholerae* from the network until massive doses of chlorine (to achieve 10

Table 17-8. $t_{90}$ *values in hours for various types of* V. cholerae *in various waters and wastewaters*

| *Type of water environment* | *Classical O1* | | | *El Tor O1* | | | *Non-O1* | | |
|---|---|---|---|---|---|---|---|---|---|
| | *No.* | *Arith. mean* | *Range* | *No.* | *Arith. mean* | *Range* | *No.* | *Arith. mean* | *Range* |
| Dechlorinated tap water | 8 | 22 | 3–48 | 8 | 49 | 2–163 | ND | ND | ND |
| Well water | 1 | 36 | NA | 13 | 116 | 5–264 | ND | ND | ND |
| Surface water | 8 | 18 | 0.16–36 | 10 | 53 | 1–230 | 4 | 8 | 8–8 |
| Seawater | 3 | 95 | 0.36–161 | 7 | 56 | 3–235 | ND | ND | ND |
| Sewage | 1 | 12 | NA | 9 | 66 | 8–240 | 2 | 8 | 8–8 |
| Sterilized well water, surface water or sewage | 7 | 34 | 3–65 | 9 | 59 | 32–168 | 6 | 39 | 31–50 |

No. Number of results.
Arith. mean Arithmetic mean.
ND No data.
NA Not applicable.

milligrams per liter throughout the system) and sulphuric acid (to lower sewage pH to 3–4) were added. Following this, no *V. cholerae* were isolated for the next 12 months.

Further evidence of multiplication and prolonged survival in some wastewater is provided by reports of the multiplication of *V. cholerae* (El Tor, Inaba) in a clinic septic tank in Japan (MMWR 1979) and the multiplication of *V. cholerae* (non-O1) in a trickling filter in Bangladesh (Daniel and Lloyd 1980*b*). These occurrences, and their relationship to environmental reservoirs of some atypical and non-O1 *V. cholerae*, await clarification.

### *A possible aquatic reservoir for* V. cholerae

Perhaps the greatest upset to traditional concepts of cholera epidemiology and bacteriology has come from the recent discoveries of *V. cholerae* and related organisms occurring in surface waters not known to be fecally contaminated or in areas where no human infection has been recorded. *V. cholerae*, El Tor and non-O1, were frequently isolated from wells, tanks, and rivers in India in the 1930s and 1940s, but their close relationship with classical *V. cholerae* O1, and their potential pathogenicity, were not recognized at that time (Read and Pandit 1941; Taylor and Ahuja 1938; Venkatraman, Krishnaswami and Ramakrishnan 1941).

Colwell, Kaper and Joseph (1977) reported the isolation of non-O1 *V. cholerae* from various parts of Chesapeake Bay (USA). Subsequently, Kaper and others (1979) described the ecology of non-O1 *V. cholerae* in Chesaspeake Bay in some detail. Concentrations were up to 7 per liter, and isolations were only made at sites with salinities between 0.4 and 1.7 percent. There was no correlation between *V. cholerae* counts and counts of total bacteria, coliforms, fecal coliforms, or salmonellae. *V. cholerae* were not especially associated with bottom sediment or oysters.

In a recent publication (Colwell and others 1980), data on *V. cholerae* isolations from various brackish and estuarine environments are summarized. *V. cholerae* isolations in Chesapeake Bay were dependent on salinity and temperature, with the highest recoveries (up to 46 per liter) being reported at salinities of 0.3 to 1.7 percent and during the summer when water temperatures were 28°C. *V. cholerae* isolations were not correlated with known fecal contamination, nor with fecal coliform counts, thus suggesting that *V. cholerae* "is an autochthonous species in the estuarine ecosystem". Both non-O1 *V. cholerae* serotypes and *V. cholerae* O1 (Inaba) have been isolated from Chesapeake Bay. *V. cholerae* O1 (Inaba) has also been isolated from Louisiana salt marshes. Some of the *V. cholerae* O1 and *V. cholerae* non-O1 strains isolated from the Chesapeake Bay and the Louisiana coast showed evidence of toxin production. A marked association of *V. cholerae* non-O1 with zooplankton was found both in the Chesapeake Bay and in surface water samples collected in Bangladesh.

Bashford and others (1979) and West, Knowles and Lee (1980) reported the isolation of up to several hundred *V. cholerae* per milliliter from streams and drainage ditches in Kent (England), including sites where there was no known sewage contamination. Isolations were more common during the summer. Except for one occasion, all isolations have been of non-O1 serotypes, and all have been nontoxigenic (J. Lee, personal communication). Müller (1978, 1979) isolated non-O1 *V. cholerae* from 33 percent of river water samples in the Federal Republic of Germany, but not from sewage treatment plant effluents. Isolations were more numerous in summer.

*V. cholerae* O1, atypical *V. cholerae* O1 and non-O1 *V. cholerae* have been isolated variously from freshwater, saline water, and wastewater in Australia, Bangladesh, Brazil, England, Germany, Guam, Japan, the USA, and the USSR (WHO Scientific Working Group 1980). Most of these isolates have been found to be nontoxigenic and nonpathogenic. They have been found in areas where cholera cases or infections are not known to occur (for example, Brazil, England, and the USA) and in waters that are not thought to have received any human fecal contamination (for example, England and the USA). It is very probable that some of these *V. cholerae* isolates are free-living aquatic organisms. Whether they are in any way related to human disease or to the epidemiology of cholera remains to be determined.

The speculation concerning a possible environmental reservoir for atypical and non-O1 *V. cholerae*, and possibly also for *V. cholerae* O1, has been increased by findings on the affinity of these organisms for chitin. Nalin and others (1979) found that about 70 percent of *V. cholerae* O1 organisms, which were shaken for 6 hours with powdered crabshell in a 4.2 percent salt solution at pH 6.2 and 20°C, adsorbed to the chitin particles. These adsorbed *V. cholerae* were then somewhat resistant to an acid environment simulating the stomach (pH) 1.6–1.8 for 13 minutes). *V. cholerae* also multiplied (>4 log increase) when incubated for 2 days at 37°C in 4.2 percent salt solution containing chitin. Other studies have shown that *V. cholerae* O1 (classical and El Tor) and non-O1 can

produce chitinase (Dastidar and Narayanaswami 1968) and that non-O1 *V. cholerae*, like *V. parahaemolyticus*, can adsorb to, and multiply on, chitinous fauna such as crab, shrimp, and zooplankton (Colwell, Kaper and Joseph 1977; Kaneko and Colwell 1973, 1975, 1978; Kaper and others 1979; Nalin 1976; Sochard and others 1979).

*In sweat*

Dodin and Félix (1972) found that *V. cholerae*, El Tor, was still viable after seven weeks at 28°C in human sweat and on gauze pads soaked in sweat and stored in humid conditions. From one quantitative experiment a $t_{90}$ of 215 hours at 28°C in sweat can be computed. This is much longer than typical $t_{90}$ values at that temperature (table 17-8). Dodin and Félix considered that these findings had considerable relevance to the epidemiology of cholera in arid areas of West Africa. Isaacson and Smit (1979) showed that *V. cholerae* (El Tor, Inaba) multiplied, and could survive for at least 120 hours, in pooled human sweat. Multiplication of *V. cholerae* in sweat was believed to have promoted the transmission of cholera among South African gold miners undergoing heat acclimatization (Isaacson and others 1974). It is not known whether *V. cholera* survives well in sweat on the skin.

*On surfaces*

*V. cholerae* survival on surfaces is usually limited because of the sensitivity of the organism to desiccation. Four studies on *V. cholerae* on various household items are summarized in table 17-9.

Table 17-9. *Survival of* V. cholerae *on surfaces*

| *Source* | *Biotype* | *Type of surface* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Felsenfeld (1965) | Classical and El Tor | Absorbent materials | | |
| | | Cotton | 28–30°C | 5–7 days |
| | | Chopsticks | | 2–3 days |
| | | Paper | | 2–3 days |
| | | Shoes | | 2–3 days |
| | | Silk | | 3–5 days |
| | | Non-absorbent materials | | |
| | | Aluminium foil | 28–30°C | 1 day |
| | | Coins | | 1 day |
| | | Tin cups | | 1 day |
| | | Plastic envelopes | | 1–2 days |
| | | China plates | | 1–2 days |
| | | Metal utensils | | 1–2 days |
| Gohar and Makkawi (1948) | Classical | Linen | Room temp. (Egypt) | 6 days |
| | | Wool | | 5 days |
| | | Leather | | 3 days |
| | | Paper and rubber | | 10 hours |
| | | Coins | | 6 hours |
| Pesigan, Plantilla and Rolda (1967) | El Tor | Frying pan | 30–32°C | 4 hours |
| | | China plates | | 4 hours |
| | | Pestle and mortar | | 4 hours |
| | | Drinking glass | | 4 hours |
| | | Metal utensils | | 24 hours |
| | | Kitchen knife | | 48 hours |
| | | Wooden chopping block | | 24 hours |
| Shousha (1948) | Classical | Cotton and cloth | Room temp. (Egypt) | 4 days |
| | | Bank note | | 3 days |
| | | Postage stamp | | 2 days |
| | | Coin | | 1 day |

*Note:* Older literature is reviewed by Pollitzer (1959).

a. Times given are those at which viable organisms could no longer be detected.

The longer persistence on absorbent materials, especially cotton, is interesting and suggests that clothing (especially clothing soaked in sweat) may act as a temporary habitat for *V. cholerae*. It is also noteworthy that survival times are markedly shorter than those reported for other enteric bacteria—for instance, *Shigella* (chapter 16)—on similar surfaces.

### *In soil*

Experiments in Israel (Gerichter and others 1975) found that *V. cholerae* (El Tor) in soil survived for up to 4 days when the soil was allowed to dry slowly, but for up to 10 days when the soil was regularly remoistened with uncontaminated sewage (initial concentrations were $10^7$ per gram of soil, and the storage temperature was 20–28°C). Nalin and others (1980) reported survival for over 6 days when *V. cholerae* (El Tor) was inoculated into sterile potting soil and stored at 26°C. In the same experiments it was found that common earthworms (*Lumbricus terrestris*) ingested *V. cholerae* in soil and subsequently died. *V. cholerae* multiplied in the earth worms and were isolated at concentrations up to $10^7$ per milliliter of worm homogenate.

### *On food and crops*

In looking at the potential of food for transmitting cholera, it is important to make the distinction between food that acts as a primary vehicle for cholera, becoming infected through direct contact with the stools of a case or carrier, and food that acts as a secondary vehicle of spread, becoming contaminated by polluted water. Most documented occurrences of foodborne cholera are of the second kind, and the most numerous of these incidents are those involving fish and shellfish. Alternatively, food can act as a secondary vehicle of cholera through the use of polluted water to irrigate or freshen vegetables.

The evidence for foods acting as the primary vehicles for cholera is very limited. This is to be expected because few studies have examined the domestic environment in a cholera area during an outbreak and carried out a systematic investigation of food for *V. cholerae*. Table 17-10 summarizes some literature on the survival of *V. cholerae* on food. It is clear that survival times of several days are commonly achieved, even at around 30°C. Survival is longest in moist, nonacidic, and sterile (that is, cooked) foods. Only two studies (Felsenfeld 1965; Neogy 1965) directly compared the survival of the classical and El Tor biotypes, and both found that El Tor survived for longer. It seems likely that some foods can and do act as a primary vehicle for spreading cholera, especially within the household or at feasts and markets.

## Inactivation by Sewage Treatment Processes

There is very little information on the fate of *V. cholerae* in sewage treatment plants partly because, as mentioned above, most people with cholera produce no sewage; therefore *V. cholerae* is only very rarely found in sewage, and even then in low concentrations.

Flu (1921) studied seeded *V. cholerae* in septic tanks in Batavia (now Jakarta; Indonesia). A total of five septic tanks were studied, and in only one was *V. cholerae* detected in the effluent. Early studies reviewed by Kabler (1959) reported a 98 percent reduction of *V. cholerae* in an activated sludge plant.

Kott and Betzer (1972) studied a 70-liter model waste stabilization pond with a retention time of 5 days. The pond was fed with diluted sewage ($BOD_5 = 200$ milligrams per liter) spiked with *V. cholerae* (El Tor). Influent coliform and *V. cholerae* concentrations were $3 \times 10^6$–$8 \times 10^8$ and $1 \times 10^3$–$8 \times 10^3$ per 100 milliliters, respectively. Effluent coliform and *V. cholerae* concentrations were $8 \times 10^4$–$4 \times 10^7$ and 0–2 per 100 milliliters respectively. The addition of 8 milligrams per liter of chlorine to the waste stabilization pond effluent eliminated all remaining *V. cholerae*.

Daniel and Lloyd (1980*a*) studied two Oxfam Sanitation Units in refugee camps near Dacca (Bangladesh). These units treated very strong sewage (17,000 and 7,400 milligrams of suspended solids per liter) in two unbaffled, flexible tanks connected in series. Each tank had a volume of 18 cubic meters, and the flow of sewage was 2.5 to 3 cubic meters per day. Thus, the total mean retention times were 12–15 days. The geometric mean inflowing concentrations of non-O1 *V. cholerae* at the two camps were $2.6 \times 10^3$ and $1.6 \times 10^2$ per 100 milliliters, respectively. The geometric mean effluent concentrations were 6.5 and 5.3 per 100 milliliters. Thus, overall removal rates at the two camps were 99.8 and 96.4 percent, respectively. These removal rates give $t_{90}$ values of 106 and 257 hours, respectively, which are longer than those reported in table 17-7, especially if the warm ambient temperature is taken into account. This suggests either short-circuiting in the tanks, which is quite probable, or non-O1 *V. cholerae* multiplication in the warm sewage in the tanks.

Table 17-10. *Survival of* V. cholerae *on food and crops*

| *Source* | *Biotype* | *Type of food* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| | | ***A. Meat*** | | |
| Cheng (1963) | El Tor | Beef | Day 1: 22°C<br>Thereafter: 3–4°C | 5 days |
| Felsenfeld (1965) | El Tor and classical | Raw beef | 2–4°C<br>28–30°C | 5–7 days<br>1–2 days |
| | | Cooked beef | 2–4°C<br>28–30°C | 1–2 weeks<br>3–7 days |
| | | Sausages (surface and inside) | 2–4°C<br>28–30°C | 1 day<br>1 day |
| Pesigan, Plantilla and Rolda (1967) | El Tor | Raw meat | 5–10°C<br>30–32°C | 4–9 days<br>2–4 days |
| | | Cooked meat | 5–10°C<br>30–32°C | 3–5 days<br>2–5 days |
| | | ***B. Fish*** | | |
| Cheng (1963) | El Tor | Lice-eye fish<br>Sliced sword-fish | Day 1: 21.5°C<br>Thereafter: 4°C | 16 days<br>10 days |
| Felsenfeld (1965) | El Tor and classical | Shrimp | 2–4°C<br>28–30°C | 1–3 days<br>1–2 days |
| | | Catfish<br>Raw | 2–4°C<br>28–30°C | 1–2 weeks<br>2–4 days |
| | | Dried | 2–4°C<br>28–30°C | 3–5 days<br>1–2 days |
| | | Salted | 2–4°C<br>28–30°C | 1–2 days<br>1 day |
| | | Cooked | 2–4°C<br>28–30°C | 2–7 days<br>1–6 days |
| Pesigan, Plantilla and Rolda (1967) | El Tor | Various fish and shellfish | 5–10°C<br>30–32°C | 4–9 days<br>2–4 days |
| | | ***C. Vegetables and fruit*** | | |
| Cheng (1963) | El Tor | Horseradish<br>Cucumber<br>Tomato<br>Orange | Day 1: 22°C<br>Thereafter: 3–4°C | 21 days<br>23 days<br>16 days<br>14 days |
| El Shawi and Thawaini (1967) | El Tor | Date<br>Melon | Room temp. (Iraq) | 3 days<br>2 days |
| Felsenfeld (1965) | El Tor and classical | A comprehensive survey of a wide range of cooked and uncooked fruits and vegetables | 2–4°C<br>28–30°C | Up to 4 weeks<br>Up to 7 days (except inside melon, which was 2 weeks); survival was especially long on cabbage, cucumber, eggplant, melon, okra, peas, and potatoes. |

Table 17-10 (*continued*)

| *Source* | *Biotype* | *Type of food* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Gerichter and others (1975) | El Tor | Parsley | 20–26°C | 1 day |
| | | Tomato and carrot | | 1.5 days |
| | | Cucumber, pepper, and okra | | 1–2 days |
| | | Lettuce | | 2–3 days |
| | | | | Mean death rates for all the above were 4–6 log units per day |
| | | Parsley | | |
| | | Wet | 20–28°C | 2 days |
| | | Dry | | 1 day |
| | | Lettuce | | |
| | | Group of leaves | 18–26°C | 68 hours |
| | | Single leaf | | 44 hours |
| | | Tomato | | |
| | | in sunlight | 22–30°C | 4 hours |
| | | Parsley | 4°C | 2 days |
| | | Lettuce | | 4 days |
| Gohar and Makkawi (1948) | Classical | Date | Room temp. (Egypt) | 4 days |
| | | Vegetables | | 6 days |
| Neogy (1965) | El Tor and classical | Papaya | Room temp. (India) | 1 day |
| | | Cucumber | | >1 day |
| | | Pineapple | | 15 minutes |
| | | Boiled rice soaked overnight | | 1 hour |
| Pesigan, Plantilla and Rolda (1967) | El Tor | Cooked fruit and vegetables | 5–10°C | 3–5 days |
| | | | 30–32°C | 2–5 days |
| | | | 5–10°C | 2–3 days |
| | | Fresh fruit | 30–32°C | 1 day |
| | | Fresh vegetables | 5–10°C | 6–9 days |
| | | | 30–32°C | 2–5 days |
| Prescott and Bhattacharjee (1969) | El Tor | Lime, lemon, and date | 20–25°C | 1 hour |
| | | Orange, grape, fig, raisin, and tomato | | 1 day |
| | | Banana, guava, papaya, onion, eggplant, pea, celery, green bean, bean sprout, and rice. | | 2–5 days |
| | | Okra, lima bean, pumpkin, and potato | | 6–8 days |
| Shousha (1948) | Classical | Onion and date | Room temp. (Egypt) | 4 days |
| | | Garlic, rice, lentil, and grape | | 3 days |
| | | Orange and lemon | | 7 hours |

Table 17-10 (*continued*)

| *Source* | *Biotype* | *Type of food* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| | | ***D. Milk and Milk products*** | | |
| Felsenfeld (1965) | El Tor and classical | Butter, unsalted | 2–4°C<br>28–30°C | 1–2 weeks<br>1 week |
| | | Cheese | 2–4°C<br>28–30°C | 2–3 weeks<br>1 week |
| | | Custard | 2–4 °C<br>28–30°C | 3–4 weeks<br>1–2 weeks |
| | | Ice cream | 2–4°C<br>28–30°C | 3–4 weeks<br>5–7 days |
| | | Milk | 2–4°C<br>28–30°C | 3–4 weeks<br>1–3 weeks |
| Lema, Ogawa and Mhalu (1979) | El Tor | Milk | 4°C<br>30°C | 3 weeks<br>4 days |
| Neogy (1965) | El Tor and classical | Milk desserts | Room temp. (India) | 1 day |
| Pesigan, Pantilla and Rolda (1967) | El Tor | Milk, ice cream, and butter | 5–10°C<br>30–32°C | 1 week- > 2 weeks<br>5–14 days |
| Prescott and Bhattacharjee (1969) | El Tor | Milk desserts | 20–25°C | 1–2 days |
| Shousha (1948) | Classical | Milk<br>Sour milk<br>Butter<br>Cheese | 4°C<br>Room temp.<br>[illegible] | > 2 days<br>2 hours<br>> 2 days<br>7 hours |
| | | ***E. Other foods*** | | |
| El Shawi and Thewaini (1967) | El Tor | Barley and wheat | Room temp. (Iraq) | 2 days |
| Felsenfeld (1965) | El Tor and classical | A comprehensive survey of a wide range of cooked and uncooked foods | 2–4°C<br>28–30°C | Up to 4 weeks<br>Not more than 7 days, except for coconut cream (10 days), coconut dishes (3 weeks), and noodles (2 weeks) |
| Gohar and Makkawi (1948) | Classical | Honey and treacle | Room temp.<br>[illegible] | 3 hours |
| Neogy (1965) | El Tor and classical | Sweet and sour curd<br>[illegible] and *sandesh* | Room temp. (India) | 5 minutes<br>1 day |
| Pesigan, Plantilla and Rolda (1967) | El Tor | Cooked noodles, rice cake, and jam | 5–10°C<br>30–32°C | 3–5 days<br>2–5 days |

Table 17-10 (*continued*)

| *Source* | *Biotype* | *Type of food* | *Temperature* | *Survival*[a] |
|---|---|---|---|---|
| Prescott and Bhattacharjee (1969) | El Tor | Wheat and nuts | 20–25°C | 3 days |
| | | Spices | 20–25°C | 1–5 days |
| Shousha (1948) | Classical | Sugar | Room temp. (Egypt) | 4 days |
| | | Bread | | 3 days |
| | | Honey | | 2 days |
| | | ***F Beverages*** | | |
| El Shawi and Thewaini (1967) | El Tor | Soft drinks | Room temp. (Iraq) | 1 day |
| Felsenfeld (1965) | El Tor and classical | Beer, carbonated water, carbonated soft drinks, lime and whisky | 2–4°C | 1 day |
| | | | 28–30°C | 1 day |
| | | Cocoa | 2–4°C | 1–2 weeks |
| | | | 28–30°C | 3–5 days |
| | | Coffee | 2–4°C | 1–2 days |
| | | | 28–30°C | 1 day |
| | | Ice cubes | 2–4°C | 4–5 weeks |
| | | Lemonade | 2–4°C | 2–3 weeks |
| | | | 28–30°C | 5–7 days |
| | | Tea | 2–4°C | 1 week |
| | | | 28–30°C | 2–3 days |
| Lema, Ogwa and Mhalu (1979) | El Tor | Coconut fluid | 4°C | 4 days |
| | | | 30°C | 2 days |
| | | Beer, gin, and traditional alcoholic beverages *chibuku* (maize and beans) and *mbege* (bananas and millet) | 4°C | 1 hour (except for *mbege*, in which survival was 2 days) |
| | | | 30°C | 1 hour |
| Pesigan, Pantilla and Rolda (1967) | El Tor | Coca cola | 5–10°C | 2 days |
| | | | 30–32°C | 4 hours |
| Prescott and Bhattacharjee (1969) | El Tor | Coca cola | 20–25°C | 1 day |
| | | Rosewater | | 2 days |
| | | Ground coffee | | 1 hour |
| | | Tea leaves | | 1 day |

*Note:* Older literature is reviewed by Pollitzer (1959).

a. Times given, for instance, as 2 days are durations at which viable organisms could no longer be detected. Times given as >2 days indicate that organisms were still viable at that time but that sampling was discontinued.

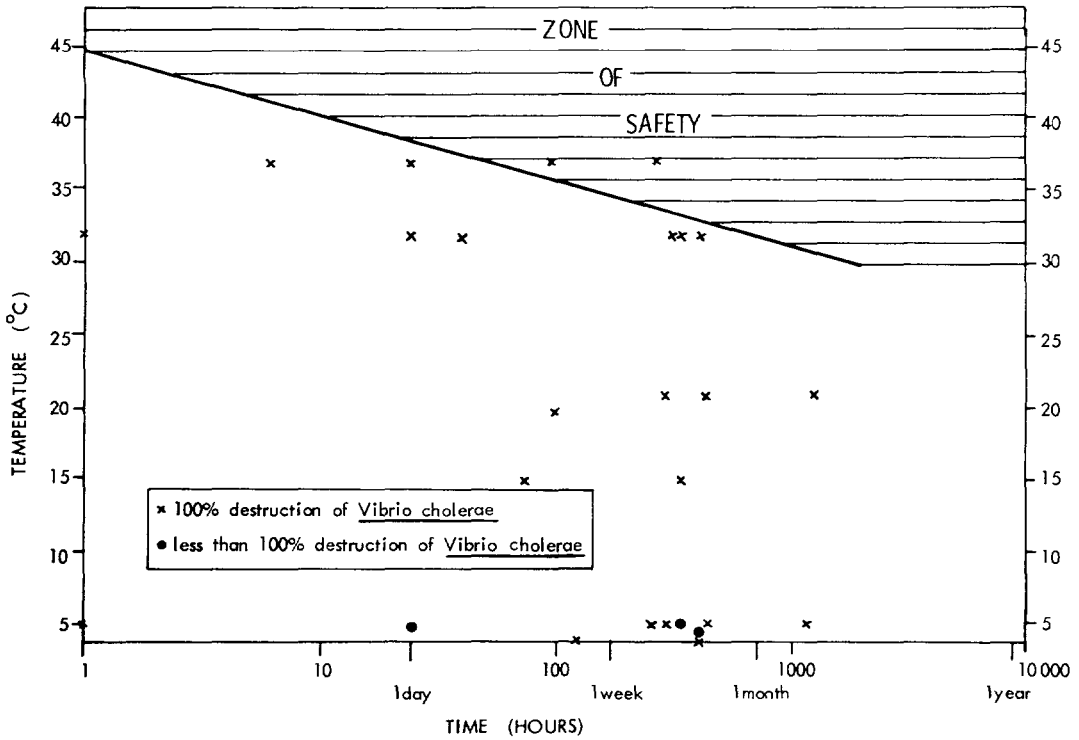


Figure 17-3. *The influence of time and temperature on* V. cholerae. The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

Daniel and Lloyd (1980*b*) reported that small trickling filters were installed to treat further the effluents from these Oxfam Sanitation Units. Influent concentrations (effluents from the second tank of the main unit (non-O1 *V. cholerae* were 3–9 per 100 milliliters, while effluents from the trickling filters contained 3–2,400 per 100 milliliters. The authors concluded that non-O1 *V. cholerae* was multiplying in ponded sewage in the trickling filters.

## Inactivation by Night Soil and Sludge Treatment Processes

No reports of *V. cholerae* reduction during night soil or sludge treatment were located. The data given in table 17-6 suggest that *V. cholerae* will be eliminated by any process having a retention time of appreciably more than 5 days in a warm climate. Time-temperature combinations lethal to *V. cholerae* are given in figure 17-3. It appears that *V. cholerae* will be eliminated by almost any sludge digestion, composting, or storage process and will certainly be removed far more readily than *E. coli* and other fecal indicator bacteria (chapter 13).

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# 18

# *Yersinia* and Yersiniosis

IT IS ONLY in the last few years that *Yersinia enterocolitica* has been recognized as an etiological agent of acute enteritis. It may therefore be grouped with *Campylobacter* (chapter 12) and the pathogenic forms of *Escherichia coli* (chapter 13) as a "new" bacterial agent of diarrheal disease—although, unlike these other two, *Yersinia* is unlikely to prove to be a major cause of diarrhea.

## Description of Pathogen and Disease

The genus *Yersinia* comprises three species, each of which is essentially an animal parasite that sometimes infects man. *Y. pestis* is the causative agent of human plague and is primarily a parasite of rodents. *Y. pseudotuberculosis* is primarily a parasite of guinea pigs and other rodents and occasionally infects humans, causing a variety of pathological conditions. *Y. enterocolitica* causes gastroenteritis and other symptoms in man and infects a wide range of wild and domestic animals. Only *Y. enterocolitica* will be dealt with in this chapter because it alone out of the three is primarily an excreted pathogen.

### *Identification*

Yersiniosis is caused by bacterial infection primarily of the intestine and blood circulatory system. The causative organism *Y. enterocolitica* most commonly gives rise to an acute enterocolitis and septicaemia. Diarrhea may be the only symptom, or it may be accompanied by abdominal pain, fever, or both. It is often difficult to distinguish the disease clinically from other enteric infections such as those produced by certain shigellae and salmonellae. However, the acute infection sometimes resembles appendicitis, and in such cases surgery will reveal inflammation of the appendix together with terminal ileitis and mesenteric adenitis. Other less frequent forms of infection include pyuria, polyarthritis, and conjunctivitis (that is, it is one of the causes of Reiter's syndrome, which involves all three), and also infections of skin, wounds, and throat.

Diagnosis is by isolation of the bacteria from fecal or blood specimens. Some of the selective media used for isolation of salmonellae are appropriate for recovery from feces, but with the important difference that an incubation temperature in the range of 22 to 29°C, rather than 37 to 42°C, is optimal. Presumptive yersinias must be typed biochemically and may be further characterized by serotyping and phage typing.

### *Occurrence*

Although the organism was first isolated in the USA in 1923, it was not recognized as a human pathogen until the early 1960s. The first human cases of infection were diagnosed in France, Belgium, and Sweden in 1963; since then it has been identified as a human pathogen in thirty countries throughout the world. In Europe and North America, *Y. enterocolitica* may be responsible for between 1 and 3 percent of recorded acute cases of gastroenteritis, but no comparable data are yet available from developing countries. It is certain that its recorded incidence and geographical distribution are artificially low as a result of widespread inadequacies in diagnostic expertise and, hence, reporting.

### *Infectious agent*

*Y. enterocolitica* possesses all the characteristics of the Enterobacteriaceae, to which family yersinias were assigned in 1966. It is a Gram-negative ovoid or rod-shaped organism measuring 0.8–3.0 micrometers by 0.8 micrometers. It is a facultative anaerobe. About thirty-four serotypes have been recognized, of which a number are characteristically associated with particular non-human animal species, whereas others are associated with several human and nonhuman animals. Serotypes O3, O8, and O9 are particularly associated with human disease.

*Reservoirs*

It is likely that wild animals including shrews, red foxes, hares, and beavers form a natural reservoir for *Y. enterocolitica*; domestic animals from which the pathogen has been isolated include cattle, sheep, pigs, dogs, chinchillas, and geese. The number of animal species identified as affected by yersiniosis continues to rise and now includes primates other than man. It has also been demonstrated that bivalves such as mussels and oysters effectively concentrate these bacteria, although they are unlikely to multiply in them in seawater. It has been suggested that this organism follows the same epidemic and epizootic pathways as the salmonellae (see chapter 15).

*Transmission*

The means by which *Y. enterocolitica* is spread are still not proven. Fecal-oral transmission is most probable, and respiratory transmission is also a possibility. Foodborne and waterborne outbreaks have been reported.

In one experiment with a human volunteer, a dose of $3.5 \times 10^9$ organisms was required to produce an infection (Morris and Feeley 1976). Under natural conditions it is likely that considerably smaller doses will produce infection in a proportion of the population. In any case, high infective doses may be obtained in contaminated food since *Y. enterocolitica* multiplies readily in many foods, even under refrigeration (Kendall and Gilbert 1980).

*Incubation period*

Three to seven days is the normal range.

*Period of communicability*

In infants and young children, the watery diarrhea may persist for 3 to 14 days. In untreated cases excretion of the organism may continue for 2–3 months. A chronic carrier state has not been demonstrated in man, but certainly exists in other animals.

*Resistance*

The infection has been identified in people of all age groups, but there is a much higher incidence in young children.

*Epidemiology*

Because *Y. enterocolitica* is a recently recognized pathogen, and possibly not a terribly important one at that, knowledge of its epidemiology is very limited. The more it is looked for, the more it is found, and the worldwide picture of its epidemiology will continue to build up slowly over the next decade. In 1966 only twenty-three cases of infection with *Y. enterocolitica* were reported worldwide (Highsmith, Feeley and Morris 1977). By 1974 this had increased to over 4,000, with most cases still being reported from Europe, where many laboratories routinely screen stool specimens for this pathogen.

The first documented foodborne outbreak of yersiniosis occurred in New York (USA; Wakelee and others 1977). Serotype O8 was isolated from children suffering from abdominal pain, fever, and, in some, diarrhea and slightly inflamed appendixes. Two hundred and eighteen children attending five county schools were affected. Out of ten possible sources of infection including water, food, and milk, only chocolate milk was associated with the illness. Serotype O8 was isolated from a carton of chocolate milk during the investigation. In the dairy plant, which supplied the schools, chocolate syrup was manually added to a large open vat of pasteurized milk. Morris and Feeley (1976) reviewed the evidence of foodborne yersiniosis. They noted that the organism is commonly found in specimens from swine slaughterhouses and has been isolated from samples of market meat, vacuum-packed beef, mussels, oysters, and ice cream. It has also been found in nonchlorinated well water used for drinking purposes.

Flies may play a role in contaminating food and thus in initiating foodborne transmission. Fukushima and others (1979) isolated *Y. enterocolitica* O3 from flies caught in the piggeries and the kitchens of two farms in Japan. *Y. enterocolitica* O3 was also isolated from a ham hung in one piggery and from the feces of pigs in both piggeries. All O3 strains were of a common phage type.

Studies of the occurrence of the various serotypes and phage types of *Y. enterocolitica* have cast doubt upon some of the more simple explanations of yersiniosis epidemiology (Mollaret 1976). Caprioli, Drapeau and Kasatiya (1978) isolated and typed *Y. enterocolitica* from 31 specimens of water and food and from 143 human specimens, in Quebec (Canada). Seventy-four percent of all isolates from human sources were serotype O3, and this was the only serotype isolated from children under 4 years old. However, no serotype O3 isolates were obtained from any environmental samples. It remains unclear whether yersiniosis is primarily an infection of nonhuman animals transmitted infrequently to man, often via food (as with salmonellosis); whether man is his own reservoir for

specific serotypes; or whether, as suggested by Mollaret (1976), animals and man both contaminate and are infected from shared environmental reservoirs.

## Control Measures

Not enough is known about the epidemiology and transmission of *Y. enterocolitica* to allow any confident recommendations about control. General techniques of environmental hygiene, food hygiene, and sanitation are likely to be most effective.

## Occurrence and Survival in the Environment

Little is known about the occurrence and survival of *Y. enterocolitica* in the environment. The organism has been isolated from a variety of environmental samples, especially food and water, but the isolated serotypes are often not those especially associated with human disease (for instance, see Caprioli, Drapeau and Kasatiya 1978).

Lassen (1972) isolated *Y. enterocolitica* from ten out of fifty drinking water samples in Norway. Saari and Quan (1976) surveyed rivers, reservoirs, and private wells in Colorado (USA). Forty-seven percent of 125 river sites, 11 percent of 26 reservoirs, and 1 percent of 563 wells tested contained this organism, with up to 5 distinct strains per water source. The authors noted that although *Y. enterocolitica* commonly occurs in Colorado waters, serotypes pathogenic to humans are rarely found. Harvey and others (1976) isolated *Y. enterocolitica* at ten out of thirty-four stream and lake sites in a mountainous area of California (USA) and considered that the organisms probably derived from wild animals. Kapperud (1977) isolated *Y. enterocolitica* from nine out of twenty-nine surface water samples collected in areas of Norway and Denmark, where the organism infected small rodents, shrews, and foxes.

A few outbreaks of cases of gastroenteritis in developed countries have been tentatively linked to waterborne transmission of *Y. enterocolitica*. For instance, Eden and others (1977) reported the isolation of *Y. enterocolitica* from well water at a Montana (USA) ski resort soon after an outbreak of gastroenteritis of unknown cause.

Schiemann (1978) examined 2,588 surface and well water samples, submitted to the Toronto (Canada) public health laboratory for routine bacteriological tests, for the presence of *Y. enterocolitica*. The organism was identified in a total of 44 samples taken from dug wells, drilled wells, a spring, lakes, bathing water, and a municipal supply. Five positive well samples were from treated supplies: three chlorinated and two filtered. The single positive spring sample was from a chlorinated supply, as was the single municipal sample, which was additionally treated by a home-installed filter. Water samples yielding *Y. enterocolitica* showed only light coliform contamination (the median fecal coliform count was 1 per 100 milliliters), and 25 percent of *Y. enterocolitica* positive samples were negative for both total and fecal coliforms. Bearing in mind that the author had no control over sample collection and did not inspect the water sources concerned, these findings suggest that at least some strains of *Y. enterocolitica* behave very differently from coliforms in water systems and may survive some water treatment processes.

There is, as yet, very little information on the ability of *Y. enterocolitica* to survive in the environment. Dominowska and Malottke (1971) studied the survival of *Y. enterocolitica* inoculated into various types of water and kept outdoors in Poland. The average survival time in unfiltered surface waters was 38 days in spring and 7 days in summer. In filtered water the bacteria survived 197 days in spring and 184 days in summer. Under laboratory conditions at 18–22°C, *Y. enterocolitica* at an inoculum of $10^3$ per milliliter survived in tap water for about 7 days and in lake water for 28 days. However, larger inocula allowed survival beyond 77 days.

Schillinger and McFeters (1978) found a 2 log reduction in *Y. enterocolitica* concentrations in stream water at 5–8.5°C after 14 days, compared with a 3–5 log reduction of *E. coli* over the same period ($t_{50}$ values were 63 hours for *Y. enterocolitica* and 25 hours for *E. coli*). In chlorinated tap water, however, *E. coli* ($t_{50} = 0.5$ hours) survived a little longer than *Y. enterocolitica* ($t_{50} = 0.4$ hours).

Highsmith and others (1977) demonstrated that *Y. enterocolitica* could grow in sterile distilled water at 4°C, 25°C, and 37°C, but not at 42°C, and that the organism could survive in sterile distilled water for over 18 months at 4°C. This, and other evidence presented by Highsmith and her coworkers, suggests that *Y. enterocolitica* may survive for considerable periods in cool, clean waters with a minimum of bacterial competition. By contrast, in sterilized saline waters (salinities = 0.5, 2.0, and 3.5 percent) and at various temperatures (4, 25, 37°C), an initial inoculum of $1.5 \times 10^7$ *Y. enterocolitica* per milliliter failed to survive for more than 4 days, with a 6 log reduction after only 1 day (Jamieson, Madri and Claus 1976).

No data are available on the survival of *Y. enterocolitica* in feces or sewage.

## Inactivation by Sewage Treatment Processes

No information is available on the destruction of *Y. enterocolitica* in sewage treatment plants or on the occurrence of this organism in sewage. In a laboratory study *Y. enterocolitica*, serotype O6, was inoculated continuously into a model activated sludge plant (volume 1 liter, retention time 8.3 hours) at a concentration of $10^6$ per milliliter. *Y. enterocolitica* and coliform removals were compared at various temperatures. Removal rates were 99.8 percent and 97 percent at 5°C, 95 percent and 80 percent at 20°C, and 99.6 percent and 98 percent at 30°C for *Y. enterocolitica* and coliforms, respectively (Lloyd, personal communication).

## Inactivation by Night Soil and Sludge Treatment Processes

No information is available on the destruction of *Y. enterocolitica* by night soil and sludge treatment processes or on the occurrence of this organism in night soil and sludge. Available laboratory techniques are still inadequate for this type of investigation.

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# *Section III.*
# *Excreted Protozoa*

Chapter

# 19

# *Balantidium* and Balantidiasis

THREE PROTOZOAL INFECTIONS of the human intestinal tract are described in this book. Two of them, amebiasis due to *Entamoeba histolytica* (chapter 20) and giardiasis due to *Giardia lamblia* (chapter 21), are of major public health importance in many countries. The third, balantidiasis due to *Balantidium coli*, is relatively rare and is included for completeness. Two others about which little is known, isosporiasis due to *Isospora belli* and sarcocystiasis due to *Sarcocystis* species, are also rare as significant diseases of man and are omitted from this study. Also omitted are the nonpathogenic and rarely pathogenic amebae, and some flagellates of no public health importance.

## Description of Pathogen and Disease

Balantidiasis is a little-studied infection, and therefore the following sections lack much of the detail that is found in other chapters.

### *Identification*

Balantidiasis is an infection of the large intestine by the ciliate protozoon, *Balantidium coli*. In many infections (perhaps 80 percent) *Bal. coli* lives as a commensal in the lumen of the colon and causes no symptoms. Less frequently invasion of the colonic mucosa takes place, giving rise to a disease known as balantidial dysentery. Sufferers may present with diarrhea, sometimes bloody, and abdominal discomfort. The colonic lesions are grossly similar to those of amebic dysentery, but may reach the lymphatic vessels deep in the intestinal wall. The parasites may then penetrate to the regional lymph nodes, where a mild reaction occurs. Hematogenous spread to distant organs does not occur, in contrast with *Entamoeba histolytica*, but *Balantidium* may attack the terminal ileum and also cause acute appendicitis. Vaginitis and cystitis have also been observed. An inadequate diet may exacerbate the pathogenesis. Death may occur through the development of extensive ulceration and gangrenous changes, and may result from hemorrhage and dehydration. Reports of mortality range from 5 to 35 percent among clinical cases in the tropics. Treatment is by antibiotics, particularly tetracycline or ampicillin.

Diagnosis depends on demonstrating the characteristic *Bal. coli* trophozoites or cysts in the stools. The stool examined should ideally be fresh, but cysts and, under favorable conditions, trophozoites can be detected in fecal material preserved in 5 percent formol-saline.

### *Occurrence*

This infection, although rare, is found worldwide, most commonly in the tropical and subtropical zones (Arean and Koppisch 1956). In areas where sanitation is poor and where pigs associate closely with man, prevalence of infection may exceed 20 percent. *Bal. coli* infects all ages, with the highest prevalence in endemic areas occurring among teenagers and adults.

### *Infectious agent*

*Bal. coli* is the only parasitic ciliate of man. It is a flattened oval organism covered with cilia, with a gullet at the anterior end (figure 19-1). The trophozoite is 30–170 micrometers long by 25–120 micrometers broad. As in *Entamoeba* infection, cysts are found in the large intestine and passed in the formed stool. The cysts are ovoid or spherical and measure 45–65 micrometers in diameter. The trophozoite also may be an infective stage; it can live several days outside the host (Svensson 1955) and can withstand passage through the guinea pig stomach.

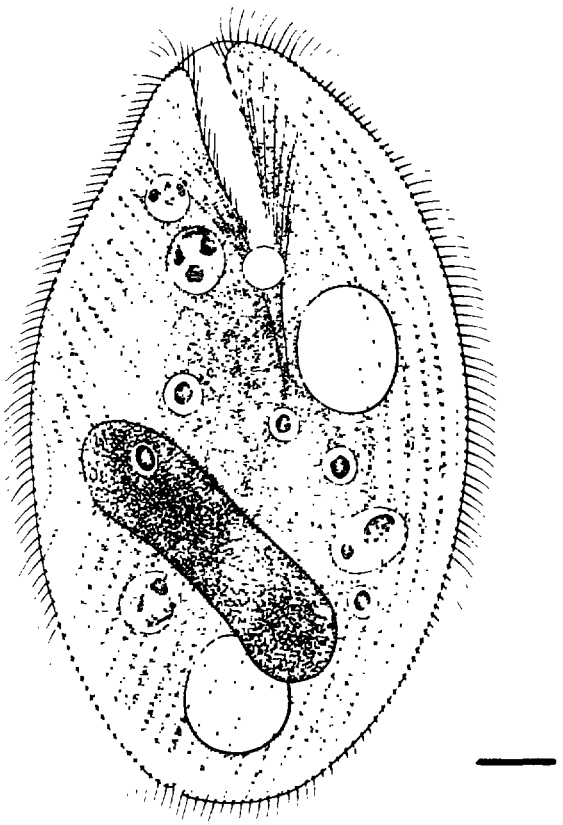

Figure 19-1. *Drawing of a trophozoite of* Balantidium coli. Scale bar = 10 micrometers. (From: Wenyon, C. M. (1926). *Protozoology*, vol. 2. London: Bailliere, Tindall and Cassel. Reproduced by courtesy of the publishers)

*Reservoirs*

Balantidiasis is a zoonosis. Many mammals are naturally infected, but it is the pig, and to a lesser extent the rat, that act as the main reservoirs for human infection (Awakian 1937; Ayeni 1973; Letonja and others 1975; Misra and others 1972). Prevalence among pigs is typically 50–100 percent, with 80 percent of pigs in the UK infected (Knight 1978). The majority of cases have a history of contact with pigs, but cases do occur in Moslem countries such as Iran. It is possible that only certain animal strains can infect man and then only under particular conditions of host susceptibility. Human strains will infect experimental animals including apes, pigs, cats, rodents, and guinea pigs (for instance, Westphal 1971), but it has not yet proved possible to infect man experimentally with cysts of human, simian, or porcine strains. *Bal. coli* has also been found in wild rats, and some human infections are thought to be derived from this source. The almost universal association of rats with domestic pigs may be relevant in this context. Sewer rats (*Rattus norvegicus*) are susceptible to infection with *Bal. coli* from man, pigs, or wild rats but are most susceptible to rat strains.

*Transmission*

It is assumed that the cyst of *Bal. coli* is the important resistant stage involved in transmission. Contamination of food, drinking water, and utensils by feces of pigs and, probably more important, by fecal material from persons carrying the infection, as well as direct fecal-oral contact appear to be the main modes of transmission. The same comments on transmission made for *Ent. histolytica* (chapter 20) apply here, though fewer cysts are produced per infected person in balantidiasis, and the proportion of infected persons producing cysts is also lower. Thus the incidence of *Bal. coli* is probably lower than that of *Ent. histolytica.* An early report suggests that the trophozoite is capable of passing the stomach barrier, so that acute cases, not producing cysts, could possibly be infective to people closely associated with them. However, other work has suggested that trophozoites are highly sensitive to pH values below 5. The median infective dose ($ID_{50}$) is not known, as man has not been successfully experimentally infected (Young 1950). It may well be comparable to that of *Entamoeba* and *Giardia*: 10–100 cysts (chapters 20 and 21).

*Prepatent and incubation periods*

Because man has not been infected experimentally and detailed epidemiological studies are lacking, the prepatent and incubation periods are not known.

*Period of communicability*

The disease is communicable for as long as the infection persists, although cysts are not often found in the stool. A 5-year history of chronic diarrhea, revealed as balantidial dysentery, has been described in one patient in Northern Ireland (Kennedy and Stewart 1957).

*Resistance*

Man appears to be a very resistant host. High prevalences of balantidiasis are found only in the exceptional circumstances of close contact with pigs. Attempts to infect man with cysts experimentally have so far failed. Disease may occur only when there is malnutrition or intercurrent infection.

### *Epidemiology*

Balantidiasis is extremely rare in many countries, and its epidemiology is not well described. An endemic focus has been reported from the Seychelles (Nuti, de Comarmond and de Bac 1980).

Most epidemiological data come from Papua New Guinea and Irian Jaya (Indonesia). Balantidiasis in the highlands of both countries is common due to the practice of keeping, and often living with, large herds of domestic pigs (Bayliss Smith and Feachem 1977; Feachem 1973). Human prevalences in different communities in Papua New Guinea range from 2 to 29 percent and are twice as high in females as in males (Radford 1973). This is due to the fact that pigs sleep in the "women's houses" with the adult females and children. In the mountains of Irian Jaya, balantidiasis is especially prevalent where domestic or semidomestic pig populations are high and where altitude and harsh climate create a greater need for pigs to shelter in human houses at night (Couvée and Rijpstra 1961; van der Hoeven and Rijpstra 1957).

Following a typhoon in May 1971 there was an outbreak of balantidiasis involving 110 persons on Truk (Caroline Islands; Pacific Islands Trust Territory) described by Walzer and others (1973). The patients presented with gastrointestinal symptoms, and there was no serious morbidity. Both tetracycline and metronidazole were used in treatment, but there was no evidence that either was effective. The epidemic terminated spontaneously in early July. In 1970 there had been 410 cases of amebiasis and 4 of balantidiasis. Before the typhoon in 1971 there had been 1 case of balantidiasis. During the outbreak the youngest case was 2 months old and the oldest 70 years. Highest rates of attack were noted in the 1–4, 30–39, and 50–59 age groups, indicating that the source of transmission was something common to all age groups.

The 30,000 people of the Truk archipelago (119 square kilometers) lived in overcrowded conditions, often 15 persons per household. Privies discharging into the lagoon were shared by several families, and there was indiscrimate defecation by children around the houses. Pigs were kept by most householders, and *Bal. coli* was found in the fresh feces of four of six pigs examined. During the typhoon most of the houses in Truk were destroyed, together with their roof-water catchment systems, and this led to a reliance on wells and streams highly contaminated with pig feces. Cases of balantidiasis occurred almost simultaneously in widely separate areas, where the only common factors were the occurrence of the typhoon, the presence of pigs, and reliance on the roof-water catchment systems. Walzer and his colleagues considered that the balantidiasis outbreak occurred because the inhabitants used ground and surface water supplies, contaminated by pigs, after their relatively clean sources of water were destroyed by the typhoon.

## Control Measures

Balantidiasis is not a major public health problem, and its control has not been studied. All the comments made about amebiasis and giardiasis control (chapters 20 and 21) may apply to balantidiasis control.

### *Individual*

Mass chemotherapy has not been tried, and there is no vaccine. Individual protection may be achieved by personal and domestic cleanliness and by care in the choice and preparation of drinking water and vegetables. Pig farmers may be especially at risk.

### *Environmental*

A single study in Venezuela showed that preschool children in houses with inside water and washing facilities had a balantidiasis prevalence of 4 percent, whereas those in other houses had a prevalence of 9 percent (van Zijl 1966). Confounding socioeconomic variables were not controlled.

The importance of hygienic excreta disposal to prevent transmission of the cysts directly to other individuals is accepted. Equally important is the hygienic disposal of porcine excreta, and its separation from human food. Personal and domestic cleanliness, encouraged by adequate water supplies and strenuous hygiene education, are essential. Where cultural and farming norms cause the cohabitation of people and pigs, as in some parts of Papua New Guinea, the control of balantidiasis may be impossible.

## Occurrence and Survival in the Environment

There is no information on *Bal. coli* cysts in the environment. It may be assumed that in most moist environments their survival is dependent on time and temperature and is similar to that of *Ent. histolytica* cysts (see figure 20-2). They are rapidly killed by desiccation.

## Inactivation by Sewage Treatment Processes

No specific information has been reported. The cysts of *Bal. coli* are appreciably larger than those of *Ent. histolytica* and are likely to settle more rapidly (no information has been found on density). Otherwise it may be assumed that *Bal. coli* cysts in sewage treatment respond like *Ent. histolytica* cysts (chapter 20).

## Inactivation by Night Soil and Sludge Treatment Processes

No specific information has been reported. It may be assumed that *Bal. coli* cysts in night soil and sludge treatment processes respond in the same way as *Ent. histolytica* cysts (chapter 20).

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# 20

# *Entamoeba histolytica* and Amebiasis

THERE ARE TWO MAIN forms of dysentery. Bacillary dysentery caused by infection with *Shigella* bacteria (chapter 16) and amebic dysentery caused by infection with the protozoon *Entamoeba histolytica*. Amebic dysentery is the subject of this chapter.

## Description of Pathogen and Disease

Amebiasis may describe an infection by any of the amebae—some of which are parasites and live in the gut and some of which are free living and occasionally infect man. In this book, however, amebiasis describes only the infection of man by *Ent. histolytica*.

### *Identification*

*Ent. histolytica* is primarily a parasite of the large intestine. Symptoms, when present, consist of diarrhea, sometimes bloody, and mild pyrexia, with or without abdominal pain. Trophozoites (vegetative forms) of an invasive *Ent. histolytica* erode the epithelial lining of the colon and colonize the submucosal tissues, forming ulcers. Migration of amebae from ulcers may take place via the hepatic portal vein to the liver and other organs, where an amebic abscess may develop. The typical hepatic abscess is in the right lobe of the liver, resulting in pain and swelling in that area. The abscess may burst into the pleural cavity and lung. Cutaneous amebiasis may develop around the anus or an abscess fistula. In general, in severe cases of amebiasis, pyrexia, sweating, a raised erythrocyte sedimentation rate, and slightly raised neutrophil white cell count occur. If there is hepatic involvement, liver function tests may show abnormalities and obstructive jaundice may develop. Diagnosis of intestinal amebiasis is primarily by direct demonstration of hematophagous trophozoites in the diarrheic stool, or of the typical 1–4 nucleate cysts in the formed stool. In cases of extraintestinal amebiasis the intestinal infection may have been lost and serological tests are used.

The degree of morbidity resulting from infections with *Ent. histolytica* is hard to assess, but seropositivity, taken as an index of active invasion, is found in 40 percent or more of asymptomatic carriers in endemic areas. The incidence of liver abscess is related to the length of time an intestinal infection has been present; frequent infection extends the period and makes abscess development more likely. The ingestion of dietary hepatotoxins, alcohol, nutritional deficiencies, and the presence of other parasites may all play a part in the development of active invasion by a potentially invasive amebic strain. Reports of case mortality vary from 0.02 to 6 percent in different countries (Elsdon-Dew 1968). Recent evidence of strain variation based on isoenzyme typing suggests marked differences in pathogenicity, and many of the previously intractable questions of the epidemiology of serious amebic disease can now be reopened.

### *Occurrence*

Amebiasis is found associated with insanitary conditions in all parts of the world. World prevalence has been estimated at 10 percent, and, although it is unusual for it to exceed 30 percent, rates of up to 80 percent or more have been reported in some communities. (Mistaken identification of *Ent. hartmanni* as *Ent. histolytica* has led in some cases to falsely high prevalence values.)

### *Infectious agent*

*Ent. histolytica* is the dysentery ameba of man. The trophozoite is about 20–25 micrometers in diameter, generally elongated, and lives in the lumen of the large intestine (commensal phase) or in the gut wall (invasive phase) (fig. 20-1). It is generally agreed that some strains of *Ent. histolytica* are more virulent than others,

Figure 20-1. *A trophozoite of* Entamoeba histolytica *under scanning electronmicroscopy.* The organism is adhered to a monolayer of human intestinal epithelial cells. Scale bar = 10 micrometers. (Photo: D. Mirelman, Department of Biophysics, Weizmann Institute of Science, Rehovot, Israel)

and also that by "adaptation" (selection) it is possible for virulence to increase or decrease. The cystic resistant stage of *Ent. histolytica* is produced in the lower parts of the large intestine. The cyst is spherical and ranges from 10–15 micrometers in diameter. The ameba *Ent. hartmanni*, which used to be called the "small race" of *Ent. histolytica*, has trophozoites much smaller than those of *Ent. histolytica* and cysts less than 10 micrometers in diameter. It is now recognized that *Ent. hartmanni* is not pathogenic. *Ent. coli*, an ameba closely related to *Ent. histolytica*, is not pathogenic and has cysts ranging in diameter from 14–20 micrometers. This organism has been used in some epidemiologic studies as an alternative to *Ent. histolytica*. *Ent. moshkovskii* is a free-living organism, found in sewage, whose trophozoites and cysts are morphologically identical to those of *Ent. histolytica*. In culture it can be distinguished because it is capable of growing at temperatures from 25 to 37°C. *Ent. histolytica* will not grow at 25°C.

### *Reservoirs*

The reservoir of *Ent. histolytica* is man, although the organism is harbored by primates and there are instances where transmission from primates to man may have occurred. *Ent. histolytica* is also found in dogs and cats and has been transmitted experimentally to many mammalian species.

### *Transmission*

An asymptomatic infected individual is estimated to produce $1.5 \times 10^7$ cysts per day in the stool. A variable proportion of these are mature quadrinucleate cysts, and these are apparently the only ones capable of further development in a new host. Little further maturation of cysts takes place after they have left the body. The trophozoite of *Ent. histolytica* is not of importance in the transmission of the disease, since it dies rapidly on exposure to air and cannot survive passage through the normal stomach. The cystic stage is produced in the intervals between active bouts of dysentery, and it is the convalescent or asymptomatic carrier, producing cysts, who is usually responsible for transmission. When viable cysts are ingested, in water, on food, or directly from fecally-contaminated hands, they hatch in the intestine and produce an infection that may or may not develop invasive characteristics and give rise to symptoms.

Infections of *Ent. coli* in man have been produced after the ingestion of a single cyst, but the median infective dose for this organism appears to be between 10 and 100 cysts (Rendtorff 1954; Rendtorff and Holt 1954*b*). In experimental *Ent. histolytica* infections in man, infections were consistently produced by inocula of 2,000–4,000 cysts.

### *Prepatent and incubation periods*

The median prepatent period in experimental infections of man with *Ent. histolytica* is 5 days. For *Ent. coli* the prepatent period ranged from 6–22 days (mean 10 days) in one study (Rendtorff 1954) and 4–14 days (mean 8 days) in another (Rendtorff and Holt 1954*b*).

The median incubation period in the 1933 Chicago outbreak was 21.4 days. Other reports indicate that the incubation period is 2–6 weeks. In extraintestinal amebiasis the incubation period may be years. Development of amebic abscess is thought to follow the action of some precipitating factor, possibly liver damage due to hepatotoxins or alcohol.

### *Period of communicability*

As long as a chronic infection is present in the gut, the cysts continue to be detectable in the stool. The median duration of untreated intestinal infections in man is about 2 years.

*Resistance*

Susceptibility to infection with *Ent. histolytica* appears to be general, although there may be cultural and racial factors affecting morbidity. Humoral antibodies are produced in response to tissue invasion by amebae. These may be detected by indirect hemagglutination, fluorescent antibody, or immunoprecipitin techniques. Various aspects of cell-mediated immunity have also been demonstrated. The steady loss of intestinal infections, with a median duration of 2 years, suggests that some of the acquired immune response is protective. Information on the protective effect of cured extraintestinal infections is lacking.

*Epidemiology*

Amebiasis occurs throughout the world and is more prevalent in poor communities with inadequate sanitation. There is no simple correlation, however, between levels of sanitation or economic development and the prevalence of amebiasis. Considerable unexplained variations in prevalence exist, even within a small geographical area.

As with other common enteric parasites, the pattern of infection is typically endemic. A poor community may have a substantial proportion of asymptomatic carriers continuously contaminating the environment with *Ent. histolytica* cysts. Over 80 percent of infected persons may be asymptomatic. Recorded prevalences of *Ent. histolytica* cyst excretion in various communities include 3–47 percent in India, 11 percent in Lagos (Nigeria), 7 percent in Bangkok (Thailand), 50 percent in Medellín (Colombia), and 72 percent in San Jose (Costa Rica) (WHO Scientific Working Group 1980).

Prevalences of *Ent. histolytica* cyst excretion among healthy children 1–5 years old in Guatemala were 18 percent of rural children, 6 percent of "low social status" urban children, and 1 percent of "high social status" urban children (Pierce and others 1962). A longitudinal study of forty-five children from birth to 3 years of age in a Guatemalan village showed that 82 percent had had one or more *Ent. histolytica* infections before their third birthday (Mata and others 1977). A survey in Egypt showed a 16 percent prevalence of cyst excretion among students (15–20 years old) in the Nile Delta and 11 percent among similar students from Upper Egypt (Arafa and others 1978). *Ent. histolytica* cyst excretion rates among healthy preschool children (0–6 years) were 4 percent in Sri Lanka, 15 percent in Iran, 6 percent in Bangladesh, and 11 percent in Venezuela (van Zijl 1966).

A major study on the epidemiology of amebiasis was reported from the Gambia (Bray and Harris 1977). Twenty-six villages throughout the country were visited in the dry season, and single stool samples were collected from fifty persons in each village. In all ages and villages, infection rates were 36 percent among both sexes, 26 percent among males, and 45 percent among females. A longitudinal survey over 2.5 years in one district showed prevalences falling to around 15 percent at the end of each dry season (April–May) and at the end of each wet season (October). Peak prevalences (30–>50 percent) occurred early in the dry season (January) and at the start of the rains (June–August). Of individuals followed throughout the 2.5-year survey, 98 percent passed cysts on one or more occasions. Combining both surveys, prevalence rates by age rose steadily from 2 percent in 0–1 year olds to 35 percent in those over 40 years. Many samples of water, hand washings, fingernail clippings, soiled clothing, houseflies, lettuce, and soil were examined, but only one sample of well water yielded *Ent. histolytica*. The authors concluded that "the lack of success in our attempts to elucidate the transmission pathway was remarkable." They found some evidence of clustering of infection by compound and suggested that defecation by small children around the houses could be the major mode of transmission within the compound. As mothers, and older female siblings, are responsible for the care of small children, this might explain the higher infection rates among females.

Endemic amebiasis is found in temperate developed countries as well as tropical developing countries. The carrier rate in the UK is 2–5 percent, with an estimated 300 hospitalized cases and 3 deaths per year (WHO Scientific Working Group 1980). Many of the clinical cases of amebiasis seen in developed countries are associated with infection while traveling abroad. The *Ent. histolytica* carriage rate in the USA is estimated as 3–4 percent overall, but may be closer to 40 percent among adult, male homosexuals (Jones 1979; Schmerin, Gelston and Jones 1977). Within developed countries, prevalences of *Ent. histolytica* infection are higher among the lower socioeconomic groups and among disadvantaged ethnic groups—for instance Indians and Eskimos in North America (see, for instance, Melvin and Brooke 1962; Sole and Croll 1980). Amebiasis is also especially common in mental hospitals (Jeffery 1960; Sexton and others 1974).

In addition to the endemic picture of amebiasis described above, outbreaks also occur. The best documented of these are in developed countries, for instance in Chicago (USA) in 1933 and Indiana (USA) in 1950, and many of these outbreaks have been

waterborne (Brooke and others 1955; LeMaistre and others 1956; Morton, Stamm and Seidelin 1952). Supposed waterborne outbreaks have occurred among persons using chlorinated water supplies, and it is suggested that the chlorine levels were able to destroy the fecal bacteria but not all the amebic cysts. Outbreaks in poor communities having endemic amebiasis are unlikely.

The role of water contaminated by sewage has been clearly established in some outbreaks of amebiasis. The major transmission routes in endemic areas remain uncertain, however. Direct fecal-oral transmission from person to person under conditions of poverty, overcrowding, and inadequate water supply and sanitation is the most likely mechanism. Several studies have pointed to family clustering of infection and intrafamilial transmission; for instance, Bray and Harris (1977) in The Gambia, Engbaek and Larsen (1979) in Denmark, Mathur and Kaur (1972*a*) in India, and Nnochiri (1965) in Nigeria. This last study, in Lagos, found that 96 percent of healthy mothers of sick children with amebiasis had *Ent. histolytica* infections. All the children and some of the mothers were treated. Six months later the prevalences of amebiasis among the treated children were 14 percent of those with treated mothers and 40 percent of those with untreated mothers. Person-to-person transmission has also been strongly implicated in the USA by studies on mental institutions (Jeffery 1960; Sexton and others 1974), on a village in Arkansas (Spencer and others 1976), and on an extended Spanish-American family in Texas (Spencer and others 1977).

The contamination of food, especially salad vegetables, is probably of some importance in transmission to higher socioeconomic groups in urban areas. This type of transmission may also be responsible for a considerable proportion of infection among tourists and travelers. Foodborne transmission can be caused both by the contamination of crops by the use of night soil as a fertilizer and by the contamination of food by infected food handlers (Schoenleber 1940).

Some studies suggest that insect vectors, such as cockroaches and flies, play a role in mechanically transporting cysts in their guts and in contaminating food with their feces and vomitus (Frye and Meleney 1936; Gupta and others 1972; Pipkin 1949; Rendtorff and Holt 1954*a*; Root 1921). However, the epidemiological importance of insects in transmitting amebiasis is uncertain.

Theoretically, transmission is likely to be greatest in the wettest and coolest season, when cysts are most able to survive outside the gut. There is very little information on the seasonality of amebiasis or on the important routes of transmission in poor communities. The relationship and comparative epidemiology of commensal or lumenal amebiasis (the parasite living in the lumen of the colon, with no evidence of invasion or disease) and invasive or pathogenic amebiasis remain unclear, and research is in progress (for instance, Sargeaunt and others 1980). For informative reviews, see Elsdon-Dew (1968, 1978) and Knight (1975).

## Control Measures

Both individual and environmental approaches to amebiasis control may be adopted, although only an environmental approach can have lasting and community-wide benefits.

### *Individual*

There are several effective drugs for the treatment of intestinal amebiasis, most of which are without serious toxic hazards at the recommended dosage. Mass chemotherapy has been applied with success and results in a rapid reduction in prevalence. In the long term, a mass chemotherapy program is unlikely to be effective, unless frequently repeated, without concomitant improvements in sanitary education, excreta disposal, and probably also improved water supplies. There is no vaccine.

Drug prophylaxis is not considered desirable. For individual prophylaxis, the treatment of drinking water by boiling—coupled with the treatment of vegetables with strong vinegar, iodine solutions, or hot water—is recommended. Personal and domestic cleanliness are essential to prevent intrafamilial spread.

### *Environmental*

*Ent. histolytica* excretion has been used as an index of excreta-related or water-related (or both) infection in several studies on the relationships between health and environmental sanitation. Some of these studies are summarized in table 20-1 and more details are given in table 2-1. Studies in India and the USA—Arkansas (two studies), Georgia, North Carolina, Tennessee and Texas—suggested that excreta disposal facilities were related to amebiasis prevalence, whereas studies in Costa Rica and Egypt suggested they were not. Studies in Costa Rica, Japan, Arkansas, and North Carolina suggested that water supplies were associated with amebiasis prevalence, whereas studies in Denmark, Egypt, Arkansas, Tennessee, and Texas

Table 20-1. *Some studies on the relationships between* Entamoeba histolytica *infection and environmental sanitation*

| Country | Finding | Source |
|---|---|---|
| Costa Rica | Prevalence of amebiasis was between 6 and 17 percent in 6 areas studied; no relationship between amebiasis prevalence and either rental value of house or sanitation facilities; association of amebiasis with one source of piped water | Moore, de la Cruz and Vargas-Mendez (1965) |
| Denmark | *Ent. coli* infection was associated with lower socioeconomic status but not with water supplies | Engbaek and Larsen (1979) |
| Egypt | Improved water supplies, bored-hole latrines, refuse disposal, and preventive work by visiting nurses failed to reduce the prevalence of protozoal infections (*Ent. histolytica*—57 percent) or the mean number of infections per person (2.3) | Chandler (1954) |
| India | Family contacts of amebiasis patients were surveyed: prevalences of *Ent. histolytica* excretion were 32 percent of those with latrines and 38 percent of those without: the lack of a latrine was associated with a generally poor domestic environment | Mathur and Kaur (1972*b*) |
| Japan | Type of water supply was believed to be an important determinant of *Ent. histolytica* prevalence | Wykoff, Fonseca and Ritchie (1955) and Wykoff and Ritchie (1960) |
| USA (Arizona, Dakota, Montana, New Mexico, and Wisconsin) | Prevalence of *Ent. histolytica* among Indians was 15 percent and was related to crowding | Melvin and Brooke (1962) |
| USA (Arkansas) | Prevalence of protozoal infection was lower (13 percent) among small children living in houses with indoor water and sewerage than among those with well water and no sewerage (37 percent) | Brooke and others (1963) |
| | *Ent. histolytica* prevalence among blacks was related to crowding and lack of indoor toilets, but not to water quality | Spencer and others (1976) |
| USA (California) | The prevalence of *Ent. histolytica* infection among white female mental patients rose from 10 to 39 percent over a 3-year period during which they were transferred to a much more hygienic new building | Jeffery (1960) |
| USA (Georgia) | *Ent. histolytica* infection among patients at a veterans' hospital was associated with having an outside latrine but not with income | Brooke, Donaldson and Brown (1954) |
| USA (North Carolina) | *Ent. histolytica* prevalences among schoolchildren were associated with sanitation facilities, type of water supply, and garbage disposal | Mackie and others (1956) |
| USA (Tennessee) | *Ent. histolytica* prevalences among rural blacks were associated with sanitation, family size, fecal contamination of the home and cleanliness but not with water pollution | Eyles, Jones and Smith (1953) |
| USA (Texas) | *Ent. histolytica* prevalence in a Spanish American extended family was related to lack of an indoor toilet but not to water supply | Spencer and others (1977) |

suggested they were not. Most of the studies failed to control the numerous confounding variables or to disentangle the effects of income, education, water, sanitation, and housing. The situation is therefore confused.

Improvements in excreta disposal and other sanitary facilities are likely to have little short-term effect on prevalence, but over a decade a marked effect should be detectable. Mass chemotherapy combined with improvements in excreta disposal would enable the lowered prevalence due to the former to be maintained. According to the mathematical model of Knight (1975), in a population where prevalence is 50 percent, halving the hypothetical "transmission constant" by improvements in hygiene will ensure the virtual disappearance of amebic infection, but this will take time. This point should be remembered in assessments of the effect of improvements in excreta disposal alone on prevalence of amebiasis.

Untreated night soil should not be used to fertilize vegetables and fruit destined to be eaten raw, and all night soil application should be halted about a week before harvesting. Polluted water should not be used to freshen the vegetables before sale.

The importance of education of the general population, and especially mothers and food handlers,

in the basic principles of hygiene cannot be overestimated. It is also likely that, despite improvements in water supply and sanitation, indiscriminate defecation by small children around houses, and by workers in agriculture, can still lead to an appreciable amount of transmission.

## Occurrence and Survival in the Environment

Despite the high prevalence of excretion of amebic cysts in many communities, there is very little information on their occurrence in the extraintestinal environment. This is partly because they are difficult to detect in water and other environmental samples. As most transmission is probably by direct fecal-oral routes within the home, the presence of cysts in the environment has not attracted much research. Although *Ent. histolytica* trophozoites are also excreted, they very rapidly die, are not responsible for transmission, and thus are of no environmental interest. The sections that follow deal entirely with cysts.

Earlier studies on the survival of cysts used morphology or exclusion of eosin or neutral red as criteria of viability. These approaches, although generally reliable, are now superseded by cultivation as a test of viability.

### *In water and water supplies*

Waterborne outbreaks of amebiasis have been circumstantially documented, but there have been very few isolations of *Ent. histolytica* cysts from domestic water supplies or from natural surface waters. Where human fecal contamination is present, cysts may be expected, but their presence has not been documented.

The survival of *Ent. histolytica* cysts in water is dependent on temperature and not on water quality. Survival times at various temperatures may be conservatively estimated from figure 20-2.

Heating water is the simplest method of destroying cysts. As shown in figure 20-2, temperatures of 60°C for 1 minute, or 55°C for 10 minutes, are effective (see also Chang 1943; Jones and Newton 1950; Rudolfs, Falk and Ragotzkie 1950).

Chlorination of water will destroy cysts, but more slowly than fecal bacteria. Cysts may therefore persist in waters that are judged bacteriologically safe. Chlorination is more cysticidal when chlorine is free (at a concentration of at least 3 milligrams per liter), at lower pH, at warmer temperatures, and with longer contact times (Chang and Fair 1941). Iodine is also effective.

### *In seawater*

*Entamoeba histolytica* cysts are not affected by salt concentrations encountered in seawater, and survival may be expected to be as in fresh water (Dobell 1928; Kheissin and Dmitrieva 1935).

### *In feces and night soil*

Cysts will frequently be present in feces and night soil, as suggested by the high prevalence rates discussed above in the section on epidemiology.

Survival in feces will be similar to that in other moist environments (Chang 1955; Simitch, Petrovitch and Chibalitch 1954), and may be conservatively estimated from figure 20-2. In desiccated feces, as with stools exposed to bright sunshine and warm temperatures, cyst survival will be very much reduced.

### *In sewage*

*Ent. histolytica* cysts may be expected in sewage but have seldom been reported. Raw sewage in Denver (Colorado, USA) contained *Ent. coli* in 100 percent of samples, at an average concentration of 52 cysts per liter. No *Ent. histolytica* cysts were found owing to the low prevalence of amebiasis in Colorado (Wang and Dunlop 1954).

The survival of *Ent. histolytica* cysts in sewage resembles that in water (Chang 1943) and may be estimated from figure 20-2.

### *On surfaces*

Cysts are killed within 10 minutes by desiccation on the surface of the hands (Spector and Buky 1934), but they survive for periods up to 45 minutes in fecal material lodged under the fingernails (Andrews 1934).

### *In soil*

Survival of *Ent. histolytica* in wet soil is as in water and may be estimated from figure 20-2 (Beaver and Deschamps 1949*a*). Survival in dry soil is very much shorter (Rudolfs, Falk and Ragotzkie 1951).

### *On crops*

Cysts of *Ent. histolytica* are extremely sensitive to desiccation, with or without the presence of additional organic matter. Crops growing in the field may become contaminated with *Ent. histolytica* directly, through irrigation with polluted water or night soil, or

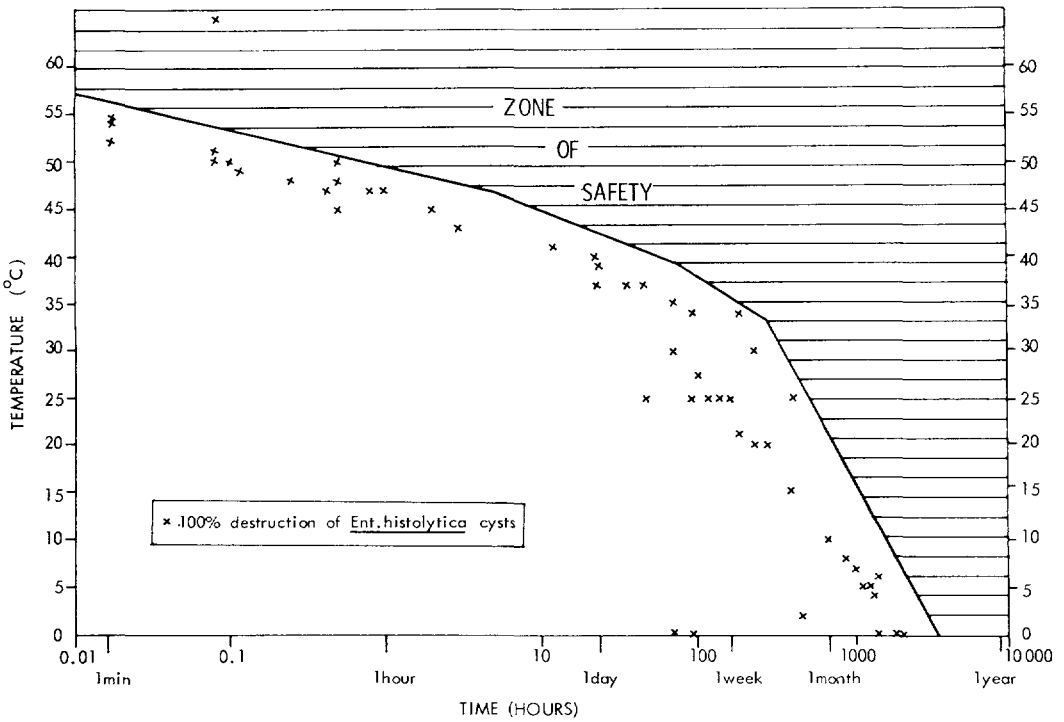


Figure 20-2. *The influence of time and temperature on* Ent. histolytica *cysts.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

indirectly, through contact with the polluted soil containing cysts. Three days of dry weather kill cysts on the surface of vegetables (Rudolfs, Falk and Ragotzkie 1951). In hot, dry climates, survival times may be less than 1 day.

Vegetables may be decontaminated by soaking in warm water (55°C) or in vinegar or salad dressing (containing 5 percent acetic acid) for 30 minutes (Beaver and Deschamps 1949*b*; Chang 1950).

*Summary*

Cysts are a protected resting stage of the parasite and are remarkably unaffected by their chemical surroundings. They can survive a wide range of pH values and osmotic pressures. They will die rapidly if dried or frozen. In the absence of desiccation, freezing, or any specifically cysticidal substance like chlorine or acetic acid, their survival depends on temperature (Chang 1943; Chang and Fair 1941). For this reason the points plotted in figure 20-2 form a smooth curve despite the very variable physicochemical conditions of the experiments. The pattern of points for enteroviruses (figure 9-2), or even a worm egg (figure 23-2), show far more scatter, and this indicates the effect of circumstances other than temperature. In most moist environments *Ent. histolytica* cyst survival may be estimated directly from figure 20-2.

*Ent. coli* is considerably more resistant to desiccation than *Ent. histolytica* (see Reardon, Verder and Rees 1952; Spector and Buky 1934), and this may partly explain the considerably higher prevalence of the former in most communities.

## Inactivation by Sewage Treatment Processes

Information on *Ent. histolytica* in sewage treatment is limited. Sewage treatment processes do not involve freezing or desiccation, and in a primarily domestic and unchlorinated sewage *Ent. histolytica* cysts will not experience any strongly cysticidal chemicals. Therefore, survival will be a function of time and temperature and may be estimated from figure 20-2 for any given process.

*By sedimentation*

Primary and secondary sedimentation remove only a small proportion of cysts because cysts are small and not dense (average diameter 12 micrometers; specific

gravity 1.06) and so have a very low settling velocity (<0.1 meters per hour) (Chang 1945).

Primary sedimentation in Denver (Colorado, USA) halved the concentration of *Ent. coli* cysts from 52 per liter to 27 per liter. In India, 2 hours of sedimentation removed 64 percent of *Ent. histolytica* cyst, whereas 1.5 hours of sedimentation removed only 27 percent (Panicker and Krishnamoorthi 1978).

*By trickling filter*

In Haifa (Israel) raw sewage contained 4 *Ent. histolytica* cysts and 28 *Ent. coli* cysts per liter (Kott and Kott 1967). After primary sedimentation and trickling filter treatment, these concentrations were reduced to 3 (25 percent reduction) and 16 (43 percent reduction) per liter, respectively. Two complete trickling filter plants (including secondary sedimentation) in India removed 74 and 91 percent of *Ent. histolytica* cysts (Panicker and Krishnamoorthi 1978).

*By activated sludge*

The activated sludge process itself will have little effect on *Ent. histolytica* cysts. The environment is wet and not hostile, temperatures are ambient, and detention times are short (6–12 hours). Cysts may become entrapped in the flocs, in which case they will be removed during secondary sedimentation. A complete activated sludge plant (including secondary sedimentation) in India removed 83 percent of *Ent. histolytica* cysts (Panicker and Krishnamoorthi 1978).

*By oxidation ditch*

A pilot-scale oxidation ditch (including sedimentation) in India removed 91 percent of *Ent. histolytica* cysts (Panicker and Krishnamoorthi 1978).

*By waste stabilization ponds*

Well-operated waste stabilization ponds with sufficient cells (at least three) and retention time (at least 20 days) produce an effluent completely free of *Ent. histolytica* cysts. A single pond in India, with 7 days retention, achieved 100 percent reduction (Arceivala and others 1970). Three ponds in India with unknown characteristics achieved 87, 94, and 100 percent reductions (Panicker and Krishnamoorthi 1978). Ponds with 20 days retention in Israel completely eliminated *Ent. histolytica* cysts (Wachs 1961).

*By aerated lagoons*

Pilot-scale aerated lagoon treatment (without secondary sedimentation) in India removed 84 percent of *Ent. histolytica* cysts (Panicker and Krishnamoorthi 1978).

*By tertiary treatment*

Certain tertiary processes can eliminate *Ent. histolytica* cysts from secondary effluents.

FILTRATION. Filtration through sand or suitable soil can remove all cysts (Cram 1943; Gordon 1941; Spector, Bayliss and Gullans 1934).

DISINFECTION. Chlorination of secondary effluents was found to eliminate cysts in Haifa (Israel: Kott and Kott 1967), but not in Denver (USA; Wang and Dunlop 1954) or Moscow (USSR; Gordon 1941). The results obtained clearly depend on the chlorine dose applied, the quality of the effluent, the contact time, and the temperature. It may be assumed that the effectiveness of chlorine on cysts in effluents is considerably lower than on enteroviruses in effluents (chapter 9).

LAND TREATMENT. Land treatment of secondary effluents should theoretically be able to remove all cysts.

## Inactivation by Night Soil and Sludge Treatment Processes

Night soil and sludge treatment processes do not involve freezing, rarely involve desiccation, and do not produce environments that are especially or particularly hostile to *Ent. histolytica* cysts. Therefore cyst destruction is simply a function of time and temperature may be conservatively predicted for any process by reference to figure 20-2.

Most mesophilic and all thermophilic sludge and night soil treatment processes will eliminate *Ent. histolytica* cysts (Cram 1943; Kawata, Cramer and Burge 1977). Aerobic thermophilic composting is the process of choice and will eliminate *Ent. histolytica* cysts in 1 hour at 50°C. The extensive studies of Scott (1952) in China demonstrated the effectiveness of aerobic composting of feces, manure, and vegetable matter in eliminating protozoal cysts.

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# 21

# *Giardia* and Giardiasis

THE PARASITE *Giardia*,[1] and the infections and diseases it causes, attracted little interest for the first 110 years after its description by Lambl in Prague in 1859. It was widely believed that *Giardia* was a commensal parasite of doubtful pathogenicity. It is now recognized that *Giardia lamblia* frequently causes a mild, self-limiting disease in man and is, more rarely, responsible for serious illness. It is now the most commonly isolated intestinal pathogenic protozoon worldwide; in countries such as the USA and the UK where worm infections are rare, it is the most commonly isolated of all intestinal pathogenic parasites. In addition, *G. lamblia* has been found to be responsible for several recent waterborne diarrhea outbreaks in the USA and USSR, and a combination of these circumstances has created a wave of interest and research in this previously neglected parasite. Recent reviews of the subject include Jakubowski and Hoff (1979), Knight (1978), Meyer and Jarroll (1980), Meyer and Radulescu (1980), Raizman (1976), WHO Scientific Working Group (1980), and Wolfe (1978, 1979*a*, 1979*b*).

## Description of Pathogen and Disease

Although the clinical picture of giardiasis is well described, several aspects of its pathology, immunology, and epidemiology remain uncertain, and some of the information in this chapter is therefore preliminary and tentative.

### *Identification*

Giardiasis is an infection of the small intestine of man by the flagellate protozoon *Giardia lamblia*. The bile duct and gall bladder may also be infected. Symptoms may be absent, but when present may include frequent diarrhea with greasy, foul-smelling stools, usually without blood. There may be fatigue, abdominal cramps, flatulence, anorexia, and in some cases fever and vomiting. During the infection, damage to the intestinal epithelium, detectable histologically, may take place; bacterial colonization of the small intestine may be a predisposing condition for this damage or may be consequent upon it. The damage leads to malabsorption of carbohydrates, fats, fat-soluble vitamins, and vitamin $B_{12}$. Malabsorption and bile duct inflammation are the most serious complications of giardiasis, since the malnutrition consequent on malabsorption increases susceptibility to other diseases. The disease is diagnosed by identifying the cysts or trophozoites of *G. lamblia* in the stools, or by recognition of the trophozoites in duodenal or jejunal aspirates or biopsies. *G. lamblia* is morphologically indistinguishable from other *Giardia* species common in many mammals. In cases of malabsorption, serology can aid the diagnosis.

### *Occurrence*

The disease is cosmopolitan, associated with poor sanitation and inadequate protection of drinking water sources. Prevalence of *Giardia* infection worldwide is estimated to be about 7 percent. It is about 3 times more common in children than in adults. Local prevalence in children may exceed 50 percent. In Europe and the USA, there is a considerable variation from place to place in the likelihood of contracting the disease, and this appears to be related to the safety of the drinking water. Intrafamilial infection is well recognized.

### *Infectious agent*

The taxonomy and nomenclature of *Giardia* are in a confused and fluid state. Two genus names are used

1. See the subsection "Infectious Agent," below, for a note on taxonomic nomenclature.

interchangeably: *Giardia* (giardiasis) is most commonly used in North America and Western Europe, whereas *Lamblia* (lambliasis) is favored in Eastern Europe and the USSR. Resolution of this issue is simply a question of agreement about the historical precedence of the rival names—the nature of the organism being described is not in doubt. It is probable that *Giardia* will become adopted worldwide, and *Giardia* is used in this book.

The definition of species within the genus *Giardia* is far more complex. In the first half of this century it was believed that *Giardia* species were highly host specific, and therefore specific names were allocated on this basis. It is now known, that some *Giardia* species infect several different animals. The morphology of the trophozoites provides the other main avenue for species definition. Three primary species have been suggested on the grounds of trophozoite morphology: *G. agilis* in frogs and tadpoles, *G. muris* in rodents and birds, and *G. duodenalis* in mammals including man. The matter remains unresolved, and research is in progress on improved species definition.

The species or subspecies that infects man is called *G. lamblia* in this book, although it is also referred to in the literature as *G. intestinalis*, *G. enterica*, or, in Eastern Europe, *Lamblia intestinalis*.

*G. lamblia* is a flagellated protozoon. The trophozoite, found in the small intestine or in diarrheic stools, is pear or kite shaped, 9–21 micrometers long by 6–12 micrometers broad, with an anterior sucking disc on the flattened ventral surface (figure 21-1). There are four pairs of flagella, and the organism is binucleate. The cysts are ovoid, 14–16 micrometers long and 6–12 micrometers broad, and are quadrinucleate.

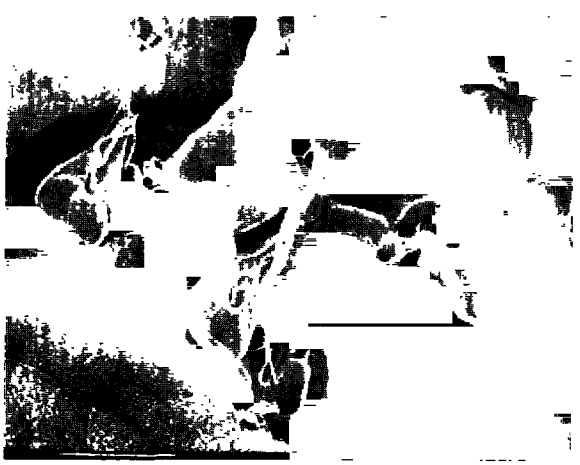

Figure 21-1. *Trophozoites of* Giardia lamblia, *on and over the surface of the small intestine of a patient with giardiasis, under scanning electronmicroscopy.* Scale bar = 10 micrometers. (Photo: R. L. Owen, Cell Biology Section, Veterans Administration Medical Center, San Francisco, USA. Reproduced by permission of the Royal Society of Tropical Medicine and Hygiene)

### *Reservoirs*

The reservoir of *G. lamblia* is man, but there is some evidence that man may acquire infections from other animals. Many mammalian species harbor their own *Giardia* species whose relationship to *G. lamblia* has not yet been properly elucidated. Beavers have been incriminated as a reservoir of infection for hikers drinking stream water that is contaminated by wild animal feces. *G. lamblia* from man has been transmitted to the rat, gerbil, guinea pig, beaver, dog, cat, racoon, bighorn, mouflon, and pronghorn (Davies and Hibler 1979; Meyer and Radulescu 1980). *Giardia* cysts from beavers and deer have caused infection in human volunteers (Davies and Hibler 1979).

### *Transmission*

Up to 900 million cysts may be passed in the stools during 1 day. The cystic infective stage is quadrinucleate and relatively thick walled, and earlier thin-walled "precysts" and the trophozoites rapidly die and are not important in transmission. The median infective dose for man is between 25 and 100 cysts (Rendtorff 1954, 1979; Rendtorff and Holt 1954*b*). On being ingested, the cyst resists stomach acid and hatches in the small intestine, where the two trophozoites that emerge multiply by longitudinal binary fission in the crypts of the duodenum, jejunum, and ileum and attach themselves to the mucosa by means of the sucking disc. Infection of the lining of the bile duct and gall bladder has been reported, although there is no evidence for invasion of other sites. The trophozoites transform into cysts in the ileum, and the cysts are passed in the stool.

Unlike the acute amebic dysentery case, the patient with marked symptoms of intestinal giardiasis is a potent source of infective cysts. This may be a reason why outbreaks of giardiasis are more common than those of amebiasis.

Transmission of *Giardia* is by fecal contamination of hands, food, and water supplies. Water supply contamination has been definitely incriminated in several outbreaks. Although houseflies and other insects have not been shown to be efficient distributors of cysts, it is likely that flyborne transmission of the

organism may play a part in areas of high prevalence. Fecal contamination of the hands probably plays an even more important part in the transmission of *Giardia* than of *Ent. histolytica.* Although transmission of *Ent. histolytica* occurs only sporadically in the UK, *Giardia* is regularly transmitted, and in this country the water supply can probably be ruled out as a possible source.

*Prepatent and incubation periods*

In man the prepatent period has been recorded as 6–36 days, averaging 9 days in one study (Rendtorff 1954), 13 days in another (Rendtorff and Holt 1954*b*), and 14 days (median) in a third (Jokipii and Jokipii 1977). The incubation period in natural outbreaks is generally between 1 and 3 weeks, and a study of thirty-five cases in Finland recorded a range of 3–42 days and a median of 8 days (Jokipii and Jokipii 1977).

*Period of communicability*

Infections in adults, demonstrable by the presence of cysts in the feces (and therefore communicable), have been experimentally observed to last up to 41 days. In children the course of the infection may be much longer and has been reported to last for several years (the possibility of self-reinfection cannot be excluded). In a report from Delhi, two-thirds of adults and children naturally infected had lost their infections within 3 months.

*Resistance*

The higher prevalence of the disease in children and the relatively short duration of an infection indicate that a protective immune response, possibly age-related, may be involved. Antibody to *Giardia* is detectable in the serum of patients with the intestinal malabsorption syndrome and a *Giardia* infection, but there is no evidence of protection in such cases. Resistance following infection is probably not long-lasting, and reinfection is common, even in adults.

*Epidemiology*

Giardiasis occurs throughout the world but is more prevalent in poor communities with inadequate sanitation. There is no simple correlation, however, between levels of sanitation or economic development and the prevalence of giardiasis. Considerable unexplained variations in prevalence exist, even within a small geographical area.

The pattern of infection is typically endemic. A poor community may have a substantial proportion of asymptomatic carriers continuously contaminating the environment with *Giardia* cysts, although the duration of carriage is typically shorter than for amebiasis (chapter 20). Recorded prevalences in various communities range from 1 to over 20 percent. Children between 1 and 5 years old have the highest infection rates.

Prevalences of *Giardia* excretion among healthy children 1–5 years old in Guatemala were 26 percent of rural children, 44 percent of poor urban children, and 15 percent of more wealthy urban children (Pierce and others 1962). A longitudinal study of 45 children from birth to 3 years of age in a Guatemalan village showed that 93 percent had had one or more *Giardia* infections before their third birthday (Mata and others 1977).

Studies in the Gambia showed that age-specific prevalences of giardiasis rose from 1 percent in infants (under 1 year), to 24 percent in children 3–4 years old and fell steadily to 6 percent in those over 40 years old (Bray and Harris 1977). Giardiasis was more common than amebiasis in children under 5 years old, but less common in the whole community. In northern Nigeria the giardiasis prevalence rose from 1 percent in 0–3 month old babies to 51 percent in 2–3 year old children and fell to 18 percent in adults (Tomkins 1981).

*Giardia* excretion rates among healthy preschool children (0–6 years) were 4 percent in Sri Lanka, 22 percent in Iran, 14 percent in Bangladesh, and 21 percent in Venezuela. Except in Sri Lanka, giardiasis was more prevalent than amebiasis (van Zijl 1966). Giardiasis prevalences among Laotian refugees in Thailand were 23 percent in the under-5 age group and 7 percent in the total population (Temcharoen and others 1979). Corresponding figures for amebiasis were 3 percent and 2 percent. Mello and others (1978) found *G. lamblia* cysts under the fingernails of 1 out of 148 schoolchildren in Brazil.

Endemic giardiasis is found in temperate developed countries as well as in tropical developing countries. Prevalences are especially high among poor communities and disadvantaged ethnic groups. Prevalences among poor children (6 months to 16 years) in Glasgow (Scotland) were 13 percent of Scots, 10 percent of Asians, 1 percent of Africans, and 1 percent of Chinese (Goel and others 1977). Sole and Croll (1980) recorded a giardiasis prevalence of 5 percent among one racial group in Labrador (Canada), with no infections occurring in adults over 20 years old. Over a 1-year period 24 percent of Cherokee schoolchildren in the USA had giardiasis, and 14 percent had *Ent. histolytica* infection (Healy

1979). A survey in Tasmania (Australia) found that *G. lamblia* was the most commonly identified intestinal parasite of any kind (Goldsmid 1981). Giardiasis in developed countries is especially common in mental institutions (Jeffery 1960), in nurseries (Black and others 1977; Brown 1948), and among male homosexuals (Mildvan, Gelb and William 1977). Giardiasis is also associated with recent foreign travel to developing countries or to Leningrad (USSR).

In addition to the endemic picture of giardiasis described above, outbreaks also occur. The best documented of these are in developed countries, especially the USA and USSR, and most are believed to have been waterborne. In the USA, between 1965 and 1977 there were twenty-three waterborne outbreaks of giardiasis reported involving 7,009 cases (Craun 1979*a*; 1979*b*). *Giardia* cysts were isolated from the water in five of these outbreaks. The outbreaks occurred mainly in the mountainous areas of the Rocky Mountains, in the Northwest, and in New England. The incriminated waters were mostly surface waters either untreated (six outbreaks) or treated only by chlorination (ten outbreaks). *Giardia*-positive beavers were implicated as the source of infection in one outbreak in Washington State. Colorado has been the most affected state and has now made the filtration and chlorination of all surface water supplies mandatory. Endemic giardiasis in Colorado has been associated with the drinking of mountain stream water by hikers (Wright and others 1977). Waterborne giardiasis in the USA is reviewed at length in Jakubowski and Hoff (1979).

There is evidence for (Gupta and others 1972; Root 1921) and against (Rendtorff and Holt 1954*a*) the transmission of *Giardia* cysts from feces to food by flies and cockroaches. It a clear theoretical possibility. but of unknown epidemiological importance.

As with amebiasis (chapter 20), waterborne outbreaks occur, but endemic transmission is much more likely to be by person-to-person transfer especially in conditions of poverty, overcrowding, and inadequate water supply and sanitation. Person-to-person transmission has been strongly implicated in nurseries, schools, and other children's institutions in Czechoslovakia (Červa 1955), the USA (Black and others 1977), and elsewhere. Person-to-person transmission, and family clustering of infection, have also been suggested in community studies (for instance, Eyles, Jones and Smith 1953).

## Control Measures

The gaps in knowledge about possible non-human reservoirs of *G. lamblia*, the survival of *Giardia* cysts in the environment, and the epidemiology of giardiasis in developing countries prohibit any definitive statements to be made on control.

### *Individual*

A number of drugs are effective in treatment (for instance, metronidazole) but are not appropriate for mass chemotherapy or prophylaxis. There is no vaccine.

Individual protection is achieved by cleanliness and care in choice and preparation of drinking water and food. Suspected water should be boiled, and suspect fruit and vegetables should be washed and treated with warm water (55°C) or vinegar for 30 minutes.

### *Environmental*

There is little direct evidence on the relative effectiveness of various environmental control approaches. In rural areas of Tennessee (USA), giardiasis prevalence was related to fecal pollution of the home, household cleanliness, personal cleanliness, and family size but not to water supply or sanitation facilities (Eyles, Jones and Smith 1953). Moving mental patients in California (USA) from old and unhygienic premises to a new and well-equipped building did not reduce *Giardia* transmission (Jeffery 1960). Improved water supplies, bored-hole latrines, refuse disposal, and preventive work by visiting nurses failed to reduce the prevalence of giardiasis in Egypt (Chandler 1954).

The comments on environmental control of amebiasis (chapter 20) apply to giardiasis. Improved personal and domestic cleanliness are probably crucial, and improved water supply and sanitation facilities may also be important.

## Occurrence and Survival in the Environment

Tests for *G. lamblia* cysts in water and other samples are currently very inadequate. Cysts may be missed altogether at concentrations below 4,000 cysts per liter of water (a high concentration), and the cyst count may underestimate the true count by as much as 99 percent. When a cyst has been detected microscopically, there is no way of proving it to be a species capable of infecting man (other than by human volunteer studies), nor of showing it to be infective (other than by feeding it to laboratory animals). Even establishing viability (by eosin exclusion or excystation *in vitro*) is complex and controversial. There will be little progress in the

environmental study of *Giardia* until laboratory techniques are greatly improved.

The recent documentation of waterborne outbreaks of giardiasis in developed countries has stimulated an explosion of research interest in this disease and engineering approaches to control. Most of this interest is focused on *Giardia* cyst removal by water treatment processes and is therefore not relevant to this book. The state of the art, as in 1978, is comprehensively described by Jakubowski and Hoff (1979).

Pending more research, the best estimate of *Giardia* cyst survival in the environment is that it is similar to *Ent. histolytica* survival and may therefore be estimated from figure 20-2.

### *In water and water supplies*

The inadequacy of laboratory techniques for isolating *Giardia* cysts from water has prevented progress in the study of *Giardia* in the environment. In five of the waterborne outbreaks of giardiasis recorded in the USA during 1965–77, *Giardia* cysts were found in raw water sources or tap water (Craun 1979*a*; Jakubowski and Ericksen 1979). Cysts were located with extreme difficulty, and, if the waterborne assumptions were correct, recorded cysts must have grossly underestimated the actual occurrence of cysts in the waters. In the case of the outbreak at Rome (New York, USA) in 1974, $1.1 \times 10^6$ liters of raw water were filtered over 10 days, and one cyst was identified microscopically in the residue. Two of ten samples of the residue induced giardiasis in beagle puppies that ingested them (Jakubowski and Ericksen 1979).

Bingham, Jarroll and Meyer (1979) studied *Giardia* cyst survival in unchlorinated tap water (pH 6.8) at various temperatures and evaluated cyst viability both by eosin-exclusion and by the ability to excyst *in vitro*. Judged by eosin staining (indicating dead cysts), 100 percent destruction took place in 24 days at 37°C, in 31 days at 21°C, and in over 77 days at 8°C. Judged by the failure to excyst, the equivalent times were 6 days, 25 days, and 77 days. Cysts were instantly rendered dead, by the criterion of failure to excyst, in boiling water.

It is widely believed that *Giardia* cysts are resistant to chlorination under conditions often found in water treatment plants. They are particularly resistant at low temperatures, and this fact has been linked to the occurrence of several waterborne outbreaks in the USA and the USSR in the winter months. Current concepts of *Giardia* disinfection come partly from early studies that adopted the very conservative criterion of eosin exclusion as the test of viability (for instance, Červa 1955; Thomas 1952), and partly from analogy with *Ent. histolytica* cysts (chapter 20). Both these approaches are unsatisfactory, and the recent development of *in vitro* excystation tests for viability will permit more informative research on *Giardia* chlorination to be conducted (Hoff 1979). The first reported study of the effect of disinfection on excystation confirmed that both chlorine and iodine compounds have considerably reduced cysticidal properties at 3°C compared with 20°C (Jarroll, Bingham and Meyer 1980).

Filtration experiments have indicated that *Giardia* cysts can be removed by coagulation plus granular medium filtration, or by diatomaceous earth filtration, but only with "conscientious, high quality filter plant operation" (Logsdon, Symons and Hoye 1979).

### *In feces and night soil*

Cyst production by an infected individual is typically $10^5$–$10^7$ per gram of stool but varies greatly. Some individuals excrete only small numbers of cysts intermittently, with 60 percent of stools negative.

### *In sewage*

The probable concentration of *Giardia* cysts in sewage in the USA has been estimated theoretically as $9 \times 10^3$–$2 \times 10^5$ per liter (Jakubowski and Ericksen 1979). These concentrations of cysts have not been confirmed, and the literature contains only the occasional, and mainly qualitative, report of *Giardia* cysts in sewage. Fox and Fitzgerald (1979) reported up to 530 *Giardia* cysts per liter of raw sewage in Chicago (USA). The cysts were more common in domestic than industrial sewage and occured more frequently during April–September.

### *Summary*

There are only a few studies on *Giardia* cyst survival and all except the most recent use overconservative tests for viability. Workers have had to rely on observations of morphological changes, and the fact that eosin or neutral red will stain the contents of cysts which are (probably) dead (see Boeck 1921*a*, 1921*b*; Červa 1955). By analogy with *Ent. histolytica*, we might expect that stained cysts would certainly be dead, whereas some unstained cysts might also be dead.

No definitive statements on *Giardia* cyst survival can be made until a new generation of experiments, using *in vitro* excystation as the measure of viability, have been completed. In the meantime the literature suggests that *Giardia* cysts are somewhat similar to

*Ent. histolytica* cysts. They are rapidly killed by desiccation and freezing. They are resistant to a wide range of pH and osmotic pressure. Their survival in most moist environments is primarily temperature dependent and may be similar to *Ent. histolytica* (figure 20-2). The most recent data, using excystation techniques, on survival in water (6 days at 37°C, 25 days at 21°C and 77 days at 8°C) show a remarkably close agreement with figure 20-2 (Bingham, Jarroll and Meyer 1979).

## Inactivation by Sewage Treatment Processes

Very little is known about the fate of *Giardia* cysts during sewage treatment. The cysts are similar in size to those of *Ent. histolytica* but are reported to be slightly denser (specific gravity 1.11; Rachmanow 1936).

The best assumption at present is that *Giardia* cysts respond to sewage treatment in the same manner as *Ent. histolytica* cysts (chapter 20). Studies on sedimentation, trickling filters, activated sludge, biodiscs, aerated lagoons, oxidation ditches, and waste stabilization ponds in India showed very similar removal rates for the two protozoa (Panicker and Krishnamoorthi 1978). All systems removed 52–93 percent of *Giardia* cysts (except waste stabilization ponds, which removed 100 percent).

## Inactivation by Night Soil and Sludge Treatment Processes

This subject has not been investigated. Night soil and sludge treatment processes do not involve freezing, very rarely involve desiccation, and do not produce environments that are especially or particularly hostile to *Giardia* cysts. Therefore, cyst destruction may be expected to be a function of time and temperature. Pending research, the best assumption is that *Giardia* cysts behave as *Ent. histolytica* cysts, and their fate during any given process can be estimated from figure 20–2.

Most mesophilic and all thermophilic digestion and composting processes are likely to eliminate *Giardia* cysts. In Chicago (USA), despite the presence of *Giardia* cysts in raw sewage, none were found in anaerobically digested sludges (Fox and Fitzgerald 1979). Thermophilic composting may be expected to eliminate *Giardia* cysts with a wide margin of safety. Storage of night soil or sludge at tropical temperatures (>20°C) for 6 weeks or more should also eliminate *Giardia* cysts (figure 20-2).

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# *Section IV.*
# *Excreted Helminths*

# 22

# *Ancylostoma, Necator*, and Ancylostomiasis

ANCYLOSTOMIASIS, or hookworm infection, is not only very common but also produces serious clinical consequences among a proportion of those infected. With the possible exception of schistosomiasis (chapter 32), it is the excreted worm with the greatest worldwide public health importance.

## Description of Pathogen and Disease

Ancylostomiasis is an infection of the small intestine with one of the two species of human hookworms, *Necator americanus* or *Ancylostoma duodenale*. Incidental infections with animal hookworms such as *A. caninum*, *A. ceylanicum* and *A. braziliense* are not considered here, as they are unrelated to the disposal of human excreta. Ancylostomiasis is a comprehensively studied infection, and only a brief summary of information on the worms and the diseases they cause is given below. Several reviews are available, and Banwell and Schad (1978) and Miller (1979) are particularly recommended.

### *Identification*

Ancylostomiasis is frequently symptomless. When it does produce illness and constitutes a public health problem, the most important features are anemia and its resulting weakness, debility and other consequences (Roche and Layrisse 1966). Gastrointestinal pain, transient cutaneous and pulmonary symptoms, and edema may also be experienced. In heavily affected endemic areas, ancylostomiasis produces its most severe clinical effects in older children and in young and middle-age adults, especially in those vulnerable groups subject to physiological iron losses, such as pregnant and lactating women or women suffering from abnormally heavy menstruation. In areas of very intense transmission, however, heavy worm burdens can be built up in early childhood, and in such cases there may be retardation of mental and physical development.

Hookworm is seldom recorded as a direct cause of death. Some grossly anemic individuals die of high-output heart failure. The disease is undoubtedly a common contributory cause of death when other normally nonfatal infections attack a severely anemic and debilitated person.

Numerous symptoms and signs cause suspicion of hookworm infection, but definitive diagnosis depends on finding eggs in fecal samples. Since the eggs of *N. americanus* and *A. duodenale* appear identical on microscopical examination, species recognition requires, in practice, either:

(i) The administration of a vermifuge, followed by collection of feces and microscopic study of the expelled adults, which are morphologically distinct, or
(ii) The cultivation of hookworm eggs to the filariform infective larval stage, when the two species can be differentiated.

### *Occurrence*

Historically, human hookworm infections were probably confined to the eastern hemisphere, *N. americanus* occurring south of 20° north latitude, and *A. duodenale* north of 20° north latitude. In the past 500 years, population migrations, most notably those involving Spanish and Portuguese colonization in the New World and Southern Africa, led to the introduction of *A. duodenale* into these areas, as well as the importation of *N. americanus* to Portugal. At the same time, the slave trade from Africa to North and South America and the Caribbean islands led to the present widespread distribution of *N. americanus* in the western hemisphere.

The present geographical distribution of the hookworm species is given in figures 22-1 and 22-2. It

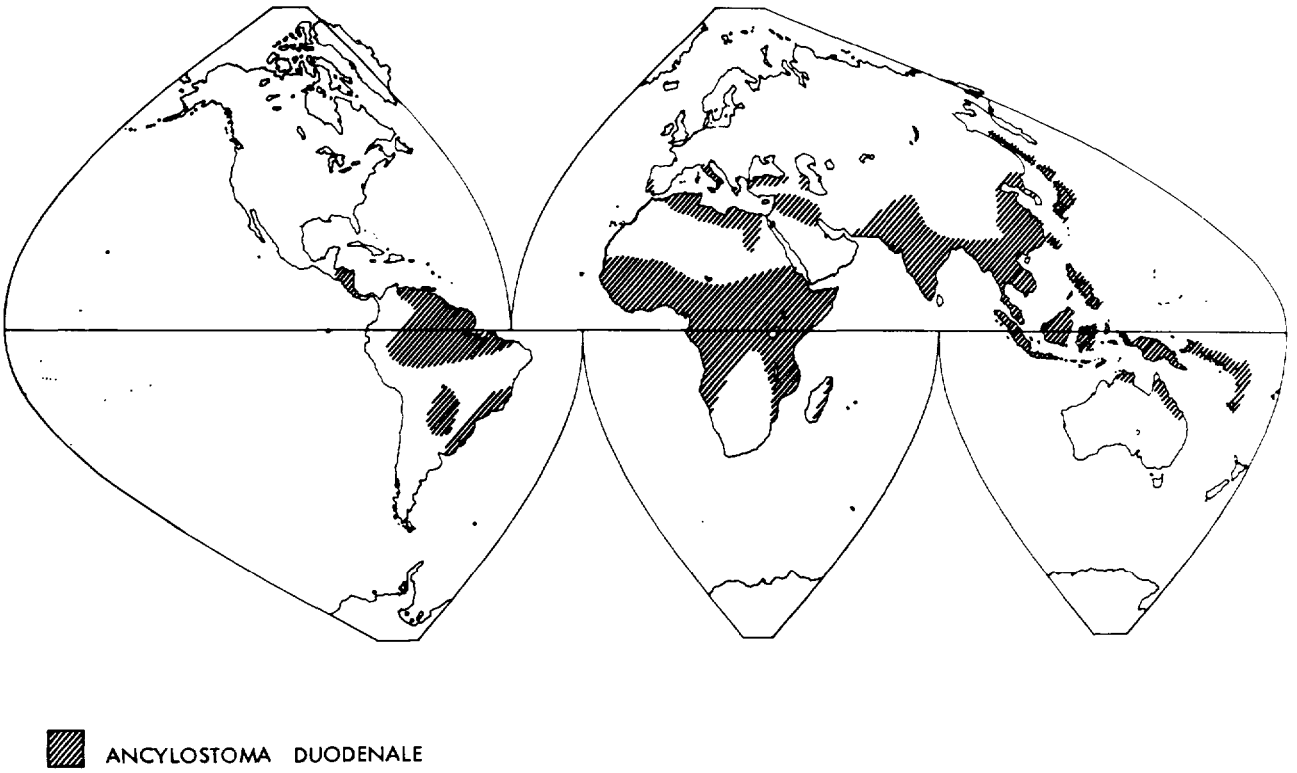


Figure 22-1. *Known geographical distribution of* Ancylostoma duodenale. The infection may occur in areas as yet unrecorded

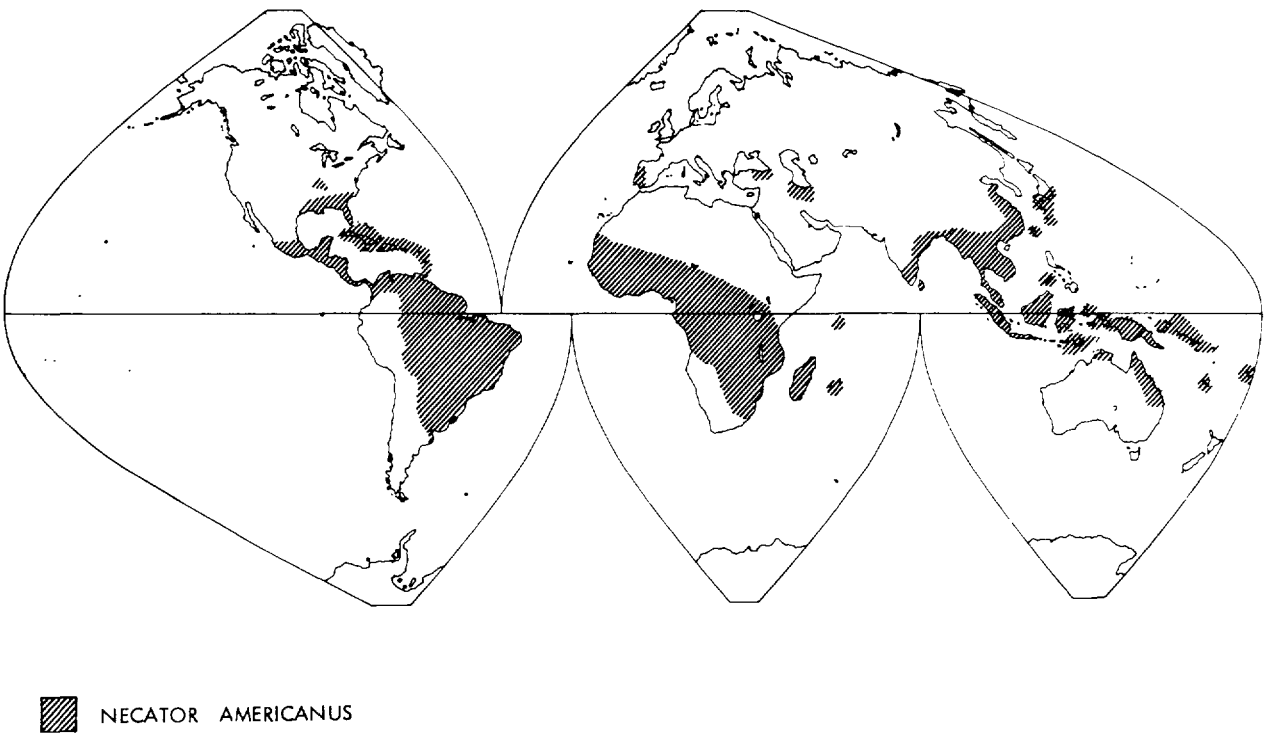


Figure 22-2. *Known geographical distribution of* Necator americanus. The infection may occur in areas as yet unrecorded

must be stressed, however, that many of the studies upon which these maps are based used egg detection techniques only and assumed the species identity of the parasites on the basis of existing knowledge for the locality. It is likely that future studies using adult worm recovery or cultivation of infective larvae will lead to considerable extensions of the geographical range of both worms. Furthermore, maps give no indication of the relative importance of each species in areas where both species are sympatric, nor do they indicate intensity of infection and, hence, clinical importance. It is essential that species prevalence and species intensity be determined for an area before the planning and execution of any control intervention.

*Infectious agents*

The two hookworms that infect man, *A. duodenale* and *N. americanus*, are diecious, sexually dimorphic roundworms belonging to the phylum Nematoda, order Strongylida, superfamily Ancylostomatoidea. The adult worms are small and off-white or rusty in color (figure 22-3). *A. duodenale* is somewhat larger than *N. americanus*, the males being 5–10 millimeters long and the females 10–18 millimeters, depending on the species. The eggs of *A. duodenale* measure 56–60 by 36–40 micrometers, and those of *N. americanus* are 64–76 by 36–40 micrometers in size.

*A. ceylanicum*, a hookworm of dogs, cats, and other animals, can infect man and develop to the adult stage. It has been reported to be of some importance in India, Surinam, West Irian (Indonesia) and elsewhere (Banwell and Schad 1978). *A braziliense* and *A. caninum*, the cat and dog hookworms, rarely develop to the adult stage in man, but their larvae can cause a creeping dermatitis called larva migrans.

*Reservoir*

Man is the reservoir for the human hookworms.

*Transmission*

Thin-shelled, ovoid, unsegmented eggs are discharged by the adult female worms into the lumen of the small intestine. The longer and stouter females of *A. duodenale* produce approximately twice the number of eggs per day ($10^4$ to $2 \times 10^4$) as do those of *N. americanus* ($5 \times 10^3$ to $10^4$). The eggs develop rapidly in the gut and are usually at the four- or eight-cell stage when evacuated in the feces. If feces are deposited in a suitable environment, the eggs hatch in 24–48 hours to give rise to rhabditiform first-stage larvae. Optimum

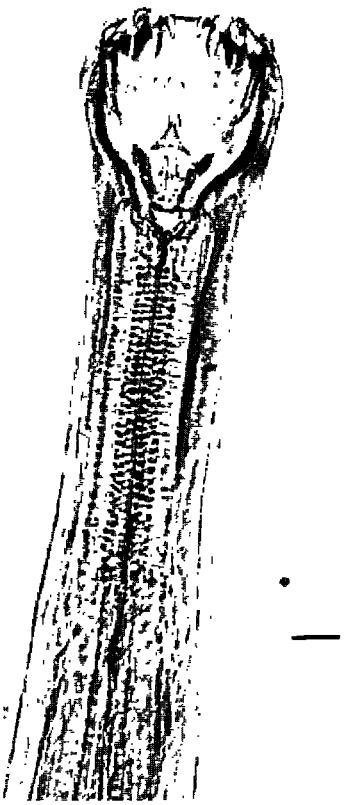

Figure 22-3. *The head (scolex) of an* Ancylostoma *under a light microscope.* Scale bar = 0.1 millimeters. (Photo: Wellcome Museum of Medical Sciences)

conditions for hatching and subsequent larval development are:

(i) Shade from strong sunlight
(ii) Soil of the right particle size, denseness and structure; ideally a light sandy loam
(iii) Adequate, but not excessive, moisture; both desiccation and water-logging are rapidly lethal to hatched hookworm larvae
(iv) A temperature between 28–32°C for *N. americanus*, and between 20–27°C for *A. duodenale*; above and below these temperature ranges, larval development is slowed down and it is completely arrested below 10°C and above 40°C
(v) Adequate decomposing organic material and microorganisms in the soil to provide a food supply for the developing larvae.

If conditions are satisfactory, the larvae undergo two moults outside the human body, on the third and fifth days of their free-living existence, giving rise to third-stage filariform larvae, which become infective to man

about 6 days after hatching from the egg. These larvae normally survive for 3–6 weeks, and have a maximum life span of 15 weeks. Larvae have a maximum vertical range of migration of about 1 meter in suitable soil, but their lateral movement is restricted to about 0.3 meters and usually much less.

Infection of man occurs most commonly when the third-stage larvae penetrate the skin, usually between the toes or on the feet and ankles. In the case of *A. duodenale* only, the third-stage larvae can also infect man when they are ingested with unwashed, raw vegetables, onto which they can migrate from the soil. It is impossible to be certain of the relative importance of cutaneous and oral routes of infection with *A. duodenale;* probably they differ in different parts of the world with the food-eating and fertilizing habits of local populations. Experimental infections of man with *A. duodenale* are more easily produced by the oral than by the cutaneous route. Oral transmission of *N. americanus* is insignificant.

After penetration of the skin, larvae of both hookworm species enter small veins or lymphatic vessels and are carried to the heart and then to the lungs. *N. americanus* undergoes a period of development in the lungs, then ascends the bronchi and trachae, is swallowed, and reaches the small intestine in 24–48 hours after skin penetration; two further moults occur, about 1 day and 13 days after reaching the small intestine, *A. duodenale* undergoes the whole of its development in the small intestine, regardless of whether it enters the human body by the oral or cutaneous routes. Sexually mature fertilized females of both species begin egg laying between 4 and 8 weeks after infection.

*Prepatent and incubation periods*

The prepatent period (the interval between infection and the appearance of eggs in the stools) ranges from 4 to 8 weeks, with averages of 5 weeks for *A. duodenale* and 6 weeks for *N. americanus.*

The incubation period (the interval between infection and the development of symptoms and signs of illness) varies from a few weeks to several years, and depends on the number of worms that parasitize an individual, the individual's daily iron intake, body iron stores, and other blood losses. In light infections in healthy people, no evidence of illness may ever appear, although such individuals may be epidemiologically significant as egg-passers.

*Period of communicability*

Eggs will continue to be excreted for as long as there are adult female worms in the small intestine. Adult hookworms may live for up to 7 years in the case of *A. duodenale* and 15 years for *N. americanus* (Miller 1979). These are exceptional life spans, however, and 80 percent of worms survive for less than 3 years.

*Resistance*

Some degree of acquired resistance appears to develop with age and repeated reinfection, at least in some individuals. The problems of distinguishing between the effects of an immune response and the effects of varied exposure to infection are enormous in ancylostomiasis. Elderly people are frequently observed with high prevalence rates and high worm burdens. At present, the problem of immunity is of more interest to research workers than to those involved in investigating and controlling hookworm in field situations.

*Epidemiology*

Hookworm infections are extremely common and occur in many countries (figures 22-1 and 22-2). Perhaps 700 million persons are infected worldwide. There are many hundreds of reports of hookworm prevalence rates from almost every country in the world. A few are summarized in table 22-1. In some localities hookworm infects over half of the population, and it may be the most common intestinal worm. In other areas it is less common and is exceeded in prevalence by *Ascaris* or *Trichuris* or both.

There are major unexplained differences among communities in their prevalence of hookworm infection and in the relative prevalence of ascariasis, trichuriasis, and hookworm. These variations may occur even in a small geographical area—for example, within both northern and southwestern Iran (Ghadirian, Croll and Gyorkos 1979; Massoud and others 1980). These variations may sometimes be related to soil type and climate and the effect of these on egg and larval development and survival. They may also be due to differences in behaviour, settlement pattern, and agricultural practice. Because hookworm transmission is sometimes particularly vigorous in or near fields, rural prevalence rates are nearly always higher than urban rates (table 22-1). Rates are also higher in lower socioeconomic groups than among the more wealthy (table 22-1).

The distribution of hookworm infection among different age groups is typically fairly even, with the exception that prevalences are lower among children under 5 years old and sometimes in elderly adults. Ages of maximum prevalence vary considerably from community to community (table 22-1). Typically,

Table 22-1. *Prevalence of hookworm infection in fifteen countries*

| *Population* | *Location* | *Age (years)* | *Prevalence (percent)* | *Age of maximum prevalence (years)* | *Source* |
|---|---|---|---|---|---|
| Bangladesh | Rural | All ages | 29 | ND | Mackay and others (1979) |
| Colombia | Urban | All ages | 35 | 10–14<br>40–49 | Faust and Mugaburu (1965) |
| Egypt | Rural | All ages | 28 | 20–39 | Chandler (1954) |
| Gambia | Rural | All ages | 23 | 40+ | Bray and Harris (1977) |
| Guatemala | Rural | 1–5 | 12 | ND | Pierce and others (1962) |
| | Urban (poor) | 1–5 | 4 | ND | |
| | Urban (wealthy) | 1–5 | 0 | ND | |
| Haiti | Rural | All ages | 24 | 15–19 | Raccurt, Vial and Pierre-Louis (1977) |
| India | Urban | All ages | 8 | ND | Biswas and others (1978) |
| | Rural | 1–11 | 68 | ND | Nawalinski, Schad and Chowdhury (1978*a*) |
| Iran | Rural | All ages | 44–71 | ND | Arfaa and others (1977) |
| | Rural | All ages | 25 | 11–15 | Massoud and others (1980) |
| | Urban | All ages | 8 | 6–10 | |
| Ivory Coast | Rural | 7–14 | 73 | ND | Nozais, Dunand and Le Brigant (1979) |
| Malaysia | Rural | 6–12 | 43 | ND | Lo and others (1979) |
| | Urban (poor) | 4–6 | 5 | 13–15 | Yan and others (1978) |
| | Urban (wealthy) | 4–6 | 0 | ND | |
| Papua New Guinea | Rural | All ages | 68 | 20–29 | Jones (1976) |
| South Korea | Urban | All ages | 14 | 30–39 | Seo and others (1969) |
| | Rural | All ages | 19 | 30–39 | |
| Taiwan | Rural | All ages | 52 | over 60 | Hsieh (1970) |
| Thailand | Rural | All ages | 61 | ND | Bhaibulaya and others (1977) |
| Zambia | Rural | All ages | 49 | 6–10 | Wenlock (1979) |

ND No data.

teenagers or young adults are the most infected, and prevalence rates are usually somewhat higher in males than in females. This may be because much transmission takes place outside the village at the communal defecation place or in the fields. Young men may therefore be most exposed, and immunological factors may prevent ever increasing prevalences among older men.

Risk of hookworm infection in some rural areas is linked to occupation. Thus, in rice-growing villages in northern Iran much transmission takes place in or near the rice fields, and the 10–40 age group has the highest prevalence rate (Ghadirian, Croll and Gyorkos 1979).

The immunological factors mentioned above have a greater influence on intensity of infection than on prevalence. Although prevalence rates are often highest in adults, average worm burdens, as estimated by average fecal egg outputs, are nearly always greatest in children.

Improved living standards and better sanitation have eradicated hookworm from some areas of Europe and North America and have made it rare in others. Nevertheless, in the USA an estimated 700,000 persons are infected (Warren 1974), and it is especially common in poor rural areas of the southeast. Hookworm prevalences of 10–20 percent are still recorded among schoolchildren in parts of Alabama, Kentucky, and Georgia. Hookworm control has, however, proved easier than control of the other major intestinal nematodes, and ascariasis and trichuriasis are typically more prevalent than hookworm in poor southern areas of the USA (Fulmer and Huempfner 1965).

Early studies on hookworm in Tennessee (Otto, Cort and Keller 1931) and Virginia (Otto and Spindler 1930) suggested that high hookworm prevalences were associated with sandy soils and that most transmission took place at moist and shady sites where older children and adults defecated. This was in contrast to *Ascaris* transmission, which was concentrated in the yard where young children defecated. It is unclear whether this model of transmission has global validity, although there is evidence for hookworm transmission in the fields in several countries, and the hard packed earth around houses is unsuitable for development of hookworm larvae.

It is possible that most percutaneous hookworm infection takes place at habitual defecation sites and during defecation. The heavy fecal contamination of the soil can lead to a high density of larvae, and these may survive for several days if the soil is moist, loose, and shaded. The act of defecation by a barefooted person ensures the prolonged contact between foot and soil needed for successful infection. Hookworm eggs and larvae can be transported by houseflies (Oyerinde 1976), and this may conceivably add to the transmission of *A. duodenale* by the oral route.

The seasonality of hookworm infection remains poorly understood. In areas where *A. duodenale* is endemic, a pronounced seasonal fluctuation in egg output has been recorded. In Assam and Bengal (Bangladesh and India), hookworms are lost during the period from the late monsoon (September) till February, and this worm loss is accompanied by falling egg outputs (Maplestone 1930, 1932; Nawalinski, Schad and Chowdhury 1978*a*, 1978*b*; Schad and others 1973). Hookworm egg outputs then rise sharply during the period March–May, before the start of the monsoon. The simple explanation of this would be an increase in transmission approximately 6 weeks before, during January–March. This is a dry hot period in Bengal, however, when larvae are rapidly killed in the soil and when soil surveys find few if any infective larvae (Banwell and Schad 1978). Rises in egg output before the rainy season have also been recorded in Taiwan (Hsieh 1970) and in Indian prisoners who were not exposed to transmission during the preceding few months (Maplestone 1930). It is postulated that hookworm larvae acquired during the rainy season of one year become dormant and subsequently develop to maturity before the next rainy season. This arrested development of *A. duodenale* remains controversial, and there are few data from areas other than Bengal. No similar seasonal phenomenon has been reported from areas where *N. americanus* is the only endemic human hookworm.

## Control Measures

Many of the comments made about ascariasis control in chapter 23 apply equally to hookworm control. The major differences are, first, that the age of greatest prevalence of infection is typically higher in hookworm than ascariasis; second, that most ascariasis transmission may take place very near the home, whereas hookworm transmission typically occurs further afield; and, third, that *Ancylostoma* and *Necator* adult worms live very much longer in the intestine than *Ascaris*, and therefore the impact of reduced transmission will become apparent more slowly in the absence of mass chemotherapy. In some countries, for instance South Korea and the USA, hookworm infection has been easier to control than ascariasis, and this may be because it is less related to the hygienic behavior of young children and also because hookworm transmission is reduced by the increase in the practice of wearing shoes, where *Ascaris* transmission is not.

### *Individual*

No special prophylactic drugs are available for hookworm infection. A valuable method of personal prophylaxis is the wearing of adequate footwear at all times when the skin might come into contact with infective larvae, especially in latrines themselves and in the immediate vicinity of human habitations, as well as during agricultural work. However, universal wearing of footwear is clearly not an immediately practicable proposition, for economic as well as customary reasons.

In rural Costa Rica it was found that hookworm prevalence was not associated with sanitation but was related to the use of shoes (Moore, de la Cruz and Vargas Mendez 1965). Males who always or sometimes wore shoes had a prevalence of 20–23 percent compared with 36 percent among males who never wore shoes. The figures for females were 13 percent for those who always wore shoes, 29 percent for those who sometimes wore shoes, and 39 percent for those who never wore shoes. Much transmission took place at customary defecation sites in coffee plantations.

Several drugs are effective against hookworm infections and are administered orally. Some of these drugs also treat infections with other common intestinal worms; for instance, mebendazole is useful for combined infections of hookworm, *Ascaris*, and *Trichuris* (Nagalingam and others 1976). Anti-hookworm drug therapy, in combination with oral iron therapy, should form part of any specific hookworm control campaign.

Mass chemotherapy will rapidly reduce the prevalence and intensity of hookworm infection. The benefits may be short lived, however, in the absence of improvements in sanitation and education, and precontrol prevalence rates and intensities of infection recur within 1–5 years of the drug campaign. Some studies on the fall and subsequent rise of hookworm prevalence following mass chemotherapy are listed in table 22-2.

*Environmental*

Improvements in excreta disposal facilities, in areas of high prevalence, have failed to achieve a marked impact (table 22-3). In some studies, a combination of mass chemotherapy and improved sanitation has failed to affect the prevalence in the long term but has caused a marked decrease in the intensity of infection. Other studies have indicated that the treatment of night soil used as a fertilizer, combined with mass chemotherapy, is effective in reducing the prevalence (and presumably the intensity) of infection. This is probably due to a reduction in transmission to people working in fertilized fields. It is to be expected that night soil treatment will be particularly effective in communities where prevalences are not extremely high, where most transmission occurs in the fields, and where hygiene in and around the home is relatively good.

There can be no doubt that a massive improvement in hygiene and excreta disposal would greatly reduce the prevalence and intensity of ancylostomiasis, as it has in Europe and North America. What is uncertain is whether limited and specific improvements can either reduce infection or maintain lower levels following a mass treatment campaign. Existing studies indicate not, but such studies have generally been very deficient in behavioral observation, and behavior is all important in the success of any excreta disposal program. Poorly used and maintained latrines will clearly not reduce ancylostomiasis, and they may act as new foci for transmission. Toddlers (1–3 years old) may continue to defecate around the home and transmit infection to their siblings. Even if excreta disposal is improved around the home, it may be unaffected in the fields or work places where transmission may continue unchecked. More work is required on this topic, and it should incorporate detailed observations on behavior and studies of community acceptance and attitude.

Available evidence indicates that excreta disposal programs will fail to control hookworm infection by themselves. They have a valuable ancillary role to play, however, in conjunction with:

- Mass repeated specific chemotherapy
- Mass oral iron therapy
- Increased use of footwear
- Intensive health education campaigns
- Development of basic health services and infrastructure.

This approach reduced the national prevalence of hookworm infection in South Korea from 39 percent in 1949 to 7 percent in 1972 (Soh 1973).

Table 22-2. *Some studies on the effect of mass chemotherapy on hookworm infection*

| *Country* | *Drug* | *Source* |
|---|---|---|
| Bangladesh | Pyrantel | Mackay and others (1979) |
| Brazil | Chenopodium | Smillie (1922) |
| Costa Rica | Thiabendazole | Arguedas and others (1975) |
| Iran | Piperazine and bephenium hydroxynaphthoate | Arfaa and others (1977) |
| Japan | ND | Kawagoe and others (1958) |
| | ND | Kozai (1962) |
| | 1-bromo-2-naphthol | Kutsumi (1969) |
| Panama | Tetrachloroethylene and chenopodium | Cort, Schapiro and Stoll (1929) |
| | ND | Sweet and others (1929) |
| Puerto Rico | ND | Hill (1925, 1926) |
| Thailand | Pyrantel | Bhaibulaya and others (1977) |
| Zaïre | Levamisole | Jancloes, Cornet and Thienpont (1979) |

ND No data.

Table 22-3. *Some studies on environmental influences on hookworm infection*

| *Country* | *Result* | *Source* |
|---|---|---|
| Brazil | Sanitation was effective in reducing hookworm intensity but not prevalence | Smillie (1922) |
| Costa Rica | Hookworm prevalence was associated with type of house floor and wearing shoes but not with sanitation | Moore, de la Cruz and Vargas Mendez (1965) |
| Egypt | A village receiving improved water supplies, latrines, and refuse collection had a lower prevalence (10 percent) of hookworm than a village with unimproved sanitation (28 percent) | Chandler (1954) |
| | Improved sanitation did not reduce hookworm prevalence | Scott and Barlow (1938) |
| Iran | Water and sanitation improvements had almost no impact on prevalence and a modest impact on intensity of hookworm infection; water and sanitation improvements plus regular mass chemotherapy added nothing to the impact of mass chemotherapy alone | Arfaa and others (1977) |
| Japan | Heat treatment of night soil using firewood led to reduced prevalence of hookworm infection | Katayama (1955) |
| | Night soil treatment, by heating with electricity, did not reduce hookworm prevalence, but delayed reinfection following mass chemotherapy | Kawagoe and others (1958) |
| | Night soil treatment with sodium nitrite and calcium superphosphate reduced but did not prevent reinfection of hookworm following mass chemotherapy | Kozai (1960*a*, 1960*b*, 1960*c* and 1962) |
| | Night soil treatment with thiabendazole reduced transmission of hookworm | Kutsumi (1969) |
| Panama | Sanitation delayed reinfection following mass chemotherapy | Cort, Schapiro and Stoll (1929) |
| | Sanitation delayed reinfection (especially in females) following mass chemotherapy | Sweet and others (1929) |
| Puerto Rico | Sanitation delayed reinfection following mass chemotherapy | Hill (1925, 1926) |
| Singapore | Poor families rehoused in modern flats had a hookworm prevalence of 1.0 percent compared with that of squatters (2.2 percent) | Kleevens (1966) |
| USA | | |
| California | Rehousing mental patients in modern buildings interrupted hookworm transmission | Jeffery (1960) |
| Virginia | Sanitation appeared to be effective in reducing hookworm prevalence and intensity | Cort, Otto and Spindler (1930); Otto and Spindler (1930) |

## Occurrence and Survival in the Environment

Hookworm eggs and larvae in nature have been found mainly in soil at places where people defecate, or where night soil is applied to fields, and it is at these same sites that most percutaneous infection takes place. Eggs and larvae are also found on crops contaminated by night soil, sewage, or sludge. This pattern of contamination and infection has meant that there has been little interest in the possible wider dissemination of hookworm eggs and larvae in the environment. There has also been little interest in the survival of hookworm eggs in the environment because they are known to be less hardy than *Ascaris* eggs, and the latter therefore provide a better indicator organism for environmental helminthology.

### *In water*

Hookworm eggs will tend to settle in water and eventually die in the bottom sediments. Their survival

in seawater has been reported as under 5 hours, compared with over 30 hours for *Ascaris* eggs under the same experimental conditions (Livingstone 1978). In river water hookworm eggs can probably survive for a few weeks.

*In feces and night soil*

High concentrations of hookworm eggs are found in feces and night soil in endemic areas. Studies in Canton (China) in the 1920s recorded up to 85,000 viable hookworm eggs per liter of night soil (Oldt 1926). More recent studies from China found 840 eggs per liter (McGarry and Stainforth 1978).

Survival times of hookworm eggs in feces and night soil are considerably less than those of *Ascaris* eggs. In southern China, hookworm eggs in night soil and water mixtures were dead after 6–12 weeks (Oldt 1926). More recent work in China showed that hookworm eggs in biogas plant liquor at 9–18°C were reduced by 77 percent after 20 days, and by 99.5 percent after 70 days in winter (McGarry and Stainforth 1978). Similar results were reported by the Szechwan Research Institute (1974). Petrik (1954) considered that *Ancylostoma* eggs did not survive for more than 3 weeks in stored excreta at 20–22°C.

*In sewage*

At Daspoort (South Africa) settled sewage contained 6 hookworm eggs per liter (Nupen and de Villiers 1975). Very much higher concentrations may be expected in sewage from poor communities in developing countries. Sewage in Colombo (Sri Lanka) in the 1920s contained up to 330 hookworm eggs per liter (Hirst 1932). More recent data from Calcutta (India) showed that there were 22–750 hookworm eggs per gram of $BOD_5$ in the sewage (Bhaskaran and others 1956). Assuming a sewage strength of 250 milligrams of $BOD_5$ per liter, a hookworm egg concentration of 6–188 per liter may be computed. Lakshminarayana and Abdulappa (1969) detected up to 254 hookworm eggs per liter of sewage in Nagpur (India).

*In sludge*

Sludge from sewage treatment plants in areas of endemic hookworm may be expected to contain substantial hookworm egg concentrations. In Colombo (Sri Lanka) in the 1920s up to 96,000 hookworm eggs per liter of sludge from Imhoff tanks were recorded (Hirst 1932). In other areas hookworm eggs will be uncommon in sludge. A survey in the USA examined sludges from California, Georgia, Indiana, Kentucky, Montana, Ohio, and Wisconsin and found hookworm eggs only in the sludge from Frankfort, Indiana (Theis, Bolton and Storm 1978). An earlier survey of hookworm eggs and larvae in sludges in the USA was reported by Wright, Cram and Nolan (1942).

Hookworm eggs do not survive for long in sludge under tropical conditions. In Colombo (Sri Lanka) hookworm eggs could not be recognized microscopically after 43 days in sludge at 27°C and could not be cultivated after 23 days (Hirst 1932).

*In soil*

Under unfavorable conditions in soil (too hot, too cold, or too dry), hookworm eggs will either die or fail to develop and hatch. In either case they pose no risk. Under favorable conditions they will hatch and the resulting larvae will survive for less than 12 weeks.

Experiments on *A. duodenale* eggs in sterilized sandy soil in India (Vinayak, Chitkara and Chhuttani 1979) showed that in the hot dry months eggs hatched after 9–17 days, and the larvae survived for an average of 24 days. During the monsoon the eggs hatched after 5–12 days, and the larvae survived for an average of 90 days; in winter the equivalent periods were 45–62 days and 33 days. Clearly, warm wet conditions favor rapid hatching and prolonged larval survival. This was also found during experiments on *A. caninum* larvae on grass plots at Urbana, USA (Mark 1975). Mean larval survival times were $<1$ day during December–February, 7 days during March–July, and 24 days during August–November.

It may be expected that under field conditions larval survival in soil is shorter than recorded in laboratory experiments with sterilized soil. Even under ideal conditions (shaded, moist, sandy loam), over 99 percent of larvae die within 1 month (Banwell and Schad 1978).

The microhabitat of the hookworm larvae in soil is the moisture film surrounding the soil particles. If the larvae are near or on the surface and the moisture film dries, they will rapidly die from desiccation. In a loamy soil, larvae can move downwards to protect themselves from desiccation and bright sunlight. Following rain, they will move up to the surface again where they are at risk if the soil surface dries out very rapidly after a shower (Beaver 1953).

*On crops*

After rain, the moisture film on the surface soil particles may be continuous with the moisture films on

low vegetation, and hookworm larvae can make their way up onto leaves and stems. If the plant surfaces dry out, the larvae will rapidly die unless they are secluded in the axillae or other moisture-retaining and sheltered sites. Vegetable contamination by hookworm larvae is of importance in *A. duodenale* endemic areas only, since it is this species that can readily infect via the oral, as well as the percutaneous, route.

In countries where night soil is widely used to fertilize vegetables, hookworm larvae are commonly isolated from market produce. In South Korea, for instance, Choi (1970) isolated hookworm eggs and larvae from leafy vegetables, watercress, and carrots.

Vegetables suspected of being contaminated may be soaked in a solution of iodine or other ovicidal chemical. Iodine concentrations needed to destroy hookworm eggs are roughly double those required to kill larvae (Thitasut 1961). It is simpler and more reliable, however, to soak the vegetables in warm water (60°C) for 10 minutes.

## Inactivation by Sewage Treatment Processes

Large numbers of hookworm eggs enter sewage treatment plants in endemic areas. The fate of hookworm eggs during sewage treatment is similar to that of *Ascaris* eggs (discussed in greater detail in chapter 23). The two major differences are that hookworm eggs are less dense and that they may hatch.

Hookworm eggs are of similar size to *Ascaris* eggs but they have a lower specific gravity (1.055 compared with 1.11). Hookworm eggs therefore have a lower settling velocity and thus are less prone to removal by sedimentation processes (Cram 1943). Sedimentation is the main mechanism of removal of helminth eggs during sewage treatment, and therefore reported removals for hookworm eggs are typically a little less than those for *Ascaris* eggs.

Unlike *Ascaris* eggs, hookworm eggs may hatch during sewage treatment. Cram (1943) observed the hatching of hookworm eggs on trickling filter stones, in activated sludge tanks, and in drying sludge. Larvae will tend to stay in the liquid fraction and be carried out in the effluent; the eggs, however, tend to be concentrated in the sludge. The larvae may survive in sewage for up to 5 days (Cram 1943), which is quite sufficient for them to be discharged in the effluent and carried some distance.

Hookworm egg removal by sewage treatment is generally quoted as a percentage reduction in concentration between influent and effluent. This obscures the fact that most eggs not found in the effluent have not been destroyed but are merely concentrated in the sludge. Conventional sewage treatment plants have short retention times and operate at ambient temperatures and therefore do not destroy hookworm eggs. Their role is to transfer the eggs from the liquid fraction (the effluent) to the solid fraction (the sludge).

### *By septic tanks*

Most hookworm eggs will settle in septic tanks and be eventually removed in the sludge. Bhaskaran and others (1956) reported 72 percent removal of hookworm eggs by a septic tank and trickling filter bed in Calcutta (India). McGarry and Stainforth (1978) reported studies in China on a biogas plant, which may be likened to a septic tank with a long retention time. The influent contained hookworm eggs in 87 percent of samples, with an average concentration of 840 per liter; the effluent contained hookworm eggs in 23 percent of samples, with an average concentration of 4 per liter.

### *By conventional treatment*

Bhaskaran and others (1956) studied sewage treatment plants in the Calcutta area (India). Hookworm egg removals were 46 percent by 1.5 hours sedimentation (compared with 67 percent for *Ascaris*), 75 percent by 2 hours sedimentation (75 percent for *Ascaris*), 100 percent by an experimental trickling filter plant, and 81 and 96 percent by two activated sludge plants.

Panicker and Krishnamoorthi (1978) reported on hookworm egg removal by a variety of sewage treatment plants in India (table 22-4). Removal rates for hookworm eggs are consistently less than those for *Ascaris* eggs, and this is almost certainly due to the lower specific gravity of hookworm eggs and the resultant poorer removal by sedimentation.

### *By waste stabilization ponds*

Waste stabilization ponds are able to eliminate hookworm eggs completely and reliably. Table 22-4 indicates that two pond systems in India achieved this, whereas two other pond systems did not. No design details are given by Panicker and Krishnamoorthi (1978) for these ponds, but it is certain that ponds not removing hookworm eggs are poorly designed or poorly operated or both. Earlier research in India had shown that some hookworm eggs will hatch in aerobic waste stabilization ponds and that the larvae may be

Table 22-4 *Reduction of helminth eggs by sewage treatment processes in India*

| *Process* | *Hookworm*[a] | Ascaris | Hymenolepis | Trichuris trichiura | Taenia *spp.* |
|---|---|---|---|---|---|
| Two hours' sedimentation | 80 | 96 | 90 | 90 | 75 |
| Complete activated sludge plant | 85 | 98 | 95 | 100 | ND |
| Two complete trickling filter plants | 82 | 95 | 80 | 93 | ND |
| | 92 | 96 | 89 | 100 | ND |
| Pilot-scale biodisc plant with 1 hour of secondary sedimentation | 50 | 79 | 60 | 60 | ND |
| Pilot-scale oxidation ditch with secondary sedimentation | 81 | 94 | 89 | 100 | 100 |
| Pilot-scale aerated lagoon without secondary sedimentation | 70 | 92 | 78 | 100 | 100 |
| Four waste stabilization pond systems | 93 | 100 | 100 | 100 | 100 |
| | 88 | 100 | 100 | 100 | 100 |
| | 100 | 100 | 100 | 100 | 100 |
| | 100 | 100 | 100 | 100 | 100 |

ND No data.

*Source:* These data all taken from Panicker and Krishnamoorthi (1978).

*Note:* Percent reductions refer to influent compared with effluent. Most eggs not found in the effluent are concentrated in the raw sludge.

a. *Ancylostoma duodenale* and *Necator americanus.*

found in the effluent (Lakshminarayana and Abdulappa 1969). In a three-cell pond system with only 6 days' overall retention time, all hookworm eggs settled in the first and second ponds, but some larvae passed into the final effluent.

*By tertiary treatment*

Tertiary treatment of secondary effluents by filtration, land treatment, or lagooning will remove the remaining hookworm eggs (Cram 1943). Effluent chlorination will have little effect on hookworm eggs.

## Inactivation by Night Soil and Sludge Treatment Processes

Hookworm eggs and larvae are less resistant to night soil and sludge treatment processes than *Ascaris* eggs. If the recommendations on *Ascaris* egg destruction are followed (chapter 23), the destruction of hookworm eggs is guaranteed.

The application to pasture or arable land of raw or inadequately treated sludges containing hookworm eggs is especially undesirable because, once eggs are in the soil, development will continue and infective larvae will be produced. The risks may be reduced by plowing the sludge deeply in, or by injecting it below the surface (Romanenko 1967), but if the soil is loose and moist hookworm larvae will still be able to rise to the surface.

*By digestion*

Sludge digestion at temperatures below 40°C does not eliminate hookworm eggs (Petrik 1954). Cram (1943) found that hookworm eggs did not develop in digesting sludge but that they could survive for up to 64 days at 20°C and 41 days at 30°C.

*By ovicides and larvicides*

The addition of chemical ovidices and larvicides to night soil and sludge is a treatment option. Thiabendazole (Kutsumi and Komiya 1965) and several other chemicals (see, for instance, Sturrock 1966) have been tried or proposed. Hookworm eggs are considerably less resistant to ovicides than *Ascaris* eggs, and the treatments found effective against *Ascaris* (table 23-4) will certainly eliminate hookworm. For instance, the concentrations of thiabendazole necessary to destroy helminth eggs in 3 days in night soil at 15°C were 100 milligrams per liter for *Ascaris*, 6 milligrams per liter for *Trichuris*, and 1.6 milligrams per liter for hookworms (Kutsumi and Komiya 1965). This approach to night soil treatment will, however, be impractical and unaffordable in many circumstances. Of greater interest is the possibility that some chemical

fertilizers when added to night soil or sludge may have the combined effect of enhancing the agricultural value of the product and eliminating hookworm eggs and larvae. Oldt (1926) in China found that ammonium sulphate, lime, and gypsum were effective, whereas Penso (1933) in Italy preferred ferrous sulphate. In Japan, the application of sodium nitrite to night soil that had been acidified by adding calcium superphosphate controlled hookworm eggs in the night soil and also delayed reinfection in the community following mass chemotherapy (Kozai, 1960*a*, 1960*b*, 1960*c* and 1962).

*By drying*

Cram (1943) found that hookworm eggs hatched in drying sludge and that the larvae remained viable for up to 62 days until the moisture content of the sludge had fallen to 10 percent. At warmer temperatures, hookworm elimination in sludge is more rapid; Hirst (1932), working in Sri Lanka, suggested that 1 month of sludge storage would eliminate hookworm eggs.

*By heating*

*Ascaris* eggs are considerably more hardy than hookworm eggs, and time-temperature combinations that destroy the former (see figure 23-2) will certainly destroy the latter. Data on time-temperature combinations required to destroy hookworm eggs are presented in figure 22-4. Table 22-5 presents data on the upper and lower temperature tolerances for *A. duodenale* and *N. americanus* eggs and larvae. Clearly, eggs are considerably more resistant than larvae to both heat and cold. Also, *N. americanus* is more tolerant of higher temperatures and *A. duodenale* of lower. This is one underlying factor in their geographical distribution.

The most practical method of achieving the elevated temperatures needed to destroy hookworm eggs and larvae is by composting as described below. In Japan, heating night soil with firewood (Katayama 1955), or with cheap-rate night-time electricity (Kawagoe and others 1958), has been found to kill hookworm eggs in night soil and to reduce hookworm infection in the community. These techniques use large amounts of energy, however, and are not generally applicable.

*By composting*

Composting of night soil or sludge with garbage, woodchips, or other suitable carbonaceous bulking material is highly effective in eliminating hookworm

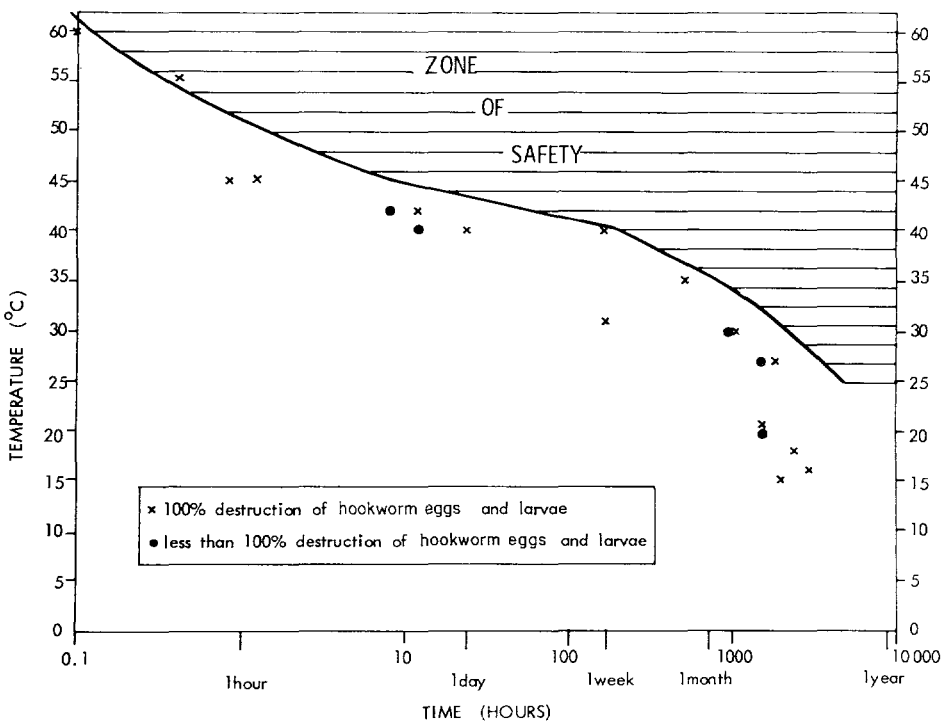


Figure 22-4. *The influence of time and temperature on hookworm eggs and larvae.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death. See also table 22-5

Table 22-5. *Tolerance of hookworm eggs and larvae to high and low temperatures*

| *Temperature limit* | Ancylostoma duodenale | | Necator americanus | |
|---|---|---|---|---|
| | *Eggs* | *Larvae* | *Eggs* | *Larvae* |
| Upper | 50°C for 5 minutes<br>60°C for 1 minute | 45°C for 5 minutes<br>50°C for 1 minute | 50°C for 5 minutes<br>65°C for 1 minute | 50°C for 5 minutes<br>55°C for 1 minute |
| Lower | −5°C for 9 hours<br>(41 percent dead after 3 hours) | 0°C for 7 days | −5°C for 9 hours<br>(93 percent dead after 3 hours) | 5°C for 1 day |

*Note:* See also figure 22-4.

eggs and larvae. Time-temperature combinations shown in figure 22-4 must be achieved throughout the compost pile. Hookworm eggs may hatch before temperatures have risen, and the resulting larvae may migrate to the cooler edges of the pile. Regular turning or good pile insulation is therefore required.

Studies on hookworm elimination by composting have been reported from China (Hou and others 1959; Oldt 1926), Sri Lanka (Nicholls and Gunawardana 1939), and the USSR (Gudzhabidze and Lyubchenko 1959). Other studies are reviewed by Petrik (1954) and Wiley (1962). *Ascaris* eggs are considerably more resistant to composting than hookworm eggs; in areas where both nematodes are endemic, it therefore is preferable to monitor *Ascaris* eggs in the final compost.

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# 23

# *Ascaris* and Ascariasis

ASCARIASIS IS AN INFECTION of particular importance to those engaged in sanitation programs because it is extremely common in most parts of the world and because the eggs of the *Ascaris* worm are very persistent in the environment and difficult to eliminate by sewage or night soil treatment processes.

## Description of Pathogen and Disease

Knowledge of the biology and epidemiology of ascariasis is extensive, and only a brief summary is presented in this section.

### *Identification*

Ascariasis is a helminthic infection of the small intestine by the human roundworm, *Ascaris lumbricoides*. About 85 percent of infections are symptomless, although the presence of even a few worms is potentially dangerous. The earliest symptoms are a pneumonitis with cough, dyspnea, substernal pain, fever, moderate eosinophilia, and sometimes blood-stained sputum (which may contain larvae). This is known as Loeffler's syndrome. These symptoms begin 5–6 days after infection, usually last 10–12 days, and are caused by the *Ascaris* larvae migrating and developing.

Heavy burdens of adult worms in the small intestine may cause digestive disorders, nausea, abdominal pain, vomiting, restlessness, and disturbed sleep. Adult worms may be passed in the feces or by mouth. Serious complications, especially among children, include bowel obstruction (figure 23-1) or death due to the migration of the adult worms to the liver, gall bladder, or appendix and, rarely, due to perforation of the intestine.

Where the prevalence and intensity of infection are high, the nutritional consequences of ascariasis in an undernourished population may be considerable. It has been estimated that a child who has twenty-six worms may lose 10 percent of his total daily intake of protein. There is also evidence that ascariasis in children may contribute to vitamin A and C deficiencies. The effect on child growth of light *Ascaris* infections, and the nutritional benefits from regular deworming, are subjects of current debate and research (Freij and others 1979; Gupta and others 1977; Stephenson 1980; Stephenson and others 1980; Willett, Kilama and Kihamia 1979).

Diagnosis is by microscopic identification of the eggs in the feces. The number of eggs counted gives an indication of the number of adult worms present.

### *Occurrence*

Ascariasis occurs worldwide and especially in warm climates and among poor people. It is one of the most prevalent human helminths and infects 700–1,000 million people. Prevalence and intensity of infection are particularly high in preschool and young school children, among whom prevalences of 60–90 percent are reported. The fatality rate is about 0.02 percent and higher in children.

### *Infectious agent*

*A. lumbricoides*, a nematode, is the roundworm of man. Females are 200–400 millimeters in length, whereas males are 150–300 millimeters (figure 23-1). The fertile eggs are ovoid and measure 45–70 micrometers by 35–50 micrometers. The pig roundworm, *A. suum*, closely resembles *A. lumbricoides*. There is evidence that *A. suum* may occasionally develop to maturity in man and that the larvae can cause pneumonitis even if the parasite does not mature. Several of the studies on *Ascaris* egg survival reported here use *A. suum* eggs because they are more readily available. The identical appearance of the eggs of *A. lumbricoides* and *A. suum* has almost certainly

Figure 23-1. Ascaris *in situ*. Shown is the small intestine of a person who died from intestinal obstruction caused by large numbers of entangled *Ascaris* worms. (Photo: Wellcome Museum of Medical Science)

confused some of the environmental studies of ascariasis in communities where domestic pigs are numerous.

*Reservoir*

The reservoir of *A. lumbricoides* is man. Pigs and dogs may disperse the undeveloped eggs of *A. lumbroicoides* by eating them in human feces and excreting them later at another place.

*Transmission*

Female worms lay up to 200,000 eggs per day. The unsegmented, fertilized eggs are passed in the feces. About 15 percent of the excreted eggs are infertile, and these are longer and narrower than the fertile eggs. They occur either because rapid production of eggs allows some to pass through unfertilized or because only female worms are present.

The first-stage larva in the egg must moult to produce a second-stage larva before the egg is infective. The proportion of eggs that develop and the time of development will depend on environmental conditions. Under ideal conditions of moist, shady soil at 22–33°C, a minimum of 10–15 days is required for about 75 percent of freshly passed eggs to become infective. Unfavorable conditions will retard development or may even interrupt it completely—to be recommenced on return to a favorable environment. Infective eggs can survive for long periods, and 7 years survival in soil has been recorded. During these long periods, eggs may be widely dispersed from the site of original defecation.

When the infective eggs are ingested—on hands, food, utensils, dust, and so forth—the larvae hatch in the duodenum. They are carried in the lymphatics or blood vessels, through the liver and heart, to reach the lungs in 3 days. The larvae develop further in the lungs, penetrate the air passages, ascend the trachea, are swallowed, and pass down the esophagus to reach the small intestine. They develop into adults in about 60–75 days and then live for up to 1.5 years.

The dominant vehicles for *Ascaris* egg ingestion are contaminated fingers, objects that have been placed on the ground (and can be sucked by children), dirt from the yard (that can be eaten by children) and

contaminated vegetables. Waterborne transmission is possible but is of very minor importance. Infection may take place by the inhalation of eggs stuck to particles of wind-blown dust. This is especially likely in dry and windy regions or seasons. There is practically no firm evidence on the degree to which this mode of transmission takes place, and the subject has scarcely advanced since the early review by Lane (1934).

There is now a considerable body of evidence to indicate that ascariasis in highly endemic areas is a familial infection. Most transmission occurs when the house, floor, yard, or area around the house is contaminated by promiscuous defecation by small children. This contaminated soil then reinfects other children and adults in the same family. Visitors may also be infected. Infection is clustered by family and is strongly associated with soil contamination around a family's home.

*Prepatent and incubation periods*

The prepatent period (the interval between ingesting infective eggs and the appearance of eggs in the stools) is about 2 months.

The incubation period (the interval between ingesting infective eggs and the development of symptoms and signs of illness) varies from a few days (in the case of symptoms due to migrating larvae) to several months. The development of clinical illness depends upon the number of worms in the body and the state of health of the host. In many light infections in healthy people, no evidence of illness ever appears.

*Period of communicability*

Adult worms generally live for less than 10 months, with maximum life spans of up to 1.5 years. As long as mature fertilized female worms are living in the intestine, eggs will be passed in the feces.

*Resistance*

Susceptibility is general, but there is evidence of limited immunity. Some children experience decreasing worm burdens despite continuing exposure.

*Epidemiology*

*Ascaris* infection is extremely common in many countries, and there are many hundreds of reports on its prevalence in various communities. Surveys of 1–5 year old children in Guatemala revealed ascariasis prevalences of 46 percent among rural children, 26 percent among poor urban children, and 3 percent among more wealthy urban children (Pierce and others 1962). In one village in Guatemala, 93 percent of children had at least one *Ascaris* infection between birth and the age of 3 years (Mata and others 1977). Surveys of preschool children showed prevalences of 11 percent in Iran, 48 percent in Sri Lanka, 53 percent in Venezuela, and 69 percent in Bangladesh (van Zijl 1966).

A survey of 4–6 year old children in Kuala Lumpur (Malaysia) found a prevalence of ascariasis of 64 percent among poor children and 2.5 percent among "upper middle class" children (Yan and others 1978). It was noted that, in the poor communities, most adults (90 percent) did not know how these worms are transmitted and that children under 10 years old defecated indiscriminately around the houses because the rudimentary latrines available were unsafe for children to use on their own. A study of rural school children (6–12 years old) 72 kilometers from Kuala Lumpur found an 87 percent prevalence of ascariasis (Lo and others 1979). Sanitation facilities did not affect ascariasis prevalences among these children.

There are major unexplained variations among communities in the prevalence of *Ascaris* and in the relative prevalences of *Ascaris*, *Trichuris*, and hookworm. These may be related to soil types; it has been suggested that hookworm is especially common in areas of sandy soil, whereas *Ascaris* rates are highest in areas of fine silts and clays. Such theories appear to have no global validity. Climatic factors certainly influence egg development in the soil and, therefore, transmission. Temperatures of 20–32°C are ideal, with little development taking place below 18°C. A moist, shady environment also encourages egg development, and eggs may be killed or inhibited by desiccation or exposure to sunlight (Nolf 1932). Spindler (1929) concluded that the different moisture requirements of the eggs was the primary influence on the variation in the relative prevalences of ascariasis and trichuriasis and caused trichuriasis to be the more common in wetter areas, whereas the reverse was true in drier areas.

A study of the distribution of ascariasis in Tennessee (USA) concluded that the marked variations in prevalence were due neither to soil type, rainfall, nor temperature, but to behavior, and especially to the practice of defecating in the yard near the house (Otto, Cort and Keller 1931). Similarly, rainfall was found not to be a determining factor in *Ascaris* distribution in Panama (Cort and others 1929), nor soil type in Virginia (USA; Cort, Otto and Spindler 1930).

In areas where ascariasis is highly endemic, one of two distinct age distributions is found. In some

communities there is a peak prevalence among children, typically 70–95 percent in the 5–9 age group, with prevalence gradually decreasing in older age groups to 10–50 percent among old adults. This pattern has been reported from China (Cort and Stoll 1931), Colombia (Faust and Mugaburu 1965), Haiti (Raccurt, Vial and Pierre-Louis 1977), Iran (Massoud and others 1980), Mexico (Biagi and Rodriguez 1960), Panama (Cort and others 1929), Papua New Guinea (Jones 1976*b*), and the USA (Cort, Otto and Spindler 1930; Schliessmann and others 1958). In other localities there is a high prevalence (typically 40–80 percent) in all age groups. This has been found in China (Cort and Stoll 1931), Iran (Ghadirian and others 1973; Mobedi, Arfaa and Movafegh 1971; Sahba and Arfaa 1967), Japan (Kutsumi 1969), Philippines (Cabrera, Arambulo and Portillo 1975), South Korea (Seo and others 1969; Soh 1973) and Tunisia (Thiers, Lassoued and Abid 1976).

Possible explanations for these two patterns of age-specific prevalence are differences in the parasites or in host reactions. Geographical differences in the parasites or in immune responses to childhood infection are theoretically possible although not recorded. Immune response differences between racial groups are an unlikely explanation because both patterns have been found in a single racial group (for instance, in China—Cort and Stoll 1931). It is more likely, therefore, that the two patterns of age-specific prevalence derive from differences in exposure due to differences in behavior, housing, agricultural practice, sanitation, and other environmental and cultural factors. Cort and Stoll (1931) suggested that the heavy and common infection of adult males with *Ascaris* in some parts of China was due to the intensive use of human feces in agriculture.

Several surveys, for instance in Virginia (USA; Cort, Otto and Spindler 1930) and Panama (Cort and others 1929), have revealed a somewhat higher prevalence of infection in women than in men. This difference is not seen in children. If the main source of infection is eggs deposited by infected children around the house, women may be more exposed because they spend more time than men working in the yard and tending to children. If vegetables fertilized by night soil are a major source of infection, women may also have greater exposure if they are responsible for their harvesting, cleaning, and preparation.

Ascariasis is common in some developed countries. There are an estimated 4 million people infected in the USA, with the disease being especially common in the southeast (Warren 1974). As in other countries, infection rates are highest among children, and there are about 2 cases of intestinal obstruction caused by ascariasis per 1,000 preschool children per year there; 3 percent of these cases of obstruction die (Blumenthal 1977). In the poorer parts of the USA it has been noticed that, whereas the wearing of shoes has caused a considerable decline in hookworm prevalence, *Ascaris* and *Trichuris* remain common (up to 60 percent of schoolchildren infected) owing to a lack of adequate improvements in sanitation and hygienic behavior (Fulmer and Huempfner 1965).

Intensity of infection (the number of worms living in the intestine) is quite as important in the epidemiology of ascariasis as prevalence (the proportion of persons having at least one worm). Heavy infections have more serious clinical consequences and are the infections most likely to be seen at clinics. In addition, persons with heavy infections are excreting huge numbers of eggs (up to 200,000 per female worm per day) and are therefore causing considerable potential for transmission. Individual egg outputs of up to about 300,000 per gram of feces are reported; assuming 100 grams of feces per day, this suggests at least 150 mature female worms in the intestine. The ratio of male to female worms in the intestine varies from about 0.3 to 1.

Children have higher egg outputs, owing to greater intensity of infection, than adults. This applies in both the age-specific prevalence situations described above. Thus, whether or not prevalence falls with age, intensity does, and this suggests an immune response that does not prevent infection but limits the intensity of infection by reducing the proportion of ingested eggs that develop into mature adults. Women may have higher egg outputs, indicating heavier infections, than men, which may be explained by the greater exposure of women discussed above.

A fundamental influence on the epidemiology of ascariasis is that a small minority of infected people are excreting the majority of the eggs. In China it was found that 5 percent of those infected excreted half of all eggs (Cort and Stoll 1931). Heavy infections tend to be single-brood infections. In other words, they are not an accumulation of worms from eggs ingested regularly over several months; rather, they are worms of a single age arising from the ingestion of a batch of eggs at one time. This may be due to an immune mechanism triggered by adult worms in the intestine and acting against the migrating larvae resulting from subsequent egg ingestions. More research is required in this area.

Not much is known about the seasonality of ascariasis. During a pronounced dry season transmission may decrease because the *Ascaris* eggs are desiccated in the dry soil. Therefore, there may be few

new infections, and these will tend to be light. Since natural death is occurring among adult worms, intensity of infection, and possibly prevalence, will fall during the dry season. Transmission may increase with the rains and may cause an increased intensity, and possibly prevalence, about 2 months later. These climatic factors may be reinforced or negated by other seasonal changes, such as agricultural and dietary practices, that may influence exposure and resistance to infection. A November–December peak in transmission in Korea was attributed to contaminated pickled vegetables (Seo, Cho and Chai 1979).

The climatic influence on transmission described above might cause a pronounced seasonal pattern in intensity of infection (mean egg output) but little change in prevalence. Studies in Colombia (Faust and Mugaburu 1965), Panama (Cort and others 1929), and Taiwan (Chen and Hsieh 1969) have confirmed this seasonal picture. Seasonality in transmission may also lead to a seasonal peak in pneumonitis associated with *Ascaris* larvae migration, as has been reported from Saudi Arabia (Gelpi and Mustafa 1967).

There are three primary contexts for *Ascaris* transmission:

- Transmission in yards and compounds that have been contaminated by feces, especially those of children
- Transmission to persons working in fields where night soil or sewage is used as fertilizer
- Transmission to persons consuming vegetables that have been grown in fields enriched with night soil or sewage.

Yard transmission is probably the dominant mode in areas of poverty and inadequate sanitation. Families with heavily polluted yards tend to have high prevalences and intensities of ascariasis, and ascariasis is typically clustered by household. Familial aggregation of ascariasis has been reported from China (Winfield 1937), Tennessee (USA; Otto, Cort and Keller 1931), Virginia (USA; Cort, Otto and Spindler 1930; Hendley, Williams and Burke 1973), and Panama (Brown 1927; Cort and others 1929). The studies in Tennessee and Virginia also suggested that children from households with good sanitation could become infected when they visited heavily infected families and that rural schools with inadequate sanitation and heavy soil pollution acted as dissemination points for ascariasis in the community. *Ascaris* eggs were recovered in Egypt from the playgrounds of schools with no latrines, or with poorly maintained latrines, but not from schools with well-maintained latrines (Chandler 1954).

The pattern of yard contamination and infection may be modified by coprophagous domestic animals. Chickens, cats, and dogs in Tennessee (USA) were all found able to ingest *A. lumbricoides* eggs and to pass a proportion of them unharmed in their feces (Otto, Cort and Keller 1931). Pigs are enthusiastic consumers of human feces and may be especially important in distributing human *Ascaris* eggs. Jones (1976*a*) suggested that pigs in Papua New Guinea may play a role in transporting *Ascaris* eggs into villages, or even into homes, from human defecation sites that are often in thick undergrowth away from dwellings.

Transmission may take place not only in the fecally contaminated yard, but also in the home if the floor is made of a material that will permit the maturation of *Ascaris* eggs. Brown (1927) found that eggs deposited by small children onto hut floors in Panama developed to maturity in 14 days. Viable eggs have also been isolated from house floors in China (Winfield 1937) and Egypt (Chandler 1954). Studies in the USSR found that *Ascaris* eggs could develop on household objects and in floor cracks if humidities were high, and could remain viable for over 3 months in cool, humid environments such as cellars (Barchenko 1955).

The other two contexts for transmission mentioned above, transmission in the fields and via contaminated vegetables, are prominent either where feces are much used in agriculture or where hygiene is improved to the point at which yard transmission becomes relatively unimportant. The role of field infection has been much stressed in the Chinese literature, whereas both fields and contaminated vegetables are emphasized by workers in Japan, Korea, Taiwan and neighboring areas. The importance of vegetable contamination as a single factor has been stressed by writers from countries in which a well-educated and moderately wealthy population lives near to, and buys vegetables from, a poor community with endemic ascariasis; prominent examples in the literature are Israel, South Africa, and parts of the USSR.

The very high prevalences of ascariasis around Darmstadt (Federal Republic of Germany) in the late 1940s were due to the widespread practice of applying untreated sewage and sludge to fields and vegetable gardens (Baumhogger 1949). The prevalence of ascariasis among the Jewish population of Jerusalem decreased from 35 percent in the period 1934–47, to 1 percent in the period 1947–60. The decrease was attributed to the cessation of supplies of sewage-irrigated vegetables from Jordan after the partition of the city in 1948. In contrast, the prevalence of ascariasis in the Jordanian section of Jerusalem remained high, at 78 percent, in the early 1950s (Ben-Ari 1962). Khalil

(1933) documented a 96 percent prevalence of ascariasis among 5–16 year old children in the oasis of Siwa (Egypt) and attributed this to use of human wastes in agriculture.

Workers in formal sewage farms or sewage effluent irrigation schemes are also exposed to increased risk of *Ascaris* infection (Clark and others 1976). A study in Germany during 1954–56 showed ascariasis prevalences of 3 percent among sewermen, 16 percent among sewage treatment plant workers, 30 percent among sewage irrigation workers, and 8 percent among a control group (Sinnecker 1958).

This section is concluded by citing the account by Winfield (1937) of ascariasis in northern China in the 1930s. Each house had a combined animal shed and pit latrine. This comprised a pit, 3–5 meters square and about 2 meters deep, lined with brick or stone and with a base of tamped lime and clay, so that the whole pit was fairly watertight. The family defecated into the pit and also placed animal manure, organic refuse, and slops in the pit. Field earth was added at the rate of about one basketful per day. During dry weather, water was added to keep the pit contents wet; in the rainy season, the pit contained standing water and was the site of mosquito breeding. Adjacent to the pit was a shed where pigs and other animals were housed. Steps led down into the pit to allow the pigs to enter to eat fresh feces and to wallow in the muck. Usually the pit was emptied every spring and the waste was piled along the streets or on the village threshing floor. After a few days the waste was carted to the fields and placed carefully about the roots of the winter wheat or worked into the ground in preparation for the spring crop. Some families emptied their pits more than once a year, in which case the waste would be piled along the streets with a layer of straw-reinforced mud as a protective cover. It would then be used at the time of the next spring sowing.

*Ascaris* infection was very common, especially in those families having contaminated yards. *Ascaris* eggs were readily found in samples of soil from yards, floors and streets. Winfield concluded that fecal contamination of the yards and floors was the dominant cause of transmission and that contaminated water and vegetables were unimportant (see also Winfield and Yao 1937). Yard and floor contamination were caused by the casual defecation of children, who tended not to use the latrines because of inconvenience and fear of the pigs. *Ascaris* eggs were also distributed in the yards by chickens and dogs that fed on human excreta, both in the latrine and elsewhere. Contamination of the yard also occurred during the periodic emptying of muck from the pit for transport to the fields.

## Control Measures

Only environmental and behavioral changes can have a sustained impact on ascariasis, but mass chemotherapy may be used to reduce infection rates in the short term.

### *Individual*

No vaccines or prophylactic drugs are available. Individual protection can be obtained by scrupulous personal hygiene and care in choice and preparation of food, especially vegetables.

The most immediately effective method yet applied in areas with high prevalence is mass chemotherapy. The administration of appropriate drugs (such as levamisole, mebendazole, or pyrantel pamoate) to all individuals, or to all children, at regular intervals has dramatically reduced prevalences in several local trials. Mass treatment should start at the period in the year when worm burdens are highest and should ideally be repeated at intervals of not more than 60 days for as long as infective eggs remain in the soil. In most regions it would be impossible to prevent the reintroduction of infection following a mass treatment program, and therefore it is unlikely that mass treatment will be effective in the long term without concurrent improvements in excreta disposal and hygiene. In the absence of these preventive measures, prevalence may return to pretreatment levels within 6–12 months, although intensities (as measured by mean egg output) take somewhat longer to build up again. Literature on mass chemotherapy and reinfection in various countries is listed in table 23-1.

### *Environmental*

Transmission of ascariasis generally occurs following the contamination with feces of the house floors, the yard, or the area around the house. Eggs develop to the infective stage and reinfect a child or adult who accidentally ingests a particle of contaminated soil or dirt. In areas of high prevalence, there is good evidence that most infection takes place in, or close to, the house and is clustered by family. Eggs are deposited primarily by small children who may defecate promiscuously in or near the home. Eggs may also be deposited by pigs, dogs, or chickens that have fed on human feces and can pass the human *Ascaris* eggs unharmed in their own feces.

This kind of family-centred transmission can be controlled, in theory, by providing a hygienic toilet *for all members of the family* and by providing the necessary

Table 23-1. *Some studies on the reduction and subsequent rise of ascariasis prevalence following mass chemotherapy*

| *Country* | *Drug* | *Source* |
|---|---|---|
| Colombia | Pyrantel pamoate | Spillmann (1975) |
| India | Tetramisole | Gupta and others (1977) |
| Iran | Piperazine | Arfaa and others (1977) |
| | Levamisole | Arfaa and Ghadirian (1978) |
| | Levamisole | Massoud (1980) |
| Mexico | Piperazine | Biagi and Rodriguez (1960) |
| Panama | Tetrachlorethylene and chenopodium | Cort, Schapiro and Stoll (1929) |
| Philippines | Pyrantel pamoate | Cabrera, Arambulo and Portillo (1975) |
| | Piperazine | Garcia and others (1961) |
| | Levamisole | Jueco and Cabrera (1971) |
| Réunion (Indian Ocean) | Thiabendazole<br>L-tetramisole | Coumbaras and others (1976) |
| Taiwan | Piperazine | Chen and Hsieh (1969) |
| Thailand | Pyrantel pamoate | Bhaibulaya and others (1977) |
| USA | Piperazine | Atchley, Wysham and Hemphill (1956) |
| Zaire | Levamisole | Jancloes, Cornet and Thienpont (1979) |

health education to ensure that the toilet is used. Because children are the main excreters of *Ascaris* eggs, it is essential that any toilets should be acceptable and appropriate for use by children. In addition, it is necessary to clear up the stools of babies who are too young to use a toilet. Fresh stools are not immediately infective for *Ascaris*, and so they may be cleaned up any time up to a few days after deposition and still interrupt *Ascaris* transmission—except that delay in clearing up will increase the risk of dispersion or ingestion of eggs by domestic animals.

Improvements in excreta disposal facilities, defecation behavior, and child care can greatly reduce transmission. Bearing in mind that adult worms die naturally after 6–18 months in the small intestine, reduced transmission will cause a gradual fall in intensity of infection followed by a gradual fall in prevalence. Because the domestic environment may be heavily contaminated with *Ascaris* eggs that may remain infective for many months, transmission will continue for some time after sanitation has been improved, and there may be a considerable lag before reduced intensities and prevalences are measurable. There will be an even greater lag before reduced infection reaches a level at which it is apparent to mothers. This delayed response to reduced transmission is undesirable from a clinical viewpoint and will tend to decrease community enthusiasm for the preventive measures being advocated. It is for this reason that a combination of mass chemotherapy and sanitation is the best approach to ascariasis control.

In a combined program of mass chemotherapy, sanitation, and education the role of sanitation and education is to maintain the low intensities and prevalences created by the mass chemotherapy. This should be perfectly possible, and in recent years several East Asian countries have achieved notable success in ascariasis reduction by this approach. South Korea has had mass campaigns for the prevention of ascariasis including health education, stool examination and mass chemotherapy. Latrines have been widely provided. In 1949, the national prevalence of ascariasis was 81 percent, but by 1971 it was 46 percent (Soh 1973). These measures were also effective against trichuriasis (from 87 to 47 percent) and hookworm (from 39 to 7 percent).

There is no doubt that behavior is all important in the success of an excreta disposal program. Poorly used and maintained latrines will achieve little and may even increase transmission. Children may continue to defecate around the home and transmit eggs to their siblings. Even if excreta disposal is improved around the home, it may be unaffected in the fields, work places, or schools, where transmission may continue

unchecked. Following studies in southwestern Virginia (USA) in 1928, Cort, Otto and Spindler (1930) wrote:

> Several groups of negroes, one of which was extremely poor, as well as numbers of poor white families showed little or no *Ascaris* infestation because of the control of the children and the use of the privies by all members of the families. On the other hand some of the better-off rural families with well-kept yards and good privies and certain families in very well-sanitated mining camps had heavy infestations. Such infestations were almost always due to soil pollution near the houses by the young children, who were not taught to use the sanitary facilities provided.

Similarly, following work in China, Cort and Stoll (1931) wrote:

> It was of interest to find one group in the Yangtze delta with a comparatively low infestation with both parasites [*Ascaris* and *Trichuris*] associated with a good economic status and habits of cleanliness much better than the average for rural China. This shows that human infection with these parasites can be much restricted even where their eggs are spread widely by the use of human feces as fertilizer.

The application of untreated nightsoil or sludge to the land undoubtedly contributes to *Ascaris* transmission. People working in the fields may be infected, and eggs may be brought into the home on soiled vegetables. Undeveloped eggs brought into the home can subsequently become infective if they end up on an earth floor or in a moist cranny. This route of infection is likely to be of minor importance where prevalence and intensity are high and where transmission is occurring primarily around the house. However, in areas where domestic hygiene is relatively good and toilets are used, contaminated crops may be the major route by which eggs are introduced into the household. Control of this agricultural transmission route is by adequate treatment of sludge and night soil prior to land application.

Studies on the environmental control of ascariasis are listed in table 23-2; more details of some of them are given in table 2-1. In most studies confounding variables were not adequately controlled, and it is not possible to separate the effect of sanitation, for instance, from that of socioeconomic and educational changes. Sanitation appeared to have an influence on ascariasis prevalence in some communities in Costa Rica, Egypt, Germany, Iran, Singapore, South Korea, and the USA, but it appeared to have little or no influence in Egypt, Panama, and the USA. Treatment of night soil, by heat or chemical ovicides, was effective in controlling ascariasis in some communities in Japan and the USSR.

Cases in which sanitation appeared ineffective are most probably due to insufficient use of, or to poor hygiene in, the latrines or to transmission continuing away from the home at the place of work or recreation. Unfortunately, most studies do not clearly demonstrate this because they are deficient in behavioral observations. Constructing latrines and measuring health changes does not provide the data necessary to explain any impact, or lack of impact, observed. Detailed observations of actual use of latrines, especially use by children, are required but are very seldom carried out. Also rare are studies on traditional beliefs and practices concerning ascariasis—such as that conducted in rural west Malaysia by Chen (1970)—which can be invaluable in the design of health education campaigns to accompany sanitation and mass chemotherapy programs.

In conclusion, it is sobering to consider two statements made half a century ago, which remain true but sadly ignored today. Otto and Spindler (1930) wrote:

> The building of privies is a fundamental step in the control of infectious diseases spread by soil pollution, but to be successful they must be used exclusively. In the regions just discussed [Virginia] the situation is difficult because many of the people are doubtful of the value of a privy. In many cases they feel that it produces an unwise accumulation of odorous and obnoxious waste which if daily dropped in various parts of the yard would be destroyed by insects or chickens and washed into the soil by the rains. Those adults who do consent to build and use the privy often do not feel sufficiently convinced of its beneficial results to take any time to teach the young children to use it. Furthermore, as has already been pointed out, the pit privy as usually built is structurally ill adapted to children's use and frequently inconveniently situated. The seat is too high and the hole too large to be conveniently and safely used by young children. A lower seat or a step to part of the main seat with a smaller hole for the convenience of young children should certainly be encouraged. The real problem, however, is the slow process of educating these people to consider the sanitary privy as one of the most

Table 23-2. *Some studies on environmental influences on ascariasis*

| *Country* | *Result* | *Source* |
|---|---|---|
| Costa Rica | Ascariasis prevalence was lower among those with improved sanitation | Moore, de la Cruz and Vargas-Mendez (1965) |
| Egypt | A village receiving improved water supplies, latrines, and refuse collection had a lower prevalence (50 percent) and intensity (4,200 eggs per gram) of ascariasis than a village with unimproved sanitation (prevalence = 76 per cent; intensity = 6,900 eggs per gram) | Chandler (1954) |
| | *Ascaris* rates remained high among prisoners, despite falling rates of hookworm and schistosomiasis, owing to regular reinfection by contaminated vegetables grown on the prison sewage farm | Khalil (1926, 1931) |
| | Improved sanitation, with and without chemotherapy, did not reduce ascariasis in several villages | Scott and Barlow (1938) |
| Germany | Prevalences of ascariasis in schoolchildren in Berlin were 3 percent in sewered areas, 7 percent in unsewered rural areas, and 14 percent in unsewered urban areas | Anders (1954) |
| Iran | Water and sanitation improvements had little impact on prevalence but considerable impact on intensity of ascariasis; water and sanitation improvements plus regular mass chemotherapy added nothing to the impact of mass chemotherapy alone | Arfaa and others (1977) |
| | Prevalence of ascariasis was the same among people with and without sewerage in Isfahan | Sadighian and others (1976) |
| | Water supply, sanitation, and bathing improvements were associated with a fall in prevalence (67 to 57 percent) and intensity (11,000 to 4,000 eggs per gram) in a single village | Sahba and Arfaa (1967) |
| Japan | Heat treatment of night soil prior to agricultural application was associated with a declining prevalence of ascariasis | Katayama (1955) |
| | Heat treatment of night soil had little effect on ascariasis but was effective in maintaining a lowered prevalence achieved by mass chemotherapy | Kawagoe and others (1958) |
| | Night soil treatment with sodium nitrite and calcium superphosphate reduced but did not prevent rising prevalence of ascariasis following mass chemotherapy | Kozai (1960*a*, 1960*b*, 1960*c*, 1962) |
| | Night soil treatment with thiabendazole reduced ascariasis prevalence by 30 percent over 2 years and night soil treatment plus mass chemotherapy reduced it by 50 percent | Kutsumi (1969) |
| Panama | Sanitation did not delay reinfection following mass chemotherapy | Cort, Schapiro and Stoll (1929) |
| Singapore | Poor families rehoused in modern flats had an ascariasis prevalence of 9 percent compared with that of squatters (63 percent) | Kleevens (1966) |

Table 23-2. (*continued*)

| *Country* | *Result* | *Source* |
|---|---|---|
| South Korea | Higher intensities of ascariasis occurred in poorer parts of Seoul using vault latrines than in wealthier areas with sewerage | Soh and others (1973) |
| USA | | |
| Kentucky | Ascariasis prevalence was associated with both water supply and sanitation facilities | Schliessmann and others (1958) |
| Tennessee | Ascariasis prevalence was associated with sanitation, fecal contamination, domestic and personal cleanliness, family size, but not water quality | Eyles, Jones and Smith (1953) |
| | Pit latrines were not effective in reducing ascariasis because they were commonly not used by children, who defecated in the yard | Otto, Cort and Keller (1931) |
| Virginia | Pit latrines were not effective in reducing ascariasis because they were commonly not used by children, who defecated in the yard | Otto and Spindler (1930) |
| USSR | Cessation of use of untreated night soil as a fertilizer was associated with a marked decrease in *Ascaris* egg contamination of soil and fruit | Rosenberg (1960) |

necessary buildings on the premises of every family and its use taken as a matter of course. When this is accomplished, and not until then, will human excreta be so disposed as to protect not only against *Ascaris* infestation but against bacterial diseases as well.

Cort (1931) wrote:

In many places *Ascaris* infestation has been reduced or eliminated by sanitary programs carried out over long periods of time, especially where other factors have raised the social and economic level of the population. It has been shown, however, that very frequently sanitary work has not been successful in controlling this parasite because of the failure of young children to use the facilities provided. For *Ascaris* control, therefore, it is necessary to provide facilities well adapted for children's use and to concentrate the educational program on the prevention of soil pollution by young children. Since the presence of this parasite in both cities and rural communities over such wide areas of the world can be used as an index of the status of sanitation it seems that campaigns for its control might form a larger part than at present of programs for the improvement of sanitary conditions.

## Occurrence and Survival in the Environment

*Ascaris* eggs may be the most hardy and resistant of all excreted pathogens. They can survive a variety of environmental conditions for periods of months or even years. They need small quantities of oxygen to develop but can remain viable for long periods in anaerobic conditions.

### *In surface water*

The occurrence and survival of *Ascaris* eggs in waters is not a very important subject because little or no transmission is waterborne.

Usacheva (1951) found *Ascaris* eggs in a high proportion of river water and river sediment samples in the USSR. All samples contained *Ascaris* eggs at times of flooding. Survival experiments revealed that 12 percent of eggs in river water, and 17 percent of eggs in sediment, were viable after 15 months (presumably the temperatures were cool). Goryachev (1947) also isolated *Ascaris* eggs from river water and sediment near Omsk and found more in winter than in spring.

Iwańczuk (1969) found, on average, 0.8 *Ascaris* eggs per 100 grams of soil at six public beaches on the shores of the River Vistula in Poland. The largest number of eggs and the most advanced in development were found in the damper zones, such as the water's edge, in the shade of bushes, and near public toilets. The beaches were not close to sewage outfalls and it is probable that most eggs came from defecation by beach visitors, rather than from the river water.

*Ascaris* eggs are especially likely to occur directly downstream from sewage outfalls. Near Denver (Colorado, USA) a river contained 0–1 eggs per liter

above a sewage outfall, and 0–14 per liter below it. *Ascaris* eggs were also recovered 3 kilometers downstream from the outfall (Wang and Dunlop 1954).

*In groundwater*

*Ascaris* eggs are unlikely to occur in groundwater because their size causes them to be retained as polluted surface waters percolate down through porous strata. They may, however, occur where surface waters are flowing directly into groundwaters via fissures in metamorphic rocks or solution channels in limestone.

*In drinking water*

No reports of *Ascaris* eggs in drinking water have been found. As mentioned above, their presence is of little interest because waterborne transmission is unimportant relative to yard and field transmission. Chlorine and chloramine are completely ineffective against *Ascaris* eggs at, or even greatly above, the concentrations typically applied during water treatment.

*Ascaris* eggs were found in the raw river water supplying Ufa (USSR) but not in the tap water (Bukh 1945).

*In seawater*

Yarulin (1955) found *Ascaris* eggs in the coastal waters of the Caspian Sea near sewage outfalls. Eggs remained viable for considerable periods in seawater but did not develop until removed. Laboratory experiments in South Africa found that 97 percent of *Ascaris* eggs were killed after 2 days in seawater. They were considerably more resistant than *Trichuris*, hookworm, or *Enterobius* eggs, but somewhat less resistant than *Taenia* eggs (Livingstone 1978). The specific gravity of *Ascaris* eggs is about 1.11, and so they will settle in seawater and in seawater and sewage mixtures with specific gravities of 1.00 to 1.03.

*In feces and night soil*

The high prevalence of *Ascaris* egg excretion in some communities has been described above in the section on epidemiology. Some individuals excrete large numbers of eggs, up to 300,000 per gram of feces, and thus night soil may be rich in *Ascaris* eggs. If the prevalence is 60 percent, the mean egg output of those infected 10,000 eggs per gram, and the mean night soil volume of 2 liters per capita daily, then the night soil will contain $3 \times 10^7$ eggs per liter. Concentrations this high have not been reported. Night soil in Kiangsu Province (China) contained $2.3 \times 10^6$ *Ascaris* eggs per liter (McGarry and Stainforth 1978).

Takenouchi and others (1980) studied *Ascaris* eggs in night soil in Kochi Prefecture in Japan. *Ascaris* egg concentrations were higher in night soil from mountain areas than from coastal regions, and it is suggested that the regular enumeration of *Ascaris* eggs in night soil is a useful method of monitoring ascariasis in the community.

Survival of *Ascaris* eggs for over 1 year is possible in feces and night soil. In anaerobic conditions development is arrested but recommences when air is introduced. Urine is ovicidal and will kill eggs in 16 hours. Eggs fail to develop in dilutions of urine down to 10 percent (Hamdy 1970*a*).

*In sewage*

Raw sewage has been reported to contain up to 38 *Ascaris* eggs per liter in eleven towns in the German Democratic Republic (Kalbe 1956); 10–80 per liter in Tokyo, Japan (Liebmann 1965); 38 per liter in San Juan, Puerto Rico (Rowan and Gram 1959); 5 to 110 per liter in Denver, USA (Wang and Dunlop 1954); and 19 per liter of settled sewage in Daspoort, South Africa (Nupen and de Villiers 1975). Wang and Dunlop (1954) reported that 87 percent of the eggs in raw sewage were viable.

Sewage from some communities in developing countries may be expected to contain much higher concentrations of *Ascaris* eggs than these. In Calcutta (India) 20,000 to 213,000 *Ascaris* eggs per capita per day were reaching the sewage works; assuming 100 liters of sewage per capita per day, this implies concentrations of 200–2,130 *Ascaris* eggs per liter (Bhaskaran and others 1956). Raw sewage in Aleppo (Syria) contained 1,000 to 8,000 *Ascaris* eggs per liter due to an estimated 42 percent of the population excreting, on average, 800,000 eggs daily per person (Bradley and Hadidy 1981).

*In sludge*

The effect of many sewage treatment plants is to concentrate *Ascaris* eggs in the sludge. In South Africa raw sludges contained 0–250 *Ascaris* eggs per gram (Keller and Hide 1951; Krige 1964). In the Johannesburg area, 74 percent of eggs in raw sludge were viable (Keller and Hide 1951). In Kharkov (USSR) raw sludge from the trickling filter plant

contained 20–48 *Ascaris* eggs per gram (Vishnevskaya 1938). Near Moscow (USSR) the sludge from trickling filter plants contained up to 466 helminth eggs per gram (Vassilkova 1936).

*Ascaris* eggs were recovered from 6 to 95 percent of sludge samples collected from several sites in the USA and were the most frequently identified parasitic helminth (Theis, Bolton and Storm 1978). Sludge from Los Angeles (California) contained up to 100 *Ascaris* eggs per gram, but most sludges contained less than 50 per gram. An earlier study (Wright, Cram and Nolan 1942) found *Ascaris* eggs in 36 percent of sludge samples from seventeen army camps in the southern USA. *Ascaris* was the most frequently identified parasitic helminth.

Sludge in developing countries may contain a much higher concentration of *Ascaris* eggs. Trickling filter plant sludge in Isfahan (Iran) contained 18,100 eggs per gram (Sadighian and others 1976), and septic tank sludge in China contained 2,300 *Ascaris* eggs per gram (McGarry and Stainforth 1978).

### *In soil*

*Ascaris* eggs are found in soil in fields where night soil or sewage are applied for fertilization or irrigation and in places used as defecation sites by infected people. *Ascaris* eggs will survive for several years in soil. The maximum recorded survival time is 7 years (Kirpichnikov 1963). Survival is promoted by cool, moist, and shaded soil and by being under the surface rather than on top. Exposure to sunlight and desiccation will reduce survival time very considerably.

Gärtner and Müting (1951*a*) studied land irrigated by sewage effluent in the Federal Republic of Germany. Samples were taken from land that had been irrigated 11 months earlier and from land irrigated only 4 to 6 days previously. There were many more *Ascaris* eggs present in the latter than in the former: in both cases the concentration diminished with increased depth. In no case did *Ascaris* eggs penetrate into the sand 0.3 meters beneath the cultivated soil. No *Ascaris* eggs were found on vegetables grown in sewage-irrigated fields. Rosenberg (1960) found that soil around a village in the USSR contained *Ascaris* eggs in 100 percent of samples; 41 percent of these eggs were viable. After cessation of fertilization with night soil, the proportion of positive soil samples fell to 35 percent, and no eggs were viable.

The survival times of *Ascaris* eggs in clean silty soil in the USSR were 23–29 days on the surface, up to 1.5 years at 0.1–0.2 meters' depth and over 2.5 years at 0.4–0.6 meters. In soil with sewage sludge, survival was for up to 1 year at 0.1–0.2 meters and up to 1.5 years at 0.4–0.6 meters (Drozdova and others 1973). In the Federal Republic of Germany *Ascaris* eggs in sewage irrigated fields survived for up to 1.5 years (Gärtner and Müting 1951*b*). In contrast, *Ascaris* eggs deposited on sewage irrigated pasture in South Africa were inactivated in a few days owing to rapid dessication (Keller and Hide 1951). The run-off water from the pasture contained no eggs. The work of Lýsek and his colleagues has suggested that fungi are instrumental in killing *Ascaris* eggs in soil, especially in the tropics (Lýsek 1964; Lýsek and Bačovský 1979).

Surface soil contamination with *Ascaris* eggs can be reduced by the use of subsurface irrigation methods. Romanenko (1969) studied subsurface irrigation with raw sewage via earthenware pipes laid at a depth of 0.6 meters, over a 9-year period. No *Ascaris* eggs were found in the surface layers of soil.

Night soil cartage systems may contaminate the soil of city streets and lanes with *Ascaris* eggs. Nineteen percent of soil samples collected from the streets of Isfahan (Iran) contained *Ascaris* eggs (Hoghooghi and others 1973). The soil contamination rate and the proportion of viable eggs were highest in winter and lowest in summer. Both of these parameters were directly related to the rainfall and inversely related to the air temperature.

### *On crops*

As mentioned previously, the contamination of vegetables by *Ascaris* eggs may be an important transmission route in some communities where family-centered transmission is relatively unimportant, owing to improved sanitation and hygiene, but where night soil or sewage are applied to vegetable gardens. The subject was reviewed 30 years ago by Rudolfs, Falk and Ragotzkie (1950, 1951*a–c*).

The contamination of vegetables by *Ascaris* eggs, following the use of sewage, night soil, or sludge for fertilization, has been a major concern of parasitologists in the USSR for many years (Khaustov 1935). Vassilkova (1941) reported that cucumbers, tomatoes, and carrots, grown outside Moscow and irrigated with sewage, were contaminated by *Ascaris* eggs and that 36 percent of these were viable. On the basis of these findings she suggested that sewage irrigation should be discontinued before harvesting and that vegetables should be gathered directly into baskets and not laid on the ground. In a later study (Vassilkova 1950), she found that tomatoes and cucumbers irrigated with raw sewage contained about 20 eggs per 100 vegetables, whereas those irrigated with

settled sewage contained about 3 per 100 vegetables.

In Lithuania, 98 percent of vegetables from gardens using raw sewage contained helminth eggs, compared with 9–16 percent of vegetables from other gardens and 7–11 percent of cleaned vegetables on sale in markets. Of all helminth eggs isolated from vegetables, 94 percent were *Ascaris*, 5 percent *Trichuris*, and 1 percent *Enterobius* (Biziulevicius 1954).

Rosenberg (1960) found that the proportion of fruit that was contaminated by *Ascaris* eggs in a village dropped from 71 percent to 25 percent following the cessation of use of untreated night soil in agriculture. No helminth eggs were detectable in samples of cucumbers, beetroot, potatoes, onions, and grass grown in fields irrigated by subsurface irrigation, whereas similar vegetables grown in fields irrigated by sewage flooding were heavily contaminated with viable *Ascaris*, *Trichuris*, and *Enterobius* eggs (Romanenko 1971).

There is less literature on this topic from countries other than the USSR, but substantial vegetable contamination may be expected where night soil or sewage is being used. In the USA, 6 percent of sewage-irrigated vegetable samples were contaminated with *Ascaris* eggs despite furrow irrigation, dry climate, sandy soil, and the fact that the sewage had undergone primary sedimentation and chlorination (Dunlop and Wang 1961).

In China and neighboring countries, where night soil is widely used for vegetable gardening, extensive *Ascaris* contamination of vegetables may be expected. Vegetable leaves in South Korea contained 38 eggs per 100 grams, and carrots had 0.6 eggs per 100 grams (Choi 1970). Early studies in northern China (Winfield and Yao 1937) suggested that vegetables were unimportant in *Ascaris* transmission. During 1933–34, 275 kilograms of vegetables from Tsinan market were examined. No eggs were found. Other vegetables were examined with the same result. Soil samples were collected from vegetable gardens, of which 57 percent were positive for *Ascaris* eggs. These data were held to substantiate the finding of Winfield (1937) that, although night soil compost was used to fertilize vegetables, contaminated vegetables were not an important transmission route. Most transmission occurred in and around the home.

In a humid environment and a shady site, *Ascaris* eggs can develop to the infective stage and survive for considerable periods on vegetables (Barchenko 1953). In an arid climate, and if exposed to sunlight, a combination of desiccation and ultraviolet irradiation causes more rapid death. Rudolfs, Falk and Ragotzkie (1951*a*) sprayed *A. suum* eggs onto growing tomatoes and lettuce under hot and dry conditions and found that they did not survive for more than 1 month.

Several studies have been conducted into methods of cleaning suspect vegetables prior to eating. In Czechoslovakia, Lýsek (1959) found that thorough rinsing removed *Ascaris* eggs but that wiping did not. In South Korea, Choi (1970) found that keeping vegetable leaves in water for 10 minutes, and then shaking them 20 times, removed only 40 percent of *Ascaris* eggs. Thitasut (1961) concluded that soaking vegetables in a solution of 100 milligrams per liter of iodine for 10 minutes would kill *Ascaris* eggs without damaging the vegetables. Zaman and Visuvalingam (1967) recommended 250 milligrams per liter of iodine for 10 minutes. Soh (1960) experimented with a wide range of pickling and food preservative substances (salt, sugar, vinegar, alcohol, bean sauce, garlic, mustard, onion, pepper, clove, allspice, cinnamon, and ginger) and found that none of them was strongly ovicidal. Rudolfs, Falk and Ragotzkie (1951*b*) concluded that immersing vegetables in warm water (60°C) for 10 minutes was the most reliable method of destroying *Ascaris* eggs.

The effect of vegetable decontamination on ascariasis was studied at a boys' school in Japan (Tomomatsu and Takeuchi 1961). *Ascaris* prevalence was originally 37 percent and fell to 5 percent following administration of a vermifuge. From that time, vegetable washing began using potassium iodide. After 5 months the prevalence had risen to 12 percent, and after 8 months to 16 percent. The authors claim that the rate of reinfection would have been greater if it had not been for the vegetable washing program, but there was no control group and they were unable to substantiate their claim.

## Inactivation by Sewage Treatment Processes

*Ascaris* egg removal in sewage treatment processes is primarily a function of the degree to which sedimentation to the sludge layer takes place.

### *By septic tanks*

In an experimental septic tank in India with 3 days retention time, 99.4 percent of *Ascaris* eggs settled. However, in operational septic tanks removal was far lower than this (Bhaskaran and others 1956).

An unpublished report from the United Nations Environment Program (UNEP 1976) described the effect on *Ascaris* eggs of the Chinese three-compartment septic tank. The retention time of each

chamber was 15–20 days, assuming a daily inflow of 2.4 liters per capita. Each unit served a group of houses, and the contents of bucket latrines were emptied into it. Studies on *Ascaris* eggs indicated that 80–96 percent were retained in the first two chambers. Eggs accumulated primarily in the sludge in the first chamber (at about 3,000 eggs per gram), where 67–95 percent of them died if stored for 2 months. A different account of the Chinese three-compartment septic tank is given in McGarry and Stainforth (1978). The retention times in the three compartments were 10, 10, and 30 days, respectively. The contents of the first compartment were semisolid. specific gravity fell and egg sedimentation improved as the liquor passed through the second and third compartments. The effluent was free of *Ascaris* eggs. The sludge in the third compartment contained 2,300 eggs per gram, and all of these were judged on morphological grounds to be dead.

*By conventional treatment*

The effect of conventional sewage treatment plants is to concentrate *Ascaris* eggs in the sludge. A few may be found in the effluent, but sometimes they are absent (for intance. see Forstner 1960). Typical *Ascaris* egg reduction rates are 35–90 percent by primary sedimentation alone, 90–99 percent by a complete trickling filter plant, and 90–100 percent by a complete activated sludge plant (see Cram 1943; Feachem and others 1980; Kabler 1959; and the studies mentioned below).

At the Kharkov (USSR) trickling filter plant, raw sewage contained on average 60 *Ascaris* eggs per liter, after primary sedimentation 20, after trickling filter 13, and after secondary sedimentation 2 per liter (Vishnevskaya 1938). At two other treatment plants in the USSR, raw sewage contained the eggs of *Ascaris*, *Trichuris*, *Enterobius*, *Diphyllobothrium*, and *Taenia* at a concentration of up to 2,000 eggs per liter. Egg concentrations were reduced by 97 percent in Imhoff tanks, by 18–26 percent in trickling filters, and by 87 percent in secondary sedimentation tanks. Chlorination of the effluent had no effect. Predictably, sludge contained many helminth eggs: up to 466 per gram (Vassilkova 1936).

At the trickling filter plant in Daspoort (South Africa), the concentrations of *Ascaris* eggs per liter were 19 in the settled sewage, 1 in the trickling filter effluent, and 1 in the final effluent from the secondary sedimentation tank (Nupen and de Villiers 1975). In Denver (Colorado. USA) raw sewage contained 5–110 *Ascaris* eggs per liter, settled sewage 2–30, and chlorinated settled sewage 0–20. The relative persistence of *Ascaris* eggs versus coliform bacteria is illustrated by the egg to coliform ratios, which were 1:14.000,000 in raw sewage, 1:10,000,000 in settled sewage, and 1:7,000 in chlorinated effluent (Wang and Dunlop 1954). No loss of viability occurred in the treatment plant, and the proportion of viable eggs was 87 percent in the influent and 88 percent in the effluent.

Three types of sewage treatment works in Puerto Rico were investigated by Rowan (1964). Primary sedimentation plants removed 35–74 percent of *Ascaris* eggs, trickling filter plants removed 95–99 percent, and activated sludge plants 97–100 percent of eggs from the effluent. Typically, small numbers of *Ascaris* eggs (up to 1 per liter) could be recovered from the effluent of secondary settling tanks following either trickling filter or activated sludge treatment. It was expected that large numbers of eggs would have been found in the sludges from these treatment plants but this was not investigated.

The survival of helminth eggs was studied in five sewage works in and around Calcutta (India) over a 4-year period (Bhaskaran and others 1956). The average number of *Ascaris* eggs per capita per day reaching the sewage works ranged from 20,000 to 213,000. Far greater numbers of *Ascaris* eggs than hookworm or *Trichuris* eggs were found. Primary sedimentation for about 2 hours caused the settlement, on average, of around 70 percent of *Ascaris* eggs. *Ascaris* eggs settled more rapidly than those of hookworm or *Trichuris*. Removal of *Ascaris* eggs from the effluent of activated sludge plants was over 90 percent and was higher when the plant was well maintained and well operated.

Panicker and Krishnamoorthi (1978) summarized *Ascaris* egg removal by various treatment plants in India. Reductions were 96 percent after 2 hours primary sedimentation, 47 percent after 1.5 hours sedimentation, 98 percent by a complete activated sludge plant. 95 and 96 percent by two complete trickling filter plants, 79 percent by a pilot scale biodisc plant, 92 percent by a pilot-scale aerated lagoon without secondary sedimentation, and 94 percent by a pilot-scale oxidation ditch with secondary sedimentation (see table 22-4).

*By waste stabilization ponds*

A well-designed series of waste stabilization ponds, with 3 or more cells and an overall retention time of at least 20 days, removes all *Ascaris* eggs from the effluent. Eggs settle to the sludge layer where they die after a few months.

In India, 100 percent removal of *Ascaris* eggs was

recorded in a three-pond system with total retention of only 6 days (Lakshminarayana and Abdulappa 1969), and Panicker and Krishnamoorthi (1978) found no *Ascaris* eggs in the effluent from four-pond systems.

Sewage in Dushanbe (Tadzhik SSR, USSR) was treated in four ponds (an anaerobic pond with area 2 hectares, depth 1.5 meters and retention time 10 hours, and three following ponds with area 4–4.5 hectares and depth 0.6 meters). The total retention time was only 33 hours. The effluent from the ponds was used to irrigate a rice paddy. Owing to the short overall retention time in the ponds, the quality of the effluent was not good (coliform count $10^6$ per 100 milliliters; $BOD_5$ 39 milligrams per liter). Inflowing sewage contained an average of 23 helminth eggs per liter, whereas the effluent contained none. Most eggs were removed in the first anaerobic pond (Koltypin 1969).

Ponds with short retention time may not remove all *Ascaris* eggs, especially during heavy rain or freezing conditions. Experiments in the Ukraine (USSR) found that two-cell ponds, with total retention of 3.6 days, let through *Ascaris* eggs in winter when the ponds froze and the increased velocities of flow along the base of the ponds picked up solids and eggs and carried them out in the effluent. An average of 0.5 *Ascaris* eggs per liter was found in the effluent in winter. A similar effect was experienced during heavy summer rain, which also increased the velocity of flow through the ponds (Ptitsyna 1966).

*By tertiary treatment*

Filtration through soil or sand should remove *Ascaris* eggs from sewage effluents. The retained eggs may develop to the infective stage and survive for many months.

Effluent chlorination is of no value in *Ascaris* removal. Chlorine and chloramine are ineffective against *Ascaris* eggs at, or greatly above, the concentrations typically applied to sewage. Low chlorine doses may even accelerate *Ascaris* egg development. (Iwańczuk and Dożańska 1957; Krishnaswami and Post 1968).

## Inactivation by Night Soil and Sludge Treatment Processes

*Ascaris* eggs tend to become concentrated in the sludge of all sewage treatment processes, and high concentrations are found in night soil. Their removal from these materials is therefore of considerable importance. Unless extreme desiccation occurs or ovicidal chemicals are added, *Ascaris* egg destruction in night soil and sludge depends almost entirely on time and temperature.

*By pit latrines*

*Ascaris* eggs in pit latrines can survive for 1–2 years, especially if conditions are cool and wet and if the latrines are sealed with a covering of soil. Available data come from temperate regions, and it is probable that survival times will be shorter in warmer climates. In tropical and subtropical areas it can be assumed that pit contents will be free of viable *Ascaris* eggs after being sealed for 1 year, and the necessary period is probably less than this if the pit is in dry soil above the water table.

In early experiments in the USA, Stiles and Crohurst (1923) found that all *Ascaris* eggs died after being buried under sawdust for 38 months. Experiments on the viability of *Ascaris* eggs in pit latrines were conducted in the USSR with the object of determining the length of storage before the excreta could be safely employed for manuring fields and kitchen gardens. Experimental pit latrines were in constant use by workers of a state farm for a prescribed period of time, after which three were covered with boards and earth, while one was left open. Samples were removed once a month. When examined immediately after removal, the eggs were invariably unsegmented—indicating that they were not developing in the pit latrines. In the open pit all eggs were dead after 6 months. In the covered pits 97 percent died after 6–8 months, and 10–13 months were required for 100 percent destruction. Egg destruction was more rapid in summer, and complete *Ascaris* elimination was obtained by sealing the pit in early spring (February–March) and reopening in November. In feces diluted with water (1:1 by volume), survival was prolonged, and 95.5 percent of the eggs perished only after 20 months (Vassilkova 1940). More recent work in the USSR found that some *Ascaris* eggs in pit latrines survived for over 18 months and outlived *Trichuris* eggs (Biziulevicius 1965).

*By biogas plants*

The removal of *Ascaris* eggs in biogas plants depends on the retention time and the degree to which the design prevents short-circuiting of flow. Chinese data suggest that 2–7 percent of influent *Ascaris* eggs will appear in the effluent (Hou and others 1959; McGarry and Stainforth 1978; Szechwan Research Institute 1974). The eggs removed settle to the sludge

layer, where they remain viable for many months. Studies in Szechwan showed that adding small amounts of waste frequently, improving design to reduce short-circuiting, and increasing the organic content of the influent all reduced the egg concentration in the effluent.

*By digestion*

Mesophilic digestion, or digestion at cooler temperatures, does not greatly reduce the concentration of *Ascaris* eggs in sludge (Kabler 1959). *Ascaris* eggs have been frequently isolated from digested sludge, for instance in the USA (Cram 1943; Fitzgerald 1981) and the USSR (Vassilkova 1936), and digestion periods far longer than normally employed are required before a significant *Ascaris* reduction can be obtained.

Experiments in India showed that 40 percent of *Ascaris* eggs held at 36°C for 50 days were still viable, and a few viable eggs could still be isolated after 150 days (Bhaskaran and others 1956). *A. suum* eggs were held for 21 days at 38°C in sludge from various sewage treatment works in the Chicago area (USA). With the exception of one sample, at least 46 percent of the eggs remained viable, and some were then found to be infective to pigs (Fitzgerald and Ashley 1977).

Cram and Hicks (1944) reported that 10 percent of *Ascaris* eggs in digesting sludge were viable after 6 months, and some were still viable after 1 year. Eggs previously embryonated appeared to be more susceptible to subsequent anaerobic digestion than undeveloped eggs. In Johannesburg (South Africa) raw sludges contained between 50 and 243 *Ascaris* eggs per gram, whereas digested sludges contained 105–552 per gram. The effect of sludge digestion in concentrating eggs was noted. However, 74 percent of eggs in raw sludge were viable compared with only 39 percent viable in digested sludge (Keller and Hide 1951). At Kouřim (Czechoslovakia) up to 100 percent of *Ascaris* eggs in raw sludge were viable, whereas only 58 percent in digested sludge were viable (Králová and Šafránek 1957).

Under anaerobic conditions eggs tend to stay alive but not develop. Under aerobic conditions and temperatures above 20°C, eggs develop and may become infective. Reyes, Krusé and Batson (1963) reported studies on aerobic and anaerobic batch digestions of night soil seeded with *A. suum* eggs. At low temperatures, both aerobic and anaerobic digestion tended to preserve eggs in a viable condition. In the 25°C–35°C range, both systems resulted in some egg destruction attributable to factors other than heat. Oxygen starvation in anaerobic digestion and spontaneous hatching in aerobic digestion may have contributed to egg destruction. At 30°C, viability was better maintained when eggs were kept in night soil than when they were kept in water. Complete egg destruction was not possible unless temperatures were maintained above 45°C in aerobic processes and 38°C in anaerobic processes.

*A. suum* eggs in swine wastes (2 volumes of pig feces plus 1 volume of pig urine plus 17–32 volumes of water) readily survived aerobic and anaerobic digestion for 68 days at 12°C (over 90 percent remained viable). At 22°C over 47 days, under aerobic conditions 89 percent of eggs developed to the infective stage, whereas under anaerobic conditions eggs did not develop but remained alive (Marti, Booram, and Hale 1980).

Thermophilic digestion, like all other processes involving raised temperatures (>45°C), can be highly effective in *Ascaris* egg destruction (figure 23-2). Burden and Ginnivan (1978) studied seeded *A. suum* eggs in pig slurry (95 percent water) under aerobic thermophilic digestion at 55°C. Ninety percent of unembryonated *A. suum* eggs were dead (judged by failure to embryonate in culture) after 15 minutes, and all were dead after 30 minutes. Embryonated eggs were all dead (judged by failure to infect piglets) after 15 minutes.

*By storage*

Storage of sludge at ambient temperatures will eliminate *Ascaris* eggs if the storage period is long enough. Nine months were not sufficient in Pretoria, South Africa (Murray 1960), no loss of viability occurred after 44 days in pulverized sludge in the USA (Cram and Hicks 1944), and 2 years were required for complete elimination of *Ascaris* eggs in Moscow, USSR (Vassilkova 1936). *A. suum* eggs stored in silage for up to 4 months were still infective to mice, but eggs stored for 5 months were not (Pavlov 1958).

If storage is accompanied by drying, death will be more rapid, but survival times of several months are still possible. In laboratory experiments in India, sludge samples were kept in open dishes exposed to diffused sunlight. After 51 days, the moisture content had dropped to 3.1 percent, and yet 10 percent of eggs were still viable (Bhaskaran and others 1956).

Survival of *Ascaris* eggs in night soil is more limited than in sludge if the night soil contains urine. Tests in Japan on the viability of *Ascaris* eggs in night soil (1 volume feces to 5 volumes urine) indicated that the minimum storage temperatures and times needed for complete kill were 30°C for 40 days, 32°C for 25 days, or 34°C for 11 days (Nishi 1969).

Storage will be more effective in destroying *Ascaris* eggs in warm or dry climates than in cool or wet climates.

*By drying*

Desiccation is antagonistic to *Ascaris* eggs. At moisture contents below about 5 percent, death may be fairly rapid, especially at warm temperatures (Keller 1951*a*). These low moisture contents are never achieved in normal sludge drying practice, and a typical "dewatered" sludge contains about 75 percent water. Therefore, sludge drying processes are analogous to sludge storage (see above) in their effects upon *Ascaris* eggs, and long holding times are necessary to eliminate *Ascaris* eggs. Required holding times will be somewhat less in warm climates than in temperate regions.

After 4 years on drying beds at Kharkov (USSR), sludge still contained 18 *Ascaris* eggs per gram near the surface and 7 eggs per gram at a depth of 1 meter (Vishnevskaya 1938). When sludge was dried in the sun in South Africa for 4 months in layers ranging in thickness from 37 to 150 millimeters, *Ascaris* eggs were completely eliminated from the 37-millimeter layer, in which the moisture content had fallen from 84 percent to below 3 percent. *Ascaris* eggs still remained in the thicker layers (Hogg 1950). In other experiments, *Ascaris* survived drying to a point where the moisture content of the sludge reached 5.8 percent but failed to survive when the moisture content reached a lower figure (Cram and Hicks 1944).

*By heating*

The only method of achieving *Ascaris* egg elimination, without prolonged storage or adding ovicides, is by heating. The literature contains many studies into the time-temperature survival of eggs under different environmental conditions. These studies are sometimes contradictory. All relevant data found in the literature have therefore been plotted on figure 23-2, and an upper boundary line has been drawn. Temperatures and times above this line should guarantee complete destruction of *Ascaris* eggs.

It is clear from figure 23-2 that temperatures of over 45°C must be reached, and preferably over 50°C. The practical way of achieving this is aerobic thermophilic composting of sludge or night soil mixed with organic refuse. Other, somewhat impractical methods have been tried.

In a village in Shiga Prefecture in Japan, a sheet-iron container with a capacity of 14 liters was installed under the seat of each domestic privy (Katayama

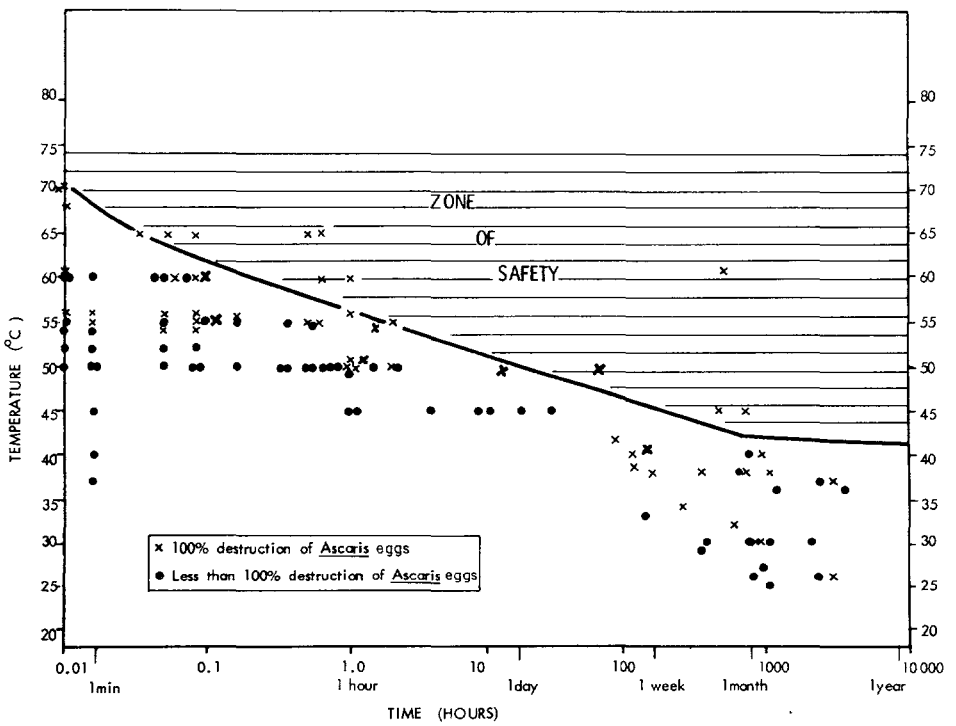


Figure 23-2. *The influence of time and temperature on* Ascaris *eggs.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

1955). The contents were emptied occasionally into a 200-liter drum and heated to 60°C with firewood. All parasite eggs and fly maggots were completely destroyed. The night soil was then used for fertilization. The effect was demonstrated by a decline in the prevalence of *Ascaris* and hookworm infections as well as by a decrease in the count of embryonated *Ascaris* eggs found in the farm soil of the village, compared with a nearby control village.

*By composting*

Aerobic composting of night soil or sludge with refuse, woodchips, straw or other carbonaceous bulking material is an efficient method of eliminating *Ascaris* eggs. Success depends on careful process management and, in particular, on regulation of the moisture content, the carbon to nitrogen ratio, and the pile temperature. It is pile temperature that is crucial to *Ascaris* egg elimination, and the required time-temperature combinations may be read off figure 23-2. To achieve these time-temperature conditions throughout the composting mass requires lagging by covering the heap with old compost or mud, forced draft aeration, regular turning, or a combination of these.

Studies on *Ascaris* eggs in composting processes are listed in table 23-3. *Ascaris* eggs are the most hardy of all excreted pathogens considered in this book. The time-temperature requirements for complete inactivation are more stringent than for other pathogens, with the exception of enteroviruses at short retention times (see figure 9-2). For this reason, and because viruses are technically difficult to enumerate in compost samples, *Ascaris* eggs make an excellent indicator of compost quality. *Ascaris* egg standards for compost have been adopted in China and Vietnam. Where facilities are excellent, a combined enterovirus-*Salmonella*-*Ascaris* standard is appropriate. Where laboratory facilities are more limited, a *Salmonella*-*Ascaris* or fecal streptococci-*Ascaris* standard should be adopted. Where laboratory facilities are poor, an *Ascaris* standard alone will prove adequate.

Table 23-3. *Some studies on* Ascaris *eggs in composting processes*

| *Country* | *Source* |
|---|---|
| China | Department of Environmental Health (1975)<br>Hou and others (1959)<br>McGarry and Stainforth (1978)<br>Scott (1952, 1953) |
| Germany Democratic Republic | Borchert and Kalbe (1955)<br>Kalbe (1955) |
| India | Bhaskaran and others (1957) |
| Malaysia | Scharff (1940) |
| Poland | Iwańczuk (1963) |
| South Africa | Keller (1951*b*, 1951*c*)<br>Krige (1964)<br>Murray (1960)<br>Steer and Windt (1978) |
| Sri Lanka | Nicholls and Gunawardana (1939) |
| USA | Brandon (1978)<br>Theis, Bolton and Storm (1978)<br>Wiley and Westerberg (1969) |
| USSR | Biziulevicius (1961)<br>Gudzhabidze and Lyubchenko (1959) |
| Reviews of several countries | Feachem and others (1980)<br>Hays (1977)<br>Petrik (1954)<br>Wiley (1962) |

*By other processes*

A variety of more technically complex sludge treatment processes is available. Those that involve heating may be highly effective; those that do not will not. The two exceptions to this are the use of chemical ovicides and irradiation.

CHEMICAL OVICIDES: An alternative to heating is to treat night soil or sludge with an ovicidal chemical. A great variety of chemicals have been tried in the field and in the laboratory. Two field trials in Japan, one of sodium nitrite in acidified night soil and one of thiabendazole, have shown that night soil treatment reduced ascariasis prevalence (Kozai 1960*a*; Kutsumi 1969). The literature on chemical ovicides is listed in table 23-4. A wide variety of chemicals have effect. Sodium nitrite and thiabendazole are the most widely tried. The practicability and cost of this method must be questioned, however. Thermophilic composting of night soil or sludge with refuse is a more realistic, and probably cheaper, method of making fecal products safe for land application. An added advantage of thermophilic composting is that it will destroy all other pathogens in the compost, which has not been claimed for the application of chemical ovicides.

IRRADIATION. Data on the effect of radiation on *Ascaris* eggs in sludge are limited. Brandon (1978) reported that 1.5 kilogray killed over 99 percent of *Ascaris* eggs. Lessel and Suess (1978) found a 100 percent kill at a dose of 3 kilogray, whereas Osborn

Table 23-4. *Some literature on* Ascaris *ovicides*

| *Ovicide* | *Media in which eggs contained* | *Source* |
|---|---|---|
| Benzylphenol | Water | Nasiłowska (1963) |
| Carbathion | Dried sludge | Chilikin (1975) |
| Carbon disulphide | Cesspool contents | Matsumura and Osawa (1954) |
| Chlorobenzyl-phenol | Water | Nasiłowska (1963) |
| Coca Cola | ND | Bacev, Kolev and Peeva (1972) |
| Creolin | Water | Šimůnek, Krč and Svoboda (1963) |
| Cresol | Water | Fujita (1959) |
| | Cesspool contents | Matsumura and Osawa (1954) |
| Detergents | Water | Jaskoski (1951) |
| | Vegetables | Kumada (1965*a*; 1965*b*) |
| Dicapthon (isochloro-thion) | Night soil | Fujita (1960*a*) |
| | Water | Fujita (1960*b*) |
| Fertilizers | Water | Hamdy (1969, 1970*b*) |
| Hexachloro-phenol | Water | Nasiłowska (1963) |
| Nitric acid | ND | Hsu and Hsu (1940) |
| *p*-Thiocresol | Cesspool contents | Matsumura and Osawa (1954) |
| Pentachloro-phenol | Water | Fujita (1959) |
| Phenol | ND | Hsu and Hsu (1940) |
| | Water | Šimůnek, Krč and Svoboda (1963) |
| | Cesspool contents | Matsumura and Osawa (1954) |
| Proteins | Night soil | Matsumura and Oda (1954) |
| Sodium nitrite | Pickle | Kim and Yoon (1966) |
| | Acidified night soil | Kozai (1960*a*, 1960*b*, 1960*c*, 1962) |
| | Acidified night soil | Kozai and Kobayashi (1961) |
| Sodium penta-chloro-phenate | Water, acidified night soil | Kutsumi (1963) |
| Sulphuric acid | ND | Hsu and Hsu (1940) |
| Thiabendazole | Pickle | Kim and Yoon (1966) |
| | Night soil | Kutsumi (1964*a*, 1964*b*, 1969); Kutsumi and Komiya (1965) |
| Xylene | Cesspool contents | Matsumura and Osawa (1954) |

ND No data.

and Hattingh (1978) reported 99 percent inactivation at 1 kilogray but less than 100 percent inactivation at 5 kilogray. It appears that the effect of radiation on *Ascaris* eggs lies somewhere between the effect on bacteria (1 log reduction per 200–300 gray), and the effect on enteroviruses (1 log reduction per 2–5 kilogray).

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# 24

# *Clonorchis* and Clonorchiasis

THE WORM *Clonorchis*, and the closely related *Opisthorchis*, follow a classic trematode life cycle from vertebrate hosts through snails and fish and back to the vertebrate hosts. These worms are of special importance in the design of sanitation systems that involve the use of human or animal excreta in aquaculture.

## Description of Pathogen and Disease

*Clonorchis* and *Opisthorchis* worms are found mainly in East Europe and Eastern Asia and are not well known to public health workers in Western Europe, North or South America, or Africa. Most of the literature on these pathogens is not in English, but an important English-language review was published by Komiya (1966).

### *Identification*

Chonorchiasis (or opisthorchiasis) is an infection of the bile ducts by trematodes of the genus *Clonorchis* (or *Opisthorchis*). In light infections, symptoms are likely to be absent or vague. Heavier infections result in diarrhea, abdominal discomfort, and some splenomegaly. Heavy parasite burdens may cause acute pain in the upper right quadrant, liver enlargement and tenderness, edema, an increased erythrocyte count, raised sedimentation rate, and up to 40 percent eosinophilia. There may be bouts of recurrent gall bladder colic. Carcinoma of the bile ducts is a lethal complication (Sonakul and others 1978), and death can also ensue from secondary bacterial infection. Clonorchiasis is a chronic disease, sometimes lasting for 30 years or longer (see, for instance, Attwood and Chou 1978).

Diagnosis is by examining feces microscopically for eggs. Treatment is by oral drug therapy but, although a large variety of drugs have been tried, they are either not freely available, too toxic, or not effective (see, for instance, Jopling 1978). Praziquantel is under trial for clonorchiasis therapy and shows promise. At present, no treatment is advised for those with asymptomatic clonorchiasis.

### *Occurrence*

*Clonorchis sinensis* occurs in China, Hong Kong, Korea, Taiwan, Japan, and Vietnam (figure 24-1). *Opisthorchis viverrini* occurs in Thailand and southern Laos. *O. felineus* occurs in Poland, various parts of the USSR (especially the Ukraine), and northern Turkey.

### *Infectious agents*

Three closely related trematode species are considered in this chapter: first *C. sinensis*, the Chinese liverfluke; second *O. viverrini*; and third *O. felineus*, the cat liver fluke. *C. sinensis* is also called *O. sinensis*, it being argued that it is not sufficiently different from *O. viverrini* and *O. felineus* to warrant a separate genus. The three species are very similar in their biology but occur in different parts of the world.

The adult worms are flat, transparent, flabby, hermaphroditic organisms that measure 11–20 by 3–5 millimeters (figure 24-2). The eggs are relatively small, measuring 23–35 by 10–20 micrometers.

### *Reservoirs*

A wide variety of fish-eating vertebrates provide the definitive hosts for these trematodes. For *C. sinensis* the main hosts are man and dogs, for *O. viverrini* cats and man, and for *O. felineus* cats. Transmission of *O. viverrini* and *O. felineus* is often maintained in the absence of man, and this is also possible for *C. sinensis*.

### *Transmission*

There can be up to 6,000 worms in the bile ducts, and each worm produces 2,000–4,000 eggs per day. The

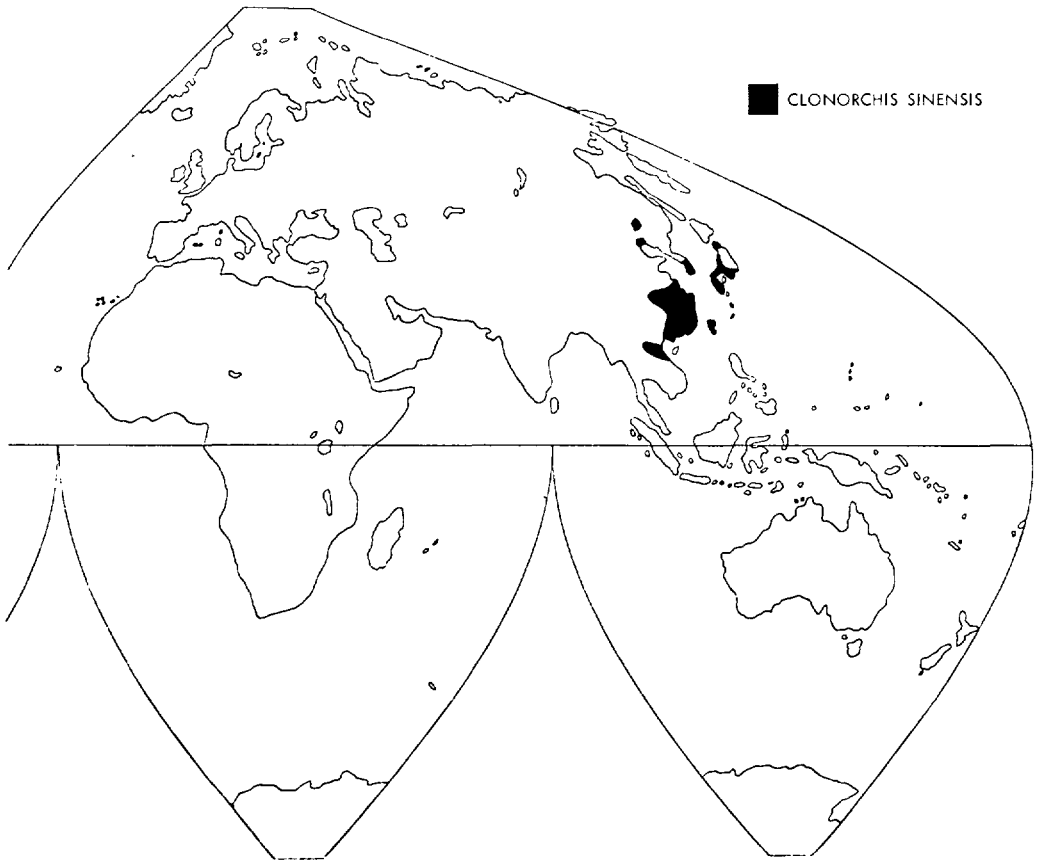


Figure 24-1. *Known geographical distribution of* Clonorchis sinensis. The infection may occur in areas as yet unrecorded

number of eggs produced by *C. sinensis* varies with the species of host and with the duration of the infection.

The eggs, containing fully formed larvae (miracidia), are passed out in the feces and for further development have to reach water and be ingested by particular species of freshwater snails. For *C. sinensis*, snail hosts include *Bulinus fuchsianus* in northern China, *Alocima longicornis* in southern China, and *Parafossarulus manchouricus* in most endemic areas. For *O. viverrini*, snail hosts include *Bithynia funiculata*, *B. laevis*, *B. goniomphalus*, and *B. siamensis*. For *O. felineus*, the host is usually *B. leachii*.

Asexual multiplication occurs in the snail, and many free-swimming cercarial larvae are released 3–4 weeks after ingestion. These live for only 1–2 days in water unless they can penetrate beneath the scales of almost any species of freshwater fish and form cysts (metacercariae) in the connective tissues. Over eighty species of freshwater fish have been incriminated as hosts of *C. sinensis*, and a maximum of 3,000 metacercariae have been found in a single fish. *O. felineus* commonly encysts in carp.

When fish are eaten raw or undercooked by man or other vertebrate host, the larvae hatch out in the duodenum and migrate up the bile ducts. Adult worms can live for 25–50 years. From each cyst ingested, one adult worm may develop and is capable of producing eggs.

### *Prepatent and incubation periods*

Worms reach maturity and start producing eggs 3–4 weeks after the ingestion of cysts. Symptoms may develop slowly or not at all. Acute clonorchiasis with an incubation period of only 10 days has been reported from China (Zhipiao, Huilan and Weiji 1979).

### *Period of communicability*

As long as mature adult worms are present, eggs may be passed in the feces. Adult worms can live for up to 50 years. Eggs can live for 1 month in water. Metacercariae in fish can survive for the life of the fish and for some weeks after its death.

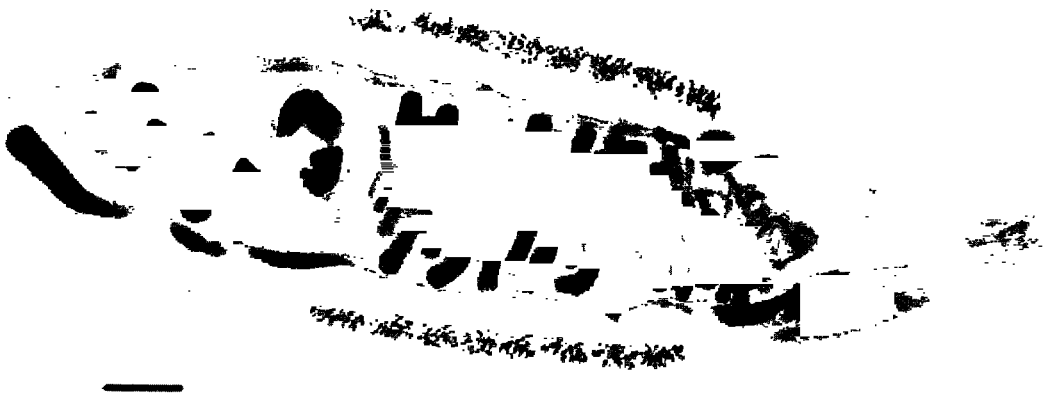

Figure 24-2. *An adult of* Opisthorchis felineus *under a light microscope.* Scale bar = 1 millimeter. (Photo: Wellcome Museum of Medical Science)

*Resistance*

There is no clear evidence for the development of immunity.

*Epidemiology*

Infection is normally contracted from eating raw or undercooked fish. The metacercarial cysts are sticky, however, and can be transferred from knives, hands, or chopping boards to other foods or directly to the mouth. Infection is much more common in adults than children and is rare in young children. It is common for adult males to have higher prevalences of clonorchiasis than adult females owing to different dietary customs. In South Korea, for instance, men eat raw fish at rice-wine drinking parties.

Transmission of *C. sinensis* is especially associated with raising fish in excreta-enriched ponds, a practice common in China and elsewhere in East Asia and one that clearly creates ideal conditions for transmission, provided that the ponds are colonized by the appropriate snail species (Faust and Khaw 1927). Infection with *O. felineus* in Poland and the USSR is especially associated with eating recently salted fish.

Clonorchiasis in Taiwan has increased in recent years (Chen and others 1980). Prevalences are up to 52 percent in school staff in some areas. Pigs are commonly infected, and up to 100 percent of some fish species in some localities harbor *Clonorchis* metacercariae. Ong and Lu (1979) studied clonorchiasis in a highly endemic area of Taiwan and found prevalences of 3 percent among primary-schoolchildren, 7 percent of middle-school children, 8 percent of high-school children, and 56 percent of government workers. Prevalences were higher among males than females and higher among those 35–45 years old than among others.

A survey in South Korea during 1967–69 revealed a nationwide prevalence of clonorchiasis of 5 percent, with prevalences rising to 15 percent locally. Prevalence and intensity of infection were higher in males than females and higher among those over 30 years old than in younger persons (Seo and others 1969).

Opisthorchiasis is extremely common in northern and northeastern Thailand. Prevalences range from 10 to 90 percent, and about 4 million people are infected (Harinasuta 1980). In the worst affected areas between 50 and 90 percent of carp are infected and harbor 20–50 metacercariae each. Laotian refugees in Thailand had an opisthorchiasis prevalence of 44 percent, with a peak age-prevalence of 64 percent among 30–34 year olds (Temcharoen and others 1979).

Other surveys of clonorchiasis or opisthorchiasis include those from China (Weng and others 1960), Japan (Yokogawa 1969), Malaysia (Rohde 1967), South Korea (Choi and others 1973), Taiwan (Cross 1969), Thailand (Viranuvatti and Stitnimankarn 1972), and the USSR (Churina 1973; Pantyukhov 1965).

## Control Measures

There is no effective and safe drug for mass chemotherapy at present. Praziquantel may well fulfil this role and is undergoing clinical trials. Personal protection is best achieved by not eating raw or partially cooked fish.

Snail control using chemicals is difficult because of the toxic effect that most molluscicides have on fish. Some workers have tried biological control using fish and crayfish, but this requires further study (Nagano 1964). Clearing aquatic vegetation from fishponds may reduce snail populations (Komiya 1966).

Viable eggs in feces must be prevented from reaching bodies of freshwater in which the snail and fish intermediate hosts live. This would be relatively straightforward except for two obstacles. First, in parts of the world where clonorchiasis is endemic, especially China and Taiwan, it is the practice to enrich fishponds with human and other feces in order to improve productivity. Second, there are several reservoir hosts apart from man, so that the control of human feces alone can only have a partial effect on transmission. The solution to the first problem is to treat excreta prior to adding to fish ponds. There is no simple solution to the second problem.

Education is needed to make the population aware of the cause of clonorchiasis and, in endemic areas, to try to change the habit of eating raw or insufficiently cooked fish.

## Occurrence and Survival in the Environment

The eggs, miracidia and cercariae of *Clonorchis* and *Opisthorchis* may be found in waters contaminated by feces of animals or man in endemic areas. A few reports exist; for instance, *Opisthorchis* eggs were isolated from river water in the USSR (Goryachev 1947).

Early experiments by Faust and Khaw (1927) showed that *Clonorchis* eggs in an isotonic solution survived for up to 3 months at 2–4°C, 6 months at 4–8°C, 1 month at 26°C, 3 weeks at 37°C, 4 days at 45°C, 1 hour at 50°C, and a few minutes at 58°C. At 6–8°C survival times were 4–5 days in fresh night soil, 2 days in 5-day-old night soil, and 1 hour in 10-day-old night soil. *Clonorchis* eggs were rapidly killed by desiccation. In river water *O. felineus* eggs survived for over 160 days at 0–5°C, and this suggests that they may overwinter in the USSR (Drozdov 1962).

*Clonorchis* metacercariae in fish persist throughout the life of the fish. Thereafter they survive for months if frozen, for several days if salted, for over 2 weeks if dried, and for several days in soy sauce, vinegar, or wine (Faust and Khaw 1927; Wykoff 1959). They are killed by thorough cooking.

*O. felineus* metacercariae were killed rapidly at 55–58°C, but in fish of 1–2 kilograms weight they were killed by cooking for not less than 1 hour (Mitrokhin 1962). *O. felineus* metacercariae were still infective to foxes after 7 days drying in fish, but not after 9 days (Mitrokhin 1960). Other Soviet workers have recommended that carp should be soaked in brine for 2–3 weeks and then dried for at least 3 weeks to kill *O. felineus* metacercariae (Yaldygina and others 1970).

## Inactivation by Sewage Treatment Processes

No information on *Clonorchis* or *Opisthorchis* eggs during sewage treatment could be located. The nearest parallel is probably that of schistosome eggs (chapter 32).

## Inactivation by Night Soil and Sludge Treatment Processes

No specific data are available but *Clonorchis* eggs are rapidly killed by storage in night soil (Faust and Khaw 1927; Komiya 1966). *Clonorchis* eggs should be destroyed after storage for 5 days at 5°C or for 2 days at 25°C.

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# 25

# *Diphyllobothrium* and Diphyllobothriasis

DIPHYLLOBOTHRIASIS has a restricted geographical distribution and is not a major public health problem in most areas where it does occur. It is caused by a cestode (tapeworm) but has a life cycle involving two aquatic hosts that is more reminiscent of the life cycles of trematodes.

## Description of Pathogen and Disease

There is a very extensive Russian literature on diphyllobothriasis. Only a brief summary of the disease is given in this chapter. The disease has been comprehensively reviewed by von Bonsdorff (1977).

### *Identification*

Diphyllobothriasis is an infection of the small intestine by the broad fish tapeworm *Diphyllobothrium latum*. There are often no clinical symptoms associated with infection, apart from eosinophilia. However, in a proportion of cases there is abdominal pain, loss of weight, anorexia, and vomiting. Megaloblastic anemia occurs in 20 percent of cases in Finland.

Diagnosis is by finding eggs, or occasionally segments, in the feces. Drug therapy is with niclosamide, or any other agent effective against *Taenia* (see chapter 34).

### *Occurrence*

Diphyllobothriasis occurs in temperate countries with many lakes: in Europe, mainly in Finland, the USSR, and Poland with sporadic cases in France, Ireland, Italy, Switzerland, and the Federal Republic of Germany; in Asia, in Japan and Siberia; in the Americas, in the Great Lakes region of Canada and the USA, among Eskimos, and in Chile and Argentina (figure 25-1). It has also been reported from lakeside regions in Africa. Where raw or partly cooked fish is eaten, prevalence may be 10–30 percent locally, and generally increases with age.

### *Infectious agent*

*Diphyllobothrium latum*, a cestode, is the broad fish tapeworm of man. The hermaphroditic adult measures 3–10 meters in length and may have 4,000 segments, with a small scolex, which has no hooks, embedded in the mucosa of the ileum (figure 25-2). Immediately behind the scolex, and several times its length, is an unsegmented neck region. The neck is followed by newly formed proglottids that become mature. The proglottids measure 2–7 by 10–12 millimeters and contain both male and female reproductive organs. Eggs are evacuated periodically through a uterine pore on each functional proglottid. The eggs measure 55–80 by 40–60 micrometers.

### *Reservoir*

Man is the most important reservoir. Dogs, bears, and other fish-eating mammals may also become infected, but the proportion of viable eggs in dog feces (1 percent) is much less than in human feces (79 percent) (Essex and Magath 1931).

Related tapeworms of nonhuman mammals, that have intermediate stages in fish, also infect man occasionally. Examples include a tapeworm of the fur seal, *Diphyllobothrium pacificum*, in Peru; a tapeworm of the gull, *D. dentricum*, in Siberia; and a tapeworm of the whale, *Diplogonoporus grandis*, in Japan. Man may also act as the intermediate host for tapeworms of the genus *Spirometra*. Adult *Spirometra* live in the intestine of carnivores (but not man), and the intermediate hosts are, first, a cyclops in water and, second, amphibia, reptiles, or mammals, including man.

### *Transmission*

Each worm produces up to 1 million unsegmented eggs daily, that pass out in the feces. If an egg reaches

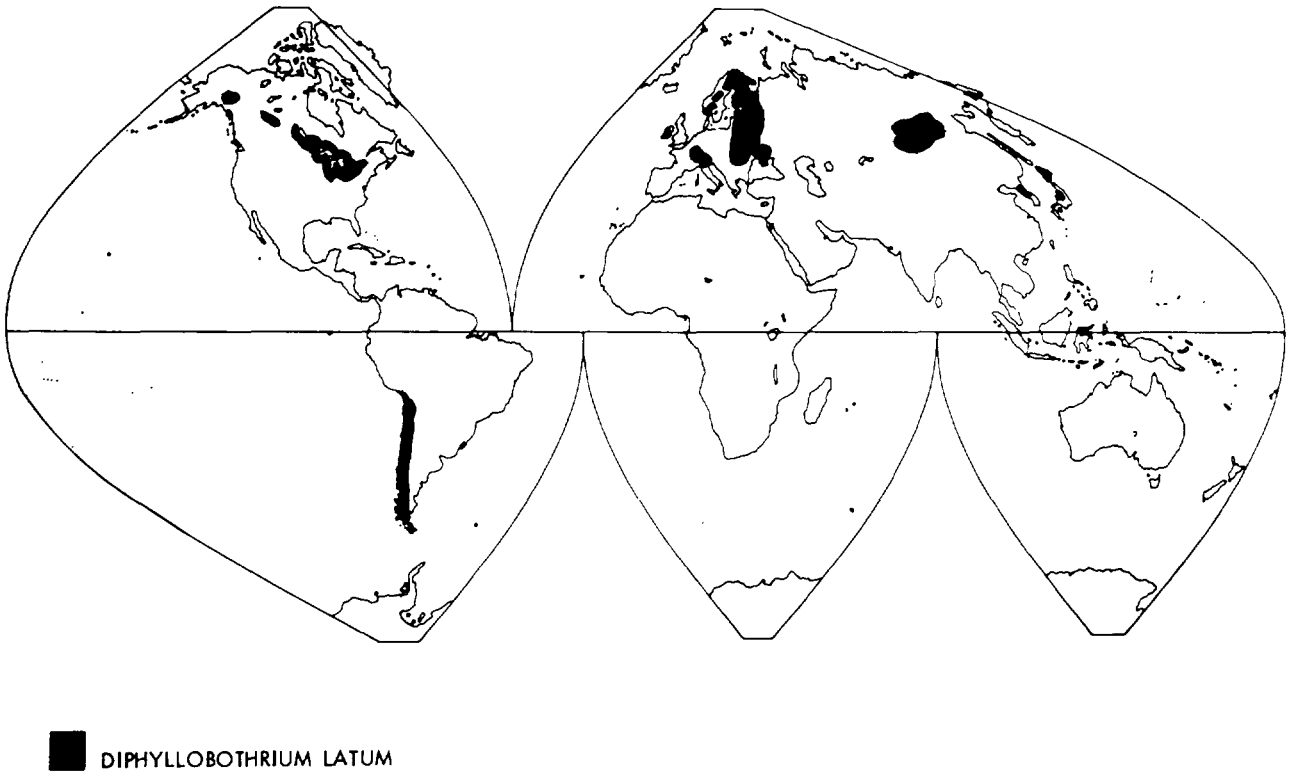


Figure 25-1. *Known geographical distribution of* Diphyllobothrium latum. The infection may occur in areas as yet unrecorded

Figure 25-2. *A length of* D. latum *expelled after treatment.* (Photo: Wellcome Museum of Medical Science)

fresh water it develops—in 12 days to many weeks, depending on the temperature—into a ciliated larva (coracidium).

The coracidium escapes into the water and swims around, surviving for 1–2 days. For further development the coracidium must be ingested by a minute freshwater copepod (of the genera *Diaptomus* or *Cyclops*). Inside the copepod there is further development of the larva, which may take 2–3 weeks. Freshwater fish (pike, perch, salmon, burbot) act as second intermediate hosts. The freshwater fish ingest the copepod, and the larva present in the infected copepod works its way through the fish tissue to the muscles, where it grows into a plerocercoid larva over about 4 weeks. When an infected fish is eaten raw by man, the larva is released in the small intestine, grows into a mature tapeworm in 3–6 weeks, and can live for up to 25 years.

One larva ingested in fish may develop into the hermaphrodite worm, which is sufficient to maintain the infection.

### *Prepatent and incubation periods*

Worms reach maturity about 5–6 weeks after infective larvae in fish are ingested. Symptoms develop slowly or not at all.

### *Period of communicability*

Eggs are passed in the feces as long as mature worms are present. Adult worms may live up to 25 years (Leiper 1936). Larvae in fish are infective to man for the life of the fish and for some time thereafter.

### *Resistance*

There is no evidence of innate or acquired resistance to infection.

### *Epidemiology*

Raw or smoked fish is the main source of infection. Pike roe (caviar) and pike spawn are also major sources of infection in the USSR (Karaseva and Egorova 1965). The age and sex distribution of infection is related to dietary habits. Diphyllobothriasis is mainly an infection of adults.

Chefranova (1964) studied the epidemiology of diphyllobothriasis in the Evenk National District (USSR). The prevalence rate of diphyllobothriasis was 69 percent. The infection rates of fish harboring larvae of *Diphyllobothrium* were: *Coregonus peles*, 82 percent; *Esox lucius*, 11 percent; *Lota lota*, 10 percent; *C. lavaratus*, 4 percent; and *Perca fluviatilis*, 4 percent. No *Diphyllobothrium* were found at autopsy of sables, gluttons, polar foxes, and wolves. One of twenty-one dogs examined was infected.

Other accounts of diphyllobothriasis include those from Canada (Turgeon 1974), Finland (Wikström 1972), Japan (Tomita and others 1979; Uhari and others 1975), Peru (Baer and others 1967), and the USSR (Artamoshin 1968, 1972).

## Control Measures

Mass chemotherapy with niclosamide, combined with health education measures, has markedly reduced prevalence locally. Thorough cooking, freezing, or salting of fish will kill larvae. Preventing untreated human feces from reaching freshwater will greatly reduce transmission.

Successful integrated control campaigns in the USSR have been reported from the Danube Delta (Smolinschi and others 1970) and the Astrakhan River (Epstein and others 1967).

## Occurrence and Survival in the Environment

*Diphyllobothrium* eggs may be found in fecally contaminated waters in endemic areas. They have been isolated from river water and sediment (Goryachev 1947; Usacheva 1951) and from sewage (Vassilkova 1936, 1941) in the USSR.

*Diphyllobothrium* eggs in freshwater at 15–25°C develop within 11–15 days. The lower the temperature, the slower the development. Eggs are killed after 2 days at −10°C or 30 days at 2–6°C (Essex and Magath 1931; Fedorov 1956). The minimum concentration of oxygen in water needed for eggs to hatch into coracidia is 1.4 milligrams per liter at 24°C (Romanov 1972). At lower oxygen levels eggs can survive for many months but will not develop unless transferred to a more oxygenated environment (Fedorov 1956). Eggs in water at depths of over about 20 meters do not hatch (Razumova and Artamoshin 1969). Eggs are rapidly killed by desiccation (Essex and Magath 1931).

Eggs in feces on the ground, or on ice in winter, die within 3 days (Chefranova 1964).

The encysted plerocercoid larvae in fish muscle and viscera live for the life of the fish and for some time after. The larvae survive in dead fish in river water for up to 10 days (Pronin 1967). The larvae can be killed by

freezing, salting, or cooking, but each of these operations must be very thorough to be effective. Studies on infected pike (Titova 1955) showed that effective freezing regimes were: at −6°C, 7 days with a 9-kilogram fish, 6 days with a 2-kilogram fish, or 3 days with a 0.7-kilogram fish. At −18°C, larvae were destroyed after 4 days in 2-kilogram fish and 2 days in 0.5-kilogram fish. Salting and cooking also need to be carried out for longer in large fish than in small fish to destroy larvae.

## Inactivation by Sewage Treatment Processes

Little is known about *Diphyllobothrium* eggs in sewage treatment plants. Sedimentation will remove a high proportion to the sludge layer and will be more effective if a coagulant is used (Döschl 1972). In the absence of specific data, it may be assumed that *Diphyllobothrium* eggs react to sewage treatment in the same manner as *Ascaris* eggs (chapter 23).

## Inactivation by Night Soil and Sludge Treatment Processes

Any process effective against *Ascaris* eggs (chapter 23) will be highly effective against *Diphyllobothrium* eggs.

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# 26

# *Enterobius* and Enterobiasis

FIVE INTESTINAL NEMATODE INFECTIONS—ancylostomiasis (chapter 22), ascariasis (chapter 23), strongyloidiasis (chapter 33), trichuriasis (chapter 35), and enterobiasis (this chapter)—are described in this book. Enterobiasis is atypical within this group because it is transmissable directly from one person to another without the need for a period of development in soil elsewhere, and because the *Enterobius* eggs are not normally excreted in the feces. Although enterobiasis is extremely common—the commonest in fact of all infections discussed in this book—it is of very minor public health importance.

## Description of Pathogen and Disease

The literature on enterobiasis is limited because it is an infection that does not commonly cause serious disease.

### *Identification*

Enterobiasis is an infection of the large intestine and appendix by the nematode *Enterobius vermicularis*. The heads of the worms are attached to the mucosa of the intestinal wall. There are usually only minor symptoms or none at all. Pruritus ani, causing disturbed sleep, is common, and there is sometimes mild catarrhal inflammation with nausea and diarrhea. Symptoms of appendicitis are a very rare occurrence. Migration of worms to the female genitalia frequently occurs. Diagnosis is by finding eggs on the perianal skin by means of sticky tape. Enterobiasis is treated by oral drug therapy with mebendazole, pyrantel pamoate, or piperazine citrate.

### *Occurrence*

Enterobiasis occurs worldwide and is extremely common, particularly in children. There are probably over 1,000 million cases in the world. It is likely that virtually every person living in a temperate country is infected some time during childhood.

### *Infectious agent*

*E. vermicularis*, a nematode, is the pinworm, threadworm, or seatworm of man. It is also known as *Oxyuris vermicularis*. The female worm measures 8–13 millimeters and contains about 10,000 eggs; and the male measures 2–5 millimeters (figure 26-1). The eggs measure 50–60 micrometers by 20–30 micrometers.

### *Reservoir*

*E. vermicularis* is exclusively a parasite of man.

### *Transmission*

The female worm migrates down the intestine and colon and emerges from the anus, usually at night. Eggs are normally laid on the perianal skin and are seldom found in the feces. Most female worms die after egg laying. The eggs develop to the infective stage in 4–7 hours at 35°C and 48 hours at 25°C. Under cool, moist conditions infective eggs remain alive for up to 8 weeks. When infective eggs are ingested, the larvae hatch in the small intestine and the adults are found in the large intestine, cecum, and appendix. Autoinfection often occurs by transmitting eggs from the anus to the mouth on contaminated fingers or by eggs hatching on the anal mucosa and the larvae migrating up into the bowel and there developing into an adult worm. Each egg ingested may develop into a male or female worm, so at least two are necessary for transmission.

### *Prepatent and incubation periods*

Adult female worms begin to pass eggs 40–50 days after ingestion of eggs. The infections are often asymptomatic.

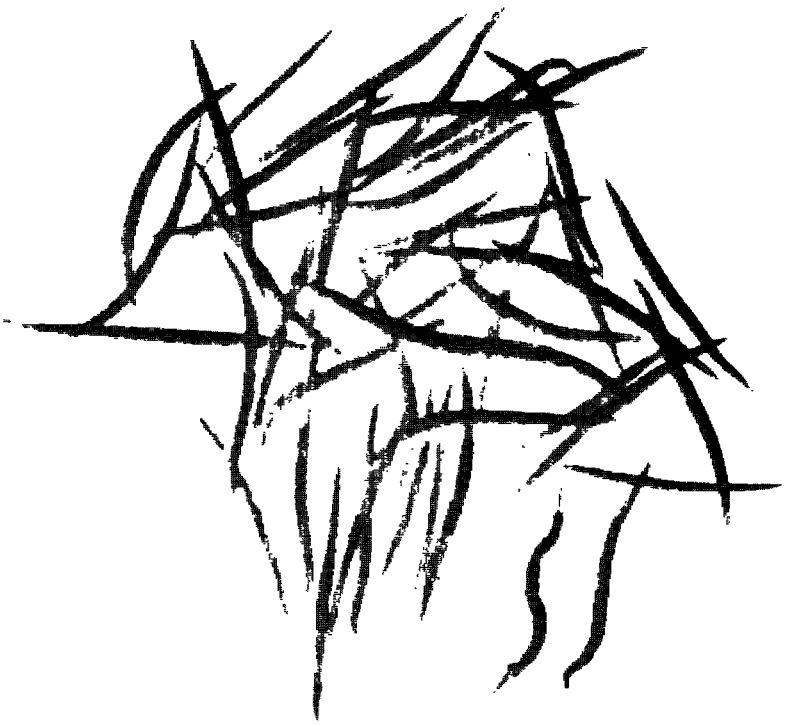

Figure 26-1. *Adult pinworms*, Enterobius vermicularis. Each worm is 5–10 millimeters long. (Photo: Wellcome Museum of Medical Science)

*Period of communicability*

The adults live for about 50 days, but because of autoinfection the period of communicability is usually much longer.

*Resistance*

Susceptibility is general, and there is no evidence of resistance due to past infection.

*Epidemiology*

Enterobiasis is a group infection, most common in children. Its transmission, by the anal-oral route, depends very much on personal hygiene. Contaminated fingers, fingernails, bed linen, table tops, doorknobs, and so forth can serve as sources of infection. In some cases airborne eggs, dislodged from contaminated areas, are inhaled and swallowed and cause infection. Overcrowding and poor housing encourage infection. Infection is most common in large families and institutions such as boarding schools, hospitals, prisons, and orphanages.

Enterobiasis is not an excreted infection in the same sense as the other worm infections described in chapters 22 to 35. Eggs are found in the feces of 5 percent or less of infected individuals. Eggs are normally laid on the perianal skin by female worms that have emerged from the anus at night. It follows that stool surveys greatly underestimate the prevalence of enterobiasis. For an accurate survey it is necessary to adopt the sticky tape method to pick eggs off the perianal skin and examine them under a microscope. For best results, the sticky tape should be applied very soon after waking and before bathing or passing stool. Even then a single survey will detect only 50 percent of infections; three tests will detect 90 percent, and five tests 99 percent (Wolfe 1978). Owing to these difficulties, practically no reliable community-wide prevalence data on enterobiasis exist, and most figures quoted are gross underestimates. This is not a matter for concern. Enterobiasis is an infection of negligible public health importance, and there is generally no reason for needing accurate prevalence data.

A survey of enterobiasis in orphanages in Taipei (Taiwan) showed an overall prevalence of 74 percent, with higher infection rates in more crowded orphanages (Chung, Chang and Horng 1978). Children's bodies were heavily contaminated with *Enterobius* eggs, and 12 percent had eggs on their ears. Bedpans,

linen, toilets, dust, stair rails, bedposts, closets, desks, and toys were contaminated by *Enterobius* eggs in decreasing order of frequency of egg detection (see also Chiu and others 1975). Seo and others (1969) found high rates (up to 80 percent) of enterobiasis among rural school children in South Korea. Other fairly recent surveys of enterobiasis in developing countries include those from Brazil (Dias 1967), Chile (Cuevas and others 1969), India (Sengbusch 1970), Mexico (Garrocho Sandoval and Rodríguez Medina 1968; Vázquez Compeán and Garrocho Sandoval 1972), the Philippines (Sengbusch 1963; Sengbusch and Sengbusch 1971), and Singapore (Kan, Siak and Singh 1971).

Enterobiasis is more common in temperate than in tropical climates because transmission is encouraged by wearing many clothes and by infrequent bathing. These practices maintain the perianal region in a cool, dark, and moist state that is ideal for the survival of *Enterobius* eggs on the perianal skin.

Enterobiasis is the most common worm infection in the USA and is found throughout the country and among all socioeconomic groups (Warren 1974). It is less common among black than white people for reasons that are not known. Prevalence is greatest among the 5–9 age group and is especially high in institutions. It was estimated that 42 million US citizens were infected in 1972 (Warren 1974).

In the course of 3 years (1975–78), systematic surveys were carried out in Azerbajian (USSR) on enterobiasis among the urban and rural populations (Chobanov and Salekhov 1979). A total of 9,914 persons were examined, using adhesive cellophane swabs. The prevalence of enterobiasis in towns and villages was similar (39 percent and 38 percent, respectively), and there was no difference in the infection rates that could be attributed to the level of sanitation and personal hygiene. The lowest infection rate was found in children of preschool age brought up at home (9 percent), whereas the highest was in children attending kindergartens, especially in the 4–7 age group (57–60 percent). The incidence among adults was considerably lower (6–11 percent). The high prevalence of enterobiasis in schools was mainly due to the introduction of the infection by children previously infected in kindergartens, for *Enterobius* eggs were found in up to 60 percent of this group, in comparison with 12 percent in schoolchildren brought up at home. A survey of 889 households showed that in childless families only 4 percent were infected, whereas in families with children the infection rate increased in proportion to their number, reaching 70 percent in families with six children and involving adult members of the family, especially women. Sanitary measures and mass treatment with specific anthelminthics of all the children and personnel of nineteen kindergartens in Baku resulted in a marked and rapid reduction in the cases of enterobiasis, which dropped from 35–68 percent to 4–17 percent a year later. At present, new pupils and personnel are admitted only after undergoing prophylactic treatment. Studies on enterobiasis in the USSR are also reported by Epifantsev and Petrov (1972) and Zhuravlev and Parfenova (1974).

## Control Measures

Drugs such as piperazine, pyrantel pamoate, and mebendazole can be used for mass chemotherapy.

Reduction of overcrowding in living accommodation and adequate facilities for hand washing and personal cleanliness help in the prevention of the infection. Educational effort should be directed to stress personal hygiene.

## Occurrence and Survival in the Environment

*Enterobius* eggs are not usually passed in the feces but are found in bedclothes and house dust. Eggs have been found in sewage in the German Democratic Republic (Kalbe 1956), India (Lakshminarayana and Abdulappa 1969), and the USSR (Vassilkova 1936); in river water contaminated by sewage in the USSR (Bukh 1945; Goryachev 1947; Usacheva 1951); in tap water in the USSR (Bukh 1945); and on crops irrigated with sewage in the USSR (Biziulevicius 1954; Khaustov 1935; Romanenko 1971). These eggs probably came from egg-filled female worms, which are often passed in the feces.

*Enterobius* eggs are not robust and survive for considerably shorter periods than *Ascaris* eggs (chapter 23). Eggs can survive for up to 8 weeks if kept cool and moist, but they are killed in a few days by desiccation. Mature eggs can remain viable for 2–3 days at 22°C and a relative humidity of 34–44 percent (Hulínská 1974).

## Inactivation by Sewage Treatment Processes

Little information is available on *Enterobius* eggs during sewage treatment. It may be assumed that their removal characteristics closely resemble those of

*Ascaris* eggs (chapter 23). *Enterobius* eggs are absent from the effluent of well-designed waste stabilization pond systems (Lakshminarayana and Abdulappa 1969).

## Inactivation by Night Soil and Sludge Treatment Processes

Nothing is known about *Enterobius* eggs during night soil or sludge treatment. *Enterobius* eggs are more rapidly killed by hostile environmental factors (especially heat and desiccation) than are *Ascaris* eggs, and they will be eliminated from night soil and sludge long before *Ascaris* eggs.

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# 27

# *Fasciola* and Fascioliasis

FASCIOLIASIS is an infection of sheep and cattle. Human infection is not common, and transmission from man to man, rather than from animal to man, is probably very rare. Fascioliasis transmission is not affected, therefore, by excreta disposal practices, and it is included in this book for completeness only.

## Description of Pathogen and Disease

The literature on fascioliasis is chiefly veterinary, and little is known of the epidemiology of human infections.

### *Identification*

Fasioliasis is an infection of the bile ducts by the trematode *Fasciola hepatica* that results in inflammation and fibrosis. In heavy infections small abscesses may be produced in the liver parenchyma. There is an early acute phase, when the larvae migrate through the liver, and a late chronic phase caused by the adult flukes in the bile ducts.

Symptoms recorded from human cases in the acute phase include dyspepsia, nausea and vomiting, abdominal pain, and irregular high fever. Allergic symptoms can also occur. Anemia occurs in a proportion of cases, as does leucocytosis and eosinophilia. In the chronic phase there is often painful liver enlargement, and occasionally an obstructive jaundice develops.

Diagnosis is by recovery of eggs from the feces or from bile duct aspirate. In about 30 percent of cases, however, eggs cannot be found in either feces or bile. Fever, liver enlargement, and high eosinophilia are very suggestive of fascioliasis, especially if there is a concomitant history of eating homegrown watercress. Treatment is by oral drug therapy with bithionol or by intramuscular injection of dihydroemetine. Both drugs are toxic and of limited effectiveness. Praziquantel, a newer drug, has been effective in some recent trials.

### *Occurrence*

Fascioliasis (or liver rot) is a disease of sheep and cattle throughout the world. Human infection is not common and has been mainly reported from Central and South America, Cuba, southern France, western England, Wales, and North America. It is to be expected that sporadic human infection will occur wherever fascioliasis is endemic in the local sheep flocks.

### *Infectious agent*

*Fasciola hepatica*, a trematode, is also known as the liver fluke. The adult is a moderately fleshy, flat, hermaphroditic fluke measuring 20–30 millimeters in length by 13 millimeters in breadth (figure 27–1). The eggs are large and ovoid, measuring 130–150 micrometers by 60–85 micrometers.

The related fluke, *F. gigantica*, measuring 25–75 millimeters by 12 millimeters, is a common parasite of cattle, camels, and other herbivores in Africa, Asia, and some Pacific islands and has been reported from man in Africa, Iran, and Hawaii.

### *Reservoirs*

The main reservoirs of *Fasciola hepatica* are sheep and cattle. Infections of rabbits, horses, donkeys, camels, pigs, and deer are also reported. Human infection is infrequent.

### *Transmission*

The eggs are laid in the proximal biliary tract of the host and are evacuated in the feces. They mature in water or in moist conditions within 9–15 days at an optimal temperature of 22–25°C. The miracidia hatch and within 8 hours invade an amphibious snail, usually a species of *Lymnaea*. During 30–40 days the larvae develop into sporocysts followed by two redial stages,

Figure 27-1. *An adult* Fasciola hepatica *under a light microscope.* Scale bar = 5 millimeters. (Photo: Wellcome Museum of Medical Science)

and cercariae emerge. The cercariae swim in the water for up to 8 hours and then encyst as metacercariae on aquatic vegetation. Sheep and cattle become infected when they eat encysted metacercariae on grass. People become infected when they eat encysted metacercariae on watercress, salad vegetables, or any water plant eaten raw. When the cysts are ingested, the larvae excyst in the duodenum, migrate through the intestinal wall, and reach the bile ducts by eating their way through the surface of the liver and through the liver parenchyma.

The life cycle of *F. gigantica* is similar, except that the snail hosts are aquatic, not amphibious.

### *Prepatent and incubation periods*

The adult fluke matures 3–4 months after ingestion of the encysted metacercariae. Acute symptoms due to the migrating larvae in the liver may be experienced soon after the ingestion of metacercariae.

### *Epidemiology*

Fascioliasis is an infection of herbivores, principally sheep and cattle. Man sometimes accidentally takes the place of the herbivore as the vertebrate host in the parasitic life cycle. Small foci of human fascioliasis have been reported, but it is a rare infection in man. There is no firm evidence of man-to-man transmission, and it is probable that human infections only occur where there is endemic fascioliasis among sheep or cattle.

A stool survey of 1,011 schoolchildren from six villages in the Peruvian Andes showed a 9 percent prevalence of fascioliasis (Stork and others 1973). Watercress was not consumed in this area, and it was considered that lettuce and alfalfa were the sources of human infections. Other accounts of fascioliasis include those from Australia (Wood, Stephens and Porter 1975), Britain (Anon 1978), Dominican Republic (Ueno and others 1973), Egypt (Farag and others 1979), France (Rondeland, Amat-Frut and Pestre-Alexandre 1982), Iran (Farid 1971), Japan (Ueno and others 1975), Madagascar (Moreau and others 1975), Norway (Brandt 1974), and Switzerland (Boray 1971). *F. gigantica* infection in animals in West Africa was reviewed by Schillhorn van Veen (1980).

## Control Measures

Because the animal reservoir is more important for transmission of fascioliasis than the human reservoir, excreta disposal will have little effect on the control of the infection. Snail control, though difficult to achieve, can be attempted by the use of molluscicides and by drainage of pasture. Watercress beds should be protected from contamination by animal feces, and the public should be informed of the danger of eating watercress, or other salad vegetables, grown in wet land where sheep or cattle have access.

## Occurrence and Survival in the Environment

There is a substantial literature on the survival and development of *Fasciola* eggs, and this information has

been used to explain the seasonal pattern of fascioliasis in animals in Europe. Eggs survive for many months below 10°C but do not develop. Therefore, in temperate climates many eggs overwinter, and the miracidia hatch out in spring. The development in snails takes place over the summer, and the encysted metacercariae on herbage build up in early autumn. These metacercariae can also survive over winter.

*Fasciola* eggs must be free of feces and surrounded by moisture to develop. They are rapidly killed by desiccation. They develop most rapidly at 25–30°C; they do not develop below 10°C and they are killed at −5°C (Becejac and Lui 1959; Rowcliffe and Ollerenshaw 1960; Tunker 1940; Valenzuela 1979).

*Fasciola* eggs survive in slightly brackish water but not in seawater (Saint Guillain and Pecheur 1967; Styczńska-Jurewicz 1965*b*). In anaerobic water, eggs survive but do not develop. Development recommences when oxygen is supplied (Becejac and Lui 1959; Styczńska-Jurewicz 1965*a*). Eggs develop in waters with pH between 4.2 and 9.0, a wider pH range than is tolerated by the snail host (Rowcliffe and Ollerenshaw 1960; Saint Guillain and Pecheur 1967).

*Fasciola* eggs develop not only in water, but also in a 1:1 mixture of water and liquid manure. The survival times in this medium are 70 days at 15–18°C and 101 days at 4–8°C (Six and Hoffman 1970).

Miracidia usually survive in water for only a few hours. Survival is enhanced by cool water temperatures, by darkness, by high oxygen levels, and by neutral pH (Dwaronat 1966; Gebauer 1958).

Encysted metacercariae of *F. hepatica* can survive for several months, especially if conditions are cool and moist. They survive on a wet surface for 12 hours at −20°C, 4 weeks at −10°C, 13 weeks at −2°C, and 19 weeks at 10–20°C. Survival times decrease at temperatures above 25°C or at lower humidities (Boray and Enigk 1964; Kakatchéva-Avramova 1963). Metacercariae of *F. gigantica* are more resistant to warm temperatures but more susceptible to desiccation (Boray and Enigk 1964).

## Inactivation by Sewage Treatment Processes

*Fasciola* eggs rarely occur in sewage and in very low concentrations. Their removal during sewage treatment is of no interest, since it is *Fasciola* eggs in sheep and cattle feces that are responsible for the maintenance of transmission.

## Inactivation by Night Soil and Sludge Treatment Processes

*Fasciola* eggs in night soil and sludge are of no interest because transmission is maintained by eggs in sheep and cattle feces. *Fasciola* eggs in manure and farm slurry are important, and it may be assumed that any processes that eliminate *Ascaris* eggs (chapter 23) will also eliminate *Fasciola* eggs.

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# 28

# *Fasciolopsis* and Fasciolopsiasis

FASCIOLOPSIASIS is a disease of rural people in areas of eastern Asia where certain water plants are eaten raw. It is not of great public health importance.

## Description of Pathogen and Disease

The literature on fasciolopsiasis is not large, although the disease is attracting increased research interest in Bangladesh and elsewhere.

### *Identification*

Fasciolopsiasis is an infection of the small intestine, particularly the duodenum, by the trematode *Fasciolopsis buski*. In the majority of cases the infection is light, and there are no symptoms. Heavy infections may cause intestinal obstruction and symptoms such as nausea, diarrhea, fever, and abdominal pains. Patients may show edema of the face, the abdominal wall, and the legs within 20 days after massive infection. Ascites is common, as is eosinophilia; secondary anemia occurs occasionally; death is rare.

Diagnosis is by finding flukes or characteristic eggs in the feces. Treatment is by oral drug therapy with hexylresorcinol, tetrachlorethylene, or bithionol.

### *Occurrence*

*Fasciolopsis buski* occurs in man in Southeast Asia, especially in central and south China. It seems to be restricted to areas where cultivation of water plants such as water caltrop, water chestnuts, water hyacinth, and water bamboo takes place and in communities that consume uncooked infected plants. Endemic areas are found in Bangladesh, Kampuchea, China, India, Indonesia, Laos, Taiwan, Thailand and Vietnam (figure 28-1). Human infections reported in Japan, the Philippines, and Malaysia probably occur in people who have emigrated from endemic areas.

### *Infectious agent*

*Fasciolopsis buski*, a trematode, is the giant intestinal fluke of man. The adult is fleshy, elongated, and ovoid and is the largest trematode of man, measuring 20–75 millimeters by 8–20 millimeters (figure 28-2). The eggs are 130–140 micrometers by 80–85 micrometers and are very similar to those of *Fasciola* hepatica.

### *Reservoirs*

Man, pigs, and dogs are definitive reservoir hosts of adult flukes. Pigs are especially important in the maintenance of endemic fasciolopsiasis in central Thailand and some other areas.

### *Transmission*

The adult worm, which lives attached to the wall of the small intestine, lays about 25,000 unembryonated eggs per day. The eggs are passed in the feces. When the eggs reach fresh water they develop and hatch under favorable conditions (temperature 27–32°C) within 3–7 weeks.

The hatched miracidia penetrate a freshwater planorbid snail, and a process of asexual multiplication occurs that results in developed cercariae, which then emerge into the water. They swim in the water and become attached to aquatic vegetation such as seed pods of water caltrop, bulbs of water chestnuts, and roots of lotus, water bamboo, and others. There they encyst as metacercariae.

When ingested with edible plants, the metacercariae excyst in the duodenum of man, and the young flukes develop.

### *Prepatent and incubation periods*

Mature flukes develop and start laying eggs within 3–4 months after infective cysts have been ingested.

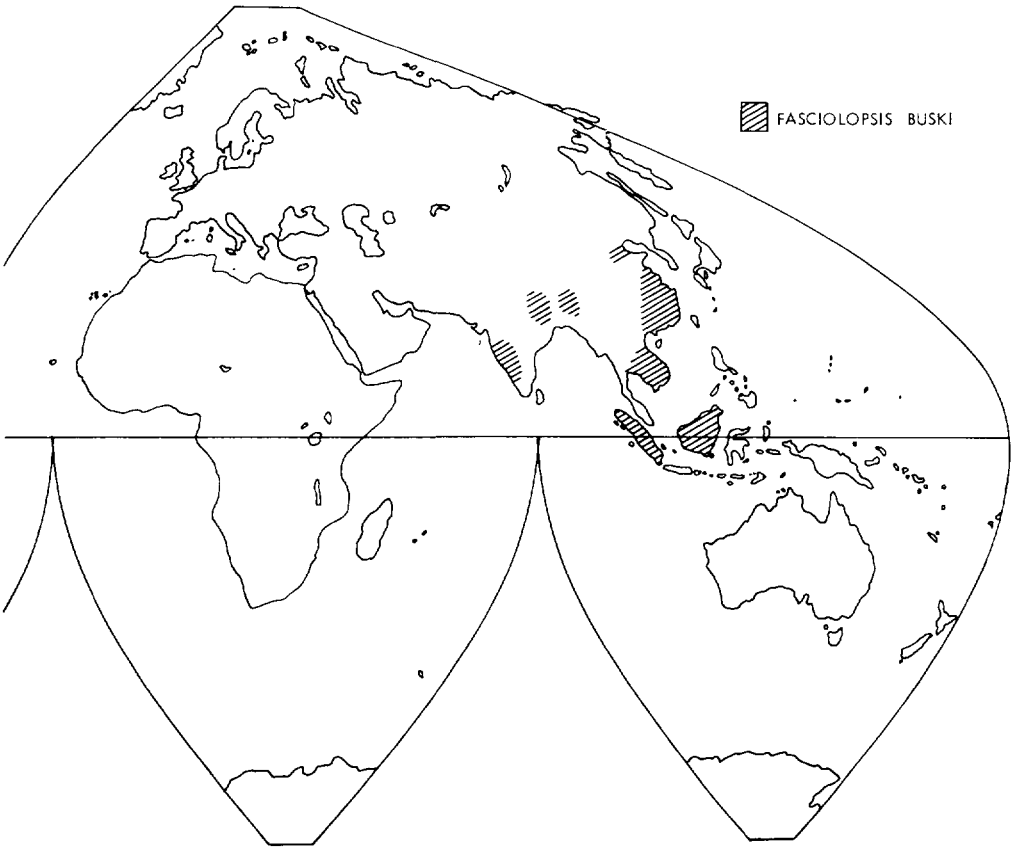


Figure 28-1. *Known geographical distribution of* Fasciolopsis buski. The infection may occur in areas as yet unrecorded

Massive infection can lead to symptoms within 20 days, but more usually symptoms develop slowly or not at all.

*Period of communicability*

Eggs may be passed in the feces as long as a mature worm is present in the intestine. Adult worms live for only about 6 months in man.

*Resistance*

There is no resistance proved. Malnourished individuals are more prone to symptoms.

*Epidemiology*

Prevalences of fasciolopsiasis are over 70 percent in some endemic foci. The seed pods of water caltrop are an important source of infection, especially where they are cultivated in ponds enriched with night soil. Water chestnut, water bamboo, water hyacinth, lotus, and watercress are also implicated as sources of infection. Before the nuts of water caltrop or the bulbs of water chestnut are eaten, the outer covering is removed with the teeth, and this may be the primary mode of infection. This practice is especially common among children, who are usually more heavily infected than adults. Peak prevalences have been reported in the 10–14 age group in central Thailand (Sadun and Maiphoom 1953) and in Taiwan (Hsieh 1960) and in the 5–14 age group in Bangladesh (Rahman, Idris and Khan 1981). In Taiwan, Thailand, and other areas, water plants are harvested and fed to pigs, and this helps to maintain a high prevalence of fasciolopsiasis in pigs.

Reports of fasciolopsiasis epidemiology include those from Bangladesh (Rahman, Idris and Khan 1981). China (Barlow 1925; Chu and others 1959), Taiwan (Hsieh 1960; Lee 1972), Thailand (Manning, Brockelman and Viyanant 1971; Manning and Ratanarat 1970; Sadun and Maiphoom 1953), and elsewhere in Southeast Asia (Cross 1969).

Figure 28-2. *An adult* Fasciolopsis buski *under a light microscope.* Scale bar = 5 millimeters. (Photo: Wellcome Museum of Medical Science)

## Control Measures

Metacercariae on plants can be killed by drying the plants or dipping them in boiling water.

The use of excreta as fertilizer in fields and ponds is an important factor in the transmission of fasciolopsiasis. It is relatively simple to treat the excreta and kill the *Fasciolopsis* eggs because these eggs are quite susceptible to adverse conditions. In areas where pigs are important in maintaining a reservoir of *Fasciolopsis*, steps must be taken to prevent pig excreta from reaching water in which plants for human consumption are grown.

The transmission of fasciolopsiasis depends on the customs and habits of inhabitants of endemic areas who grow and eat water plants. Public health education should promote changes in night soil reuse and disposal and in the consumption of raw water plants.

## Occurrence and Survival in the Environment

The four stages of *Fasciolopsis* found in the environment are eggs, miracidia, cercariae, and encysted metacercariae. Eggs hatch in water within 3–7 weeks at 27–32°C, and hatching is inhibited at temperatures above and below this range (Barlow 1925). In winter in Taiwan, with water temperatures below 20°C, immature eggs may survive but do not continue their development (Suzuki 1922). Eggs die in human urine within a few hours (Komiya 1964) and in feces within 18 days (Barlow 1925). Eggs are rapidly killed by desiccation. Miracidia in water must find an appropriate snail within 8 hours or die.

Encysted metacercariae are killed by desiccation, direct sunlight, and warm temperatures. They survive for up to 30 minutes in direct sunlight, 15 minutes at 60°C, and 1 minute in boiling water (Barlow 1925;

Komiya 1964). Metcercariae are also killed in 2 percent acetic acid in 9 days, 5 percent salt solution in 3 hours, and soybean sauce in 30 minutes (Komiya 1964).

## Inactivation by Sewage Treatment Processes

The fate of *Fasciolopsis* eggs in sewage treatment plants has not been studied. Most transmission is associated with direct enrichment of ponds with feces or night soil rather than with accidental contamination from poorly treated sewage effluents. In addition, fasciolopsiasis is endemic in poor rural areas where most people use simple latrines, or no latrines, and certainly produce no sewage.

## Inactivation by Night Soil and Sludge Treatment Processes

*Fasciolopsis* eggs in night soil or sludge may be killed by drying, freezing, heating, or storage for 18 days (Barlow 1925). Eggs survived for up to 28 days in biogas plants in China (Hou 1959).

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# 29

# *Hymenolepis* and Hymenolepiasis

THREE GENERA of tapeworm are parasites of the human intestine and are transmitted when proglottids or eggs are passed in the feces. These are *Diphyllobothrium* (chapter 25), *Taenia* (chapter 34), and *Hymenolepis* (this chapter). The *Hymenolepis* species that infects man, *H. nana*, is unusual in that it can be transmitted directly from person to person without a cystic stage in an intermediate host. This is in contrast to *Diphyllobothrium* (which has intermediate stages in a copepod and a fish) and *Taenia* (which has an intermediate stage in a cow or a pig).

## Description of Pathogen and Disease

Hymenolepiasis is not a major public health problem, although in certain localities it is common and is regarded seriously by clinicians. The literature on hymenolepiasis epidemiology is limited.

### *Identification*

Hymenolepiasis is an infection of the small intestine by the tapeworm (cestode) *Hymenolepis*. With a light infection, symptoms are often vague or absent. Heavy infections can result in enteritis with abdominal pain, diarrhea, loss of appetite, and dizziness. Epileptic fits are an occasional complication in children. Diagnosis is by identifying *Hymenolepis* eggs in the feces. Treatment is by oral drug therapy with niclosamide. Praziquantel, a newer drug under trial, shows promise (Schenone 1980).

### *Occurrence*

Hymenolepiasis occurs worldwide and is especially prevalent among children. It is more common in warm climates than in cold climates. It is most common in South America, North India, the Mediterranean countries, Eastern Europe, and the Pacific islands.

### *Infectious agent*

*Hymenolepis nana*, a cestode, is the dwarf tapeworm of man. The entire worm measures only 15–40 millimeters by 1 millimeter and has approximately 200 proglottids. The minute scolex has four suckers and a row of hooks and is embedded in the wall of the ileum (figure 29-1). The mature proglottids measure 0.22 by 0.88 millimeters and the eggs measure 30–47 micrometers in diameter.

### *Reservoirs*

The reservoir of *H. nana* is probably man. A morphologically identical tapeworm (*H. nana* var. *fraterna*) is common in mice, but it is not certain whether this normally infects man.

The rat tapeworm, *H. diminuta*, is a common parasite of rodents in many parts of the world. Like most cestodes it has an intermediate host, in this case a rat flea or flour beetle that must eat the excreted eggs. *H. diminuta* cannot be transmitted directly from rodent to rodent (Salem, Sidky and Abdel-Rehim 1980). The rodent is reinfected by eating the flea or beetle containing the encysted worm. Children occasionally ingest infected fleas or beetles, and *H. diminuta* infections are found in children in some countries. *H. diminuta* has little medical or public health importance but has been much used as a laboratory model for cestode infections, and there is a substantial literature on it. A very similar pattern of occasional human infection is found for the dog tapeworm (*Dipylidium caninum*), for which the intermediate host is a dog flea.

### *Transmission*

The gravid proglottids containing 80–200 eggs are usually broken up in the intestine so that free eggs are found in the feces. Eggs passed in feces are immediately infective if ingested by a new host. The eggs are not resistant to heat or desiccation and normally survive

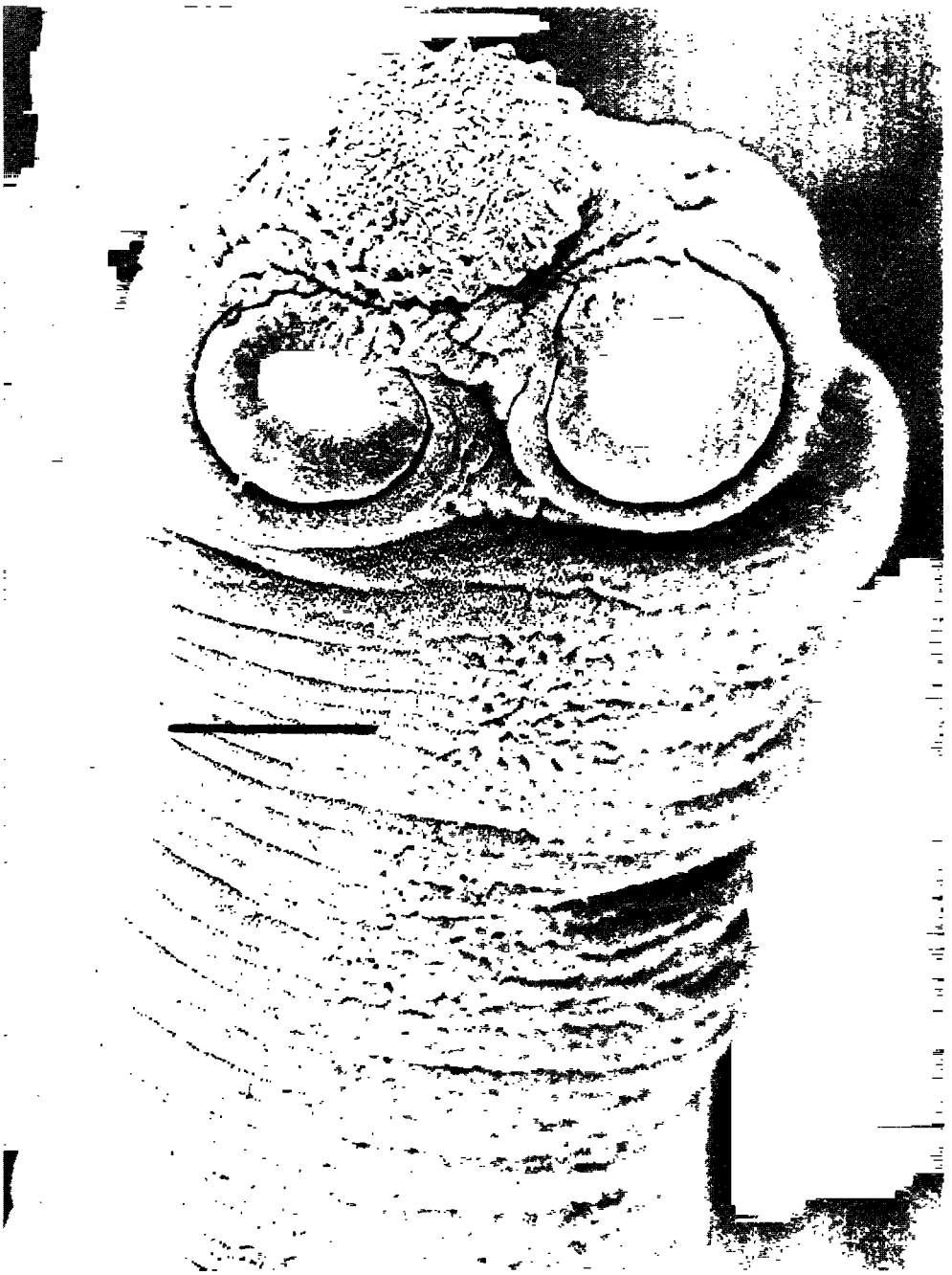

Figure 29-1. *Head (scolex) and neck of* Hymenolepis nana *under scanning electronmicroscopy.* Two of the four suckers and the protruding rostellum with a circle of hooks are prominent. The suckers and hooks attach to the wall of the small intestine while the worm hangs in the lumen. Scale bar = 0.1 millimeter. (Photo: H. Mehlhorn, Institut für Zoologie, Düsseldorf, Federal Republic of Germany.)

for less than 10 days in the environment. Transmission is usually from hand to mouth, although it can be via food or water. The contained larva is liberated in the small intestine and penetrates a villus. After development it emerges into the lumen and develops into a mature tapeworm in 10–12 days. Autoinfection is possible by eggs hatching in the intestine and developing into new tapeworms without leaving the host. One ingested egg is potentially capable of starting a new infection.

*H. nana* is an unusual cestode in that it can pass directly between two primary hosts (man) without first

encysting in an intermediate host (in contrast to *Taenia*—see chapter 34). *H. nana* can, however, develop in certain insects, and some transmission may take place via the accidental ingestion of infected insects by children.

*Prepatent and incubation periods*

Worms mature and start shedding egg-filled proglottids about 30 days after the ingestion of eggs. Symptoms may never develop.

*Period of communicability*

Eggs will be passed in the feces as long as adult tapeworms are in the intestine. Although these live only for a few months, regular autoinfection can cause eggs to be excreted for years.

*Resistance*

Although susceptibility is general, there is evidence for acquired immunity (Rifaat, Salem and Hegazi 1978).

*Epidemiology*

Hymenolepiasis infection is considerably more common in children than in adults and is also especially common in crowded homes and institutions. Major local and regional variations in prevalence exist. In a survey of children 0–6 years old, hymenolepiasis prevalences of 0 percent were found in Bangladesh and Sri Lanka, 9 percent in Venezuela, and 20 percent in Iran (van Zijl 1966).

A study of a village in Rajasthan (India) showed an overall hymenolepiasis prevalence of 6 percent. Infection was more common among those under 15 years old (10 percent) than among adults (4 percent). Larger and more crowded households were more infected than other households. Of all those infected, 35 percent had symptoms, of which the most common were diarrhea and abdominal pain (Biswas, Arora and Sehgal 1978). Other studies of hymenolepiasis in developing countries include those from Brazil (Huggins and others 1973), Egypt (Chandler 1954), India (Buscher and Haley 1972), Iran (Ghadirian and Arfaa 1972; Massoud and others 1980), South Africa (Van Niekerk and others 1979), South Korea (Seo and others 1969), and Turkey (Yaşarol, Orhan and Erefe 1970).

Hymenolepiasis occurs in the USA, especially in the southern states of Florida, Kentucky, Mississippi, North and South Carolina, and Tennessee (Eyles, Jones and Smith 1953; Melvin and Brooke 1962; Warren 1974). Warren (1974) estimated that 100,000 US citizens were infected in 1972. Hymenolepiasis is very common in some regions of the USSR and has been the subject of considerable research and control activity (Kuznetsov 1979; Lerner and others 1970). Human infection by *H. diminuta* has been described in Malaysia (Sinniah 1978), Papua New Guinea (McMillan, Kelly and Walker 1971), Thailand (Chitchang, Sooksala and Radomyos 1978), Zambia (Hira 1975), and elsewhere.

## Control Measures

Mass chemotherapy with niclosamide, praziquantel, or other suitable drugs can temporarily reduce local prevalences.

Eggs are infective when passed in the feces. Therefore, direct fecal-oral transmission is likely, particularly among children or others with poor standards of personal hygiene. Control must lie in improving personal hygiene, as well as in improving the methods for excreta disposal.

## Occurrence and Survival in the Environment

*Hymenolepis* eggs may be expected in sewage and night soil, and in environments contaminated by sewage or night soil, wherever hymenolepiasis is endemic. *Hymenolepis* eggs in the environment have not attracted much research interest, however, and few reports are available. *H. nana* eggs have been found in sewage in India (Lakshminarayana and Abdulappa 1969; Panicker and Krishnamoorthi 1978) and in sewage sludge in Czechoslovakia (Králová and Šafránek 1957) and the USA (Wright, Cram and Nolan 1942).

*Hymenolepis* eggs do not survive for long in the environment compared with *Ascaris* eggs (chapter 23). They are especially sensitive to warmth and desiccation. Table 29-1 summarizes the findings of Simitch, Bordjochki and Angelovski (1955). Another study found that *H. nana* eggs were rapidly killed at −1°C and at 45°C, and by drying for 30 minutes at 37°C (Foresi and Ruschi 1968).

## Inactivation by Sewage Treatment Processes

Data from India (table 22-4) suggest that *H. nana* eggs are removed somewhat less effectively than

Table 29-1. *Duration of infectivity of* Hymenolepis nana *eggs stored under various conditions*

| | *Duration of infectivity at various temperatures (hours)* | | | | | |
|---|---|---|---|---|---|---|
| *Environment* | *0°C* | *2°C* | *20°C* | *37°C* | *41°C* | *41°C in water* |
| Crumbled feces | ND | ND | <30 | <4 | <4 | <2 |
| Compact feces | >144 | >240 | <72 | <8 | <4 | <3 |
| Feces in water | ND | ND | >720 | <120 | <10 | <30 |

ND No data.
*Source:* Simitch, Bordjochki and Angelovski (1955).

*Ascaris* eggs during sewage treatment. Nonetheless, it is to be expected that *H. nana* eggs, like all other helminth eggs, are mainly concentrated into the raw sludge of the primary and secondary sedimentation tanks. *H. nana* eggs are completely removed from the effluent of well-designed waste stabilization pond systems (table 22-4 and Lakshiminarayana and Abdulappa 1969).

## Inactivation by Night Soil and Sludge Treatment Processes

*H. nana* eggs will be readily eliminated from night soil and sludges, especially if warm temperatures are created (table 29-1) or if desiccation takes place. Thermophilic composting destroys *H. nana* eggs (Gudzhabidze and Lyubchenko 1959), and *H. nana* eggs will be eliminated from all processes long before *Ascaris* eggs.

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# 30

# Minor Intestinal Flukes and Infections They Cause

THREE TREMATODES, in addition to *Fasciolopsis*, infect the human intestine: *Heterophyes heterophyes*, *Metagonimus yokogawai*, and *Gastrodiscoides hominis*. They are quite common in limited geographical areas. They are of only minor public health importance, and are included in this book for completeness.

## Description of Pathogens and Diseases

Little is known of these infections or their epidemiology.

### *Identification*

Heterophyiasis, metagonimiasis, and infection by *Gastrodiscoides hominis* are trematode infections of the small intestine. Infections are usually asymptomatic, but occasionally minor intestinal disturbances such as nausea, diarrhea, fever, and abdominal pain may occur.

Diagnosis is by identifying eggs in the feces. Treatment is by appropriate oral drug therapy.

### *Occurrence*

*Heterophyes heterophyes* has a disjunct distribution, being found in southern Europe (Romania and Greece), the Middle East (Egypt and Israel), and East Asia (China, Japan, Philippines, South Korea, and Taiwan) (figure 30-1). *Metagonimus yokogawai* occurs in China, Japan, Korea, Taiwan and the eastern USSR (figure 30-1). *Gastrodiscoides hominis* is found in Bangladesh, India, Philippines, and Vietnam (figure 30-2).

### *Infectious agents*

These are all hermaphroditic flattened trematodes. *Heterophyes heterophyes* and *Metagonimus yokogawai* (which are very similar in morphology and life history) are 1.5 by 0.5 millimeters (figure 30-3), and *Gastrodiscoides hominis* measures 6 by 4 millimeters (figure 30-4). The eggs of *H. heterophyes* and *M. yokogawai* measure 30 by 15 micrometers, and those of *G. hominis* 146 by 66 micrometers.

### *Reservoirs*

These are all primarily parasites of animals. *H. heterophyes* and *M. yokogawai* infect dogs, cats, foxes, and other fish-eating mammals, and perhaps birds. *G. hominis* infects pigs, monkeys, and rats. All three parasites can probably be maintained in the absence of man.

### *Transmission*

For all these parasites, eggs are passed in the feces and have to reach water for further development. Larvae develop in specific freshwater snails, and a process of asexual multiplication occurs so that some hundreds of the next free-living stage, the cercariae, are released from the snail into the water. These cercariae then form encysted metacercariae. *Heterophyes* and *Metagonimus* encyst under the scales, on the surface, or in the superficial muscle of fish. *Gastrodiscoides* encysts on water plants. The habitat of the snail and fish intermediate hosts of *Heterophyes* is brackish water. The habitat of the snail intermediate host of *Metagonimus* is freshwater, whereas the fish intermediate host lives in both fresh and brackish water. Animal or human infection takes place when raw fish or water plants are ingested. Thus the life cycles of

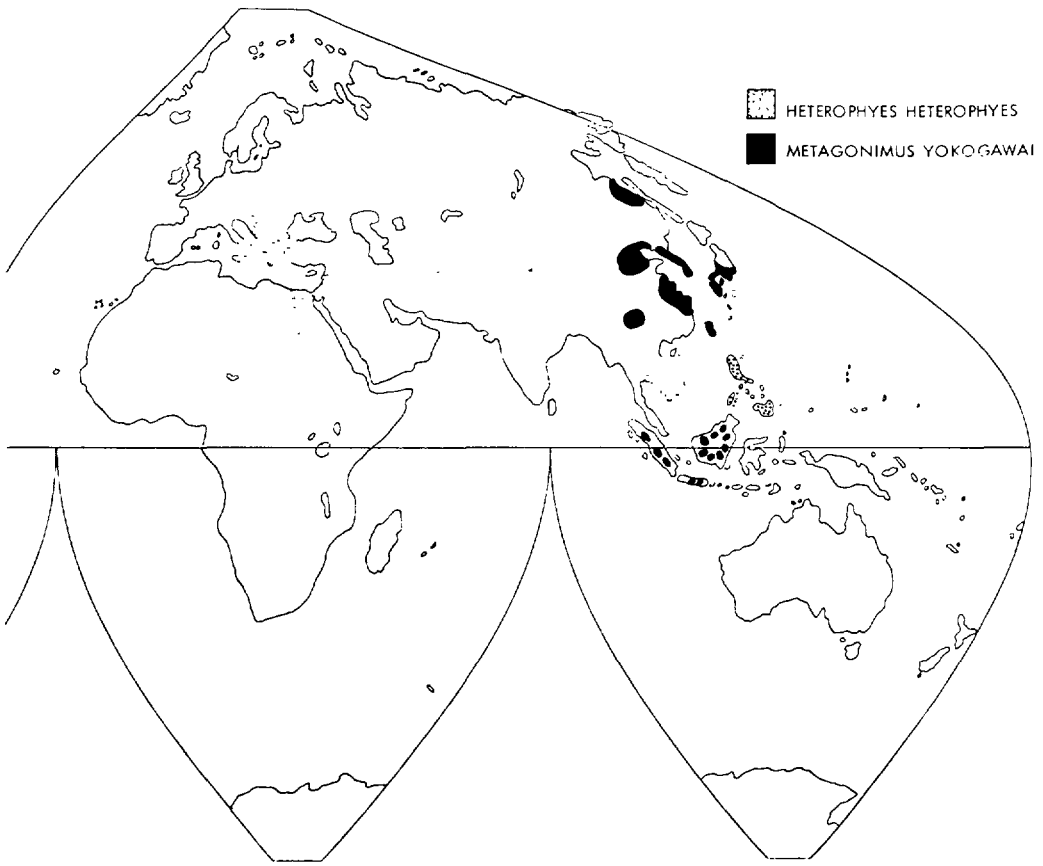


Figure 30-1. *Known geographic distributions of* Heterophyes heterophyes *and* Metagonimus yokogawai. The infections may occur in areas as yet unrecorded

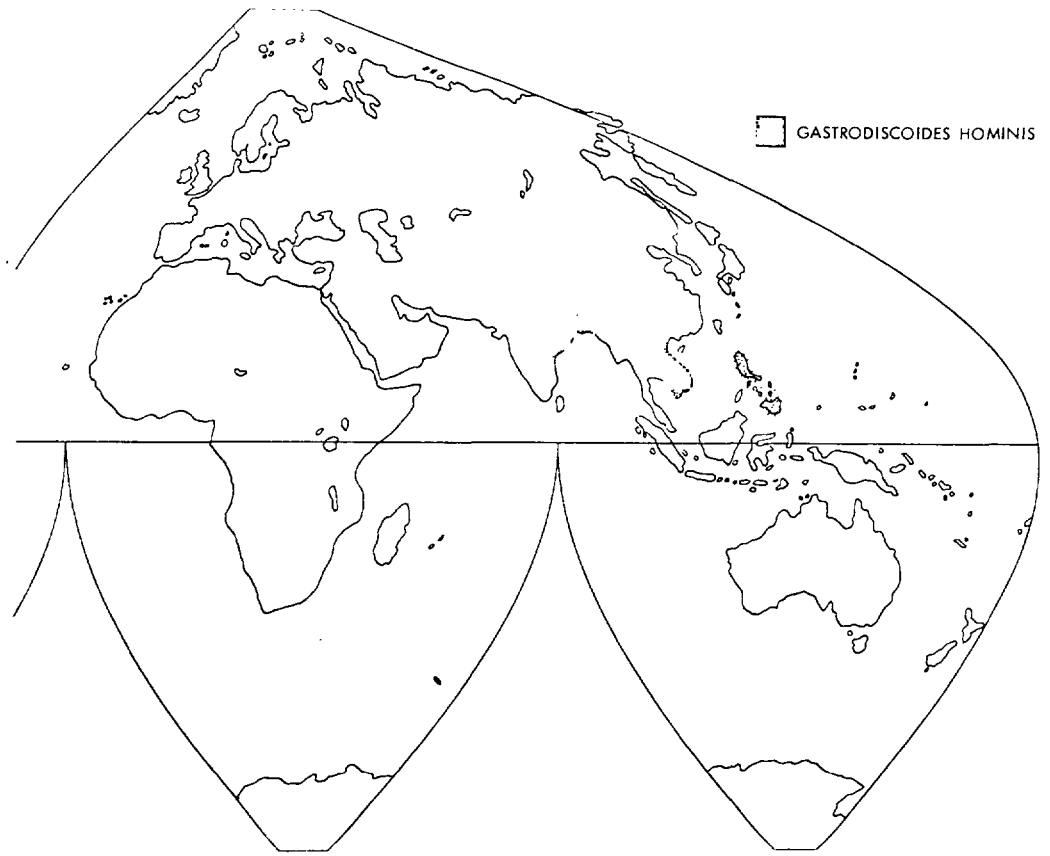


Figure 30-2. *Known geographical distribution of* Gastrodiscoides hominis. The infection may occur in areas as yet unrecorded


Figure 30-3. *Adult* Heterophyes heterophyes (*a*) *and* Metagonimus yokogawai (*b*) *under a light microscope.* Scale bars = 0.1 millimeter. (Photos: Wellcome Museum of Medical Science)

Figure 30-4. *An adult* Gastrodiscoides hominis *under a light microscope.* Scale bar = 1 millimeter. (Photo: Wellcome Museum of Medical Science)

*Heterophyes* and *Metagonimus* are similar to *Clonorchis* (chapter 24), whereas the life cycle of *Gastrodiscoides* resembles that of *Fasciolopsis* (chapter 28).

*Prepatent and incubation periods*

*Metagonimus* and *Heterophyes* flukes develop and begin to lay eggs 15–20 days after encysted metacercariae have been ingested.

*Period of communicability*

As long as mature flukes are present in the intestine, eggs will be passed. Mature flukes live for about 2 months.

*Resistance*

There is no evidence of immunity or resistance.

*Epidemiology*

Little is known of the epidemiology of *H. heterophyes* and *M. yokogawai* and almost nothing of *G. hominis.* It may be broadly assumed that the epidemiology of the first two resembles that of *Clonorchis* or *Opisthorchis* (chapter 24), whereas that of *G. hominis* resembles *Fasciolopsis* (chapter 28).

In Egypt, heterophyiasis is associated with eating freshly salted mullet (Khalil 1933; Martin and Kuntz 1955). Heterophyiasis in the Philippines was reviewed by Africa and Garcia (1935). Metagonimiasis does not occur in areas where the summer water temperatures are below 18°C because the cercariae do not emerge from the snails in cool water. Seo and others (1969) surveyed 40,000 people in South Korea and found a prevalence of metagonimiasis of 0.4 percent. Infection rates were higher in males than females and higher in adults than in children. These age and sex differences are typical of heterophyiasis and metagonimiasis and are due to differences in diet. Metagonimiasis in the USSR was discussed by Zubov, Drozdov and Chernova (1970). The epidemiology of *G. hominis* infection is poorly understood, although there have been several reports from India (Ahluwalia 1960; Buckley 1939; Dutt and Srivastava 1972; Varma 1957).

## Control Measures

Control of *H. heterophyes* and *M. yokogawai* is as recommended for *Clonorchis* (chapter 24). Control of *G. hominis* is as recommended for *Fasciolopsis* (chapter 28).

## Occurrence and Survival in the Environment

Little is known. *H. heterophyes* cercariae survive for 20 minutes in freshwater and for 2 days in seawater. *M. yokogawai* cercariae survive for 8 hours in freshwater (Ito 1964).

*M. yokogawai* encysted metacercariae in fish survive for 15 minutes at 70–80°C, 2 hours in vinegar, 6 hours in soybean sauce, 3 days in rice wine, 7 days in beer, 10 days frozen, and 14 days in tap water (Ito 1964).

## Inactivation by Sewage Treatment Processes

There is no specific information.

## Inactivation by Night Soil and Sludge Treatment Processes

There is no specific information.

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# 31

# *Paragonimus* and Paragonimiasis

THE LIFE CYCLE of *Paragonimus* is similar to that of *Clonorchis*, *Heterophyes*, and *Metagonimus* except that the metacercariae encyst in crabs and crayfish rather than fish. Paragonimiasis is therefore a disease of people who customarily eat raw crabs or crayfish.

## Description of Pathogen and Disease

Paragonimiasis can be a very serious disease and has been studied in detail, especially in China, Japan, South Korea, and Taiwan.

### *Identification*

Paragonimiasis is an infection, principally of the lungs but sometimes of the brain, with a trematode of the genus *Paragonimus*. It is characterized by severe chest pains, dyspnea, and bronchitis. Symptoms resemble those of tuberculosis, especially blood-stained sputum. Cerebral paragonimiasis may result in epileptic seizures, headache, visual disturbances, and symptoms of meningitis.

Diagnosis is by finding eggs in feces or sputum. Treatment is by oral drug therapy with bithionol or praziquantel.

### *Occurrence*

Paragonimiasis in animals occurs worldwide among mammals that feed on crabs or crayfish. Paragonimiasis in man is limited to areas where dietary customs allow infection. *P. westermani* infections occur mainly in China, Japan, Korea, the Philippines and Taiwan—with cases also reported from India, Indonesia, Malaysia, Thailand, and Vietnam. Other *Paragonimus* species occasionally infect man in Asia, Africa, and Central and South America (figure 31-1).

### *Infectious agent*

*Paragonimus westermani*, a trematode, is the lung fluke of man. The adult worm, which typically lives encapsulated in pockets of the lung, is a thick, fleshy, ovoid fluke measuring 8–16 by 4–8 millimeters (figure 31-2). The eggs are 80–110 by 50–60 micrometers.

### *Reservoir*

Paragonimiasis is an infection found in a great variety of mammals that feed on crabs. *P. westermani* can infect a range of wild animals such as tigers, lions, wild cats, and foxes and domestic animals such as cats and dogs. Although in endemic areas man is the most important reservoir, the persistence of *P. westermani* in nature does not depend only on the human reservoir.

Various other *Paragonimus* species are maintained solely by animals in most tropical areas of the world and are the cause of occasional cases in man. For instance, *P. africanus* is the lung fluke of the crab-eating mongoose and infects man in parts of eastern Nigeria and Cameroon.

### *Transmission*

The unsegmented fertilized eggs are passed out in sputum or swallowed and passed out in feces. For further development they have to reach water. At an optimum temperature of 27°C, a larva (miracidium) develops in 3 weeks. After hatching, it swims in the water and survives for around 24 hours. Further development takes place inside various operculate freshwater snails (*Semisulcospira libertina*, *S. amurensis*, *Thiara granifera*, *Oncomelania nosophora*). Asexual multiplication, taking 3 months, occurs in the snail so that a few hundred of the emerging larval stage (cercariae) are formed from each miracidium.

The cercariae can swim in the water for 24–48 hours but require another intermediate host, a freshwater

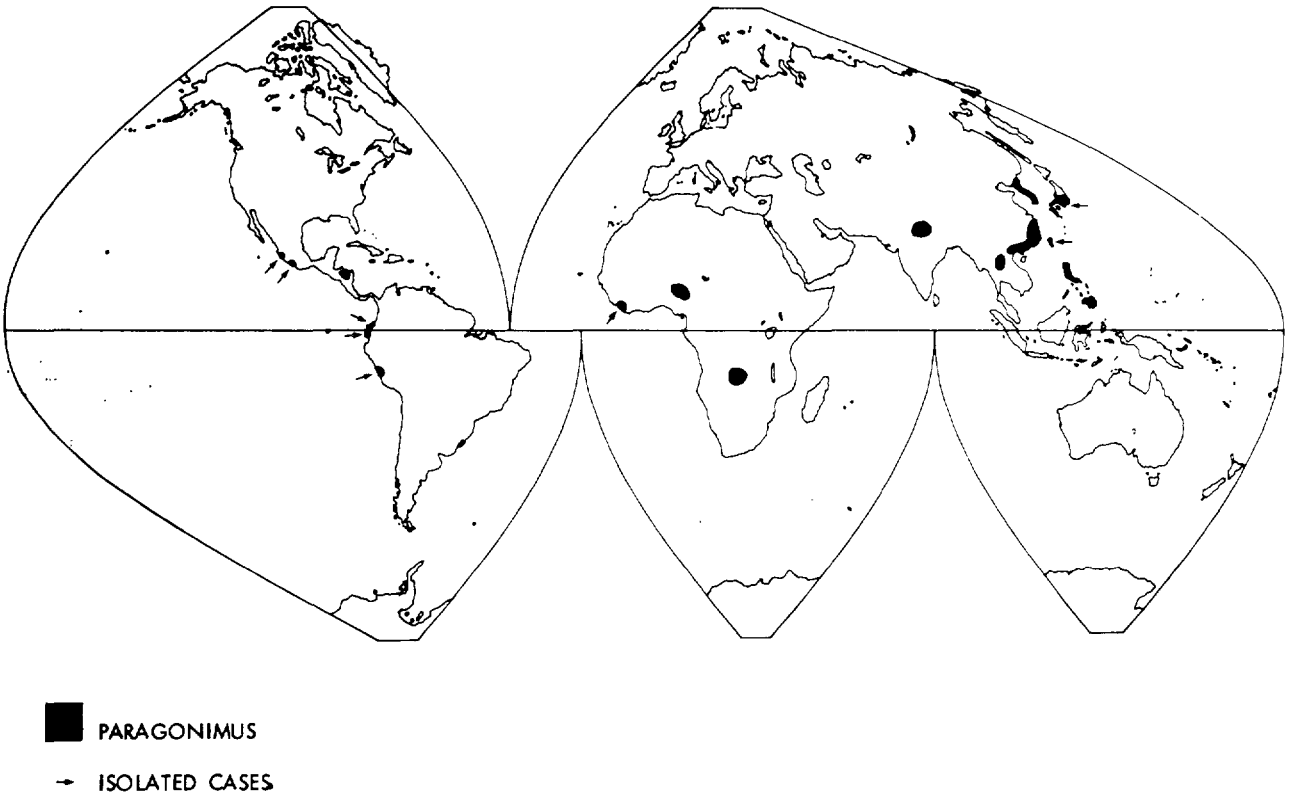


Figure 31-1. *Known geographical distribution of* Paragonimus. The infection may occur in areas as yet unrecorded

Figure 31-2. *An adult* Paragonimus westermani *under a light microscope.* Scale bar = 1 millimeter. (Photo: Wellcome Museum of Medical Science)

edible crab or crayfish. This second intermediate host lives mainly in fast-flowing mountain streams, although some species can be found in rivers and rice fields. The parasites form encysted metacercariae in the muscles or gills of the crustaceans and reach a new mammalian host when crabs or crayfish are eaten raw. The cysts hatch in the duodenum, and the young flukes migrate through the peritoneal cavity and diaphragm to the lungs, reaching maturity 5–6 weeks later and living for 6–20 years.

*Prepatent and incubation periods*

Worms reach maturity 5–6 weeks after encysted metacercariae in freshwater crustacea are ingested. Symptoms develop over longer and variable periods.

*Period of communicability*

The adult worm can live between 6–20 years and can produce eggs all this time, but worms normally become walled off after 1–2 years, and very few eggs are passed.

*Resistance*

Susceptibility is general. Increased resistance possibly develops as a result of infection.

*Epidemiology*

The distribution of paragonimiasis in man is determined by numerous factors—especially by the presence of particular snails and crustacean hosts in local streams and by dietary customs that include eating the crustaceans in a raw or semicooked state. Infection is not only associated with eating raw or pickled crabs and crayfish, but also with eating crab or crayfish juices, which are popular in parts of China, Korea, the Philippines and elsewhere. The encysted metacercariae are sticky and can contaminate knives, chopping boards, hands, and vegetables in the kitchen while crabs or crayfish are being prepared. As with clonorchiasis, infection is generally more common in adults than in children and in males than in females. This is due to different dietary customs.

In some endemic areas the crab and crayfish hosts live in mountain streams that are some distance from human settlement. It is likely that they become infected chiefly as a consequence of the contamination of these streams by animal, rather than human, feces. In such situations the eggs passed in human feces may be unimportant in maintaining transmission; therefore excreta disposal programs are irrelevant as an initial control strategy.

Major reviews of paragonimiasis include those by Yokogawa (1964, 1969) and Yokagawa, Cort and Yokogawa (1960). Other accounts of paragonimiasis include those from Africa (Nwokolo 1974), Cameroon (Sachs and Voelker 1975; Sirol, Kerfelec and Papinutto 1967), China (Chung and others 1975; Fan, Zihe and Daixia 1976), Costa Rica (Miyazaki 1974), Indonesia (Kwo and Miyazaki 1968), Ivory Coast (Nozais and others 1980), Japan (Katamine and others 1970, 1972; Sano and others 1979; Yoshida 1916), Laos (Soh 1973), Liberia (Voelker 1973), Malaysia (Miyazaki and Kwo 1969; Rohde 1967), Mexico (Martínez-Báez 1970), Nigeria (Nwokolo 1972; Voelker and Nwokolo 1973), North America (Ameel 1934), Panama (Miyazaki 1972), Peru (Miyazaki and Grados 1972), Philippines (Cabrera 1973; Cabrera and Fevidal 1974; Cabrera and Vajrasthira 1972, 1973), South Korea (Kim and Bang 1974; Rim and others 1975; Sadun and Buck 1960; Yun and others 1966), Sri Lanka (Kannangara and Karunaratne 1969), Taiwan (Huang and Chiu 1966; Huang and others 1966; Liu 1970; Liu and Cross 1971), and Thailand (Miyazaki and Vajrasthira 1967).

## Control Measures

Mass chemotherapy with bithionol has been effective in Japan and the Philippines.

In areas where the zoonotic reservoir is more important in maintaining transmission than the human reservoir (because it is primarily animal feces that reach the stream where the intermediate hosts reside), excreta disposal programs may not greatly reduce transmission. In areas, such as parts of Japan, where the human reservoir is important in maintaining transmission, any measures that prevent untreated human excreta from reaching surface waters should reduce the prevalence of infection in snails and may reduce infection in crabs and humans.

Another important approach to paragonimiasis control is to attempt to change human habits of consuming raw or insufficiently cooked crabs and crayfish. Infection often occurs when uncooked soft parts (such as leg muscles) are eaten raw. Pickling in brine, vinegar, or wine will not kill the encysted metacercariae, but heating for 10 minutes in water at 55°C is effective. Educational campaigns should inform the public of the danger of eating raw or insufficiently cooked crabs and crayfish and explain the possibility of infection through contamination of kitchen utensils while preparing infected crabs.

## Occurrence and Survival in the Environment

*Paragonimus* eggs develop most rapidly in water at 28–32°C, and miracidia hatch out in about 3 weeks. Eggs are quickly killed by freezing but survive for long periods at 4°C. Eggs do not develop at temperatures above 35°C and are rapidly killed by desiccation (Yokogawa, Cort and Yokogawa 1960). *Paragonimus* eggs in a biogas plant in China survived for 13 days (Hou and others 1959).

Encysted metacercariae can live for one week in the tissue of dead crabs at cool temperatures. Traditional methods of preparing crabs—such as soaking in a weak solution of salt, rice wine and spices–do not kill all metacercariae (Khaw 1935). Chemical treatments are also not reliable (Tsuda 1959). Metacercariae are rapidly killed above 50°C, and so most techniques of cooking, or dipping in boiling water, should destroy encysted larvae in the tissues of crabs and crayfish.

## Inactivation by Sewage Treatment Processes

No specific data are reported.

## Inactivation by Night Soil and Sludge Treatment Processes

No specific data are reported.

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# 32

# *Schistosoma* and Schistosomiasis

SCHISTOMIASIS is one of the major parasitic diseases of man. It affects many countries and, especially in Africa, it has increased in importance following the development of manmade lakes and irrigation schemes.

## Description of Pathogen and Disease

The literature on schistosomiasis is truly voluminous, and only a brief summary of the salient features can be given here. A series of major abstracted bibliographies, most recently Hoffman and Warren (1978), provides an invaluable guide to the literature on schistosomiasis for those wishing to study particular aspects in greater detail.

### *Identification*

Schistosomiasis, known in the older literature and in common usage as bilharziasis, comprises infections of the venous system by several species of the trematode genus *Schistosoma*. One species, *S. haematobium*, inhabits the veins around the bladder (urinary schistosomiasis), whereas the others predominantly involve the portal venous system that transports blood from the intestines to the liver (intestinal schistosomiasis). The most important species of intestinal schistosome are *S. mansoni* and *S. japonicum*; *S. intercalatum* is similar to these but has a localized distribution in West Africa.

The range of disease produced in infected individuals is very great. Infection is through the skin and may be accompanied by itching and skin inflammation. Early development in the lungs may give rise to marked fever and respiratory symptoms. The adult worms in the veins give rise to few disorders; the problems arise from the eggs, of which each female worm lays hundreds, or thousands in the case of *S. japonicum*, daily. A proportion of these escape into the bowel or bladder and are responsible for transmission to other persons as well as for damaging the tissues through which they pass. The majority of eggs are retained in the body, either in the bowel and bladder wall, or are carried to the liver, where many become stuck in the blood vessels, or to the lungs and even the brain or spinal cord on occasion. An impacted egg induces a chronic inflammatory response around it; the size of reaction depends in part on acquired immune responses. Gradually the egg becomes calcified (especially in the bladder wall) or destroyed.

Escaping eggs cause tissue damage with loss of blood and protein into the urine (where it is obvious) or feces. The heavier the infection, with more eggs being passed, the greater the blood loss, and up to thousands of worm pairs have been found at autopsy. With an inflamed bladder wall, caused by the passage of *S. haematobium* eggs, urine is passed frequently and painfully. Growths in the bladder lining may occur, and they or the schistosome eggs act as nuclei for the formation of urinary stones. In a few people infected with *S. haematobium*, bladder wall damage leads on to cancer and death. Reactions to retained eggs may block the escape of urine from the kidneys to the bladder in up to 20 percent of infected children, and the resulting back pressure may damage or even destroy the kidney. Where this is bilateral, renal failure and death follow, but the ureteric lesions of a fair proportion of patients are reversible with chemotherapy.

The intestinal schistosomiases (*S. mansoni* and *S. japonicum*) cause occult bleeding into the bowel, papillomata of the bowel wall, and, in heavy infections, bouts of dysentery with passage of blood. The brunt of damage falls on the liver, and although all cases have scattered reactions to impacted eggs, in a proportion (usually small but reaching 23 percent in one community) there is a proliferation of fibrous tissue to produce a fibrous liver. A fibrous liver may function badly, leading to hepatic coma, but more usually the main effect is a back pressure on the blood supply, with

great enlargement of the spleen and a series of bypasses developing that return blood to the heart other than through the liver. Such blood vessels above the stomach may burst and give rise to profuse bleeding from the mouth, which may be lethal. Yet other eggs may reach and damage the lungs or the nervous system, but a swollen abdomen with ascites from the liver damage is more frequent. These life-threatening complications occur mostly in the heavily infected, but even light infections give rise to lassitude in many patients. Others may have few or no symptoms, although even some of these, if heavily infected, have decreased ability to do physical work (Awad El Karim and others 1981) and impaired growth in childhood.

Diagnosis is by the identification of *Schistosoma* eggs in the feces (for *S. mansoni* and *S. japonicum* infections) or in the urine (for *S. haematobium* infections). Serological techniques for diagnosis are also available and are useful in mass surveillance in support of control programs (see, for instance, McLaren and others 1979). Treatment of infections is by drug therapy, and great advances in drug development have been made in recent years. Hycanthone or oxamniquine are often used for *S. mansoni* infections; hycanthone, niridazole, or metrifonate for *S. haematobium*; and niridazole for *S. japonicum*. Praziquantel, a newer drug still undergoing field trials, is effective against all three schistosome species.

### *Occurrence*

Human schistosomiasis is found in many parts of the tropics, with some 200 million cases in all. Unlike most other infections, it has been steadily spreading and increasing in intensity over much of its range for some decades, as a result of water impoundments for power and agriculture and the development of irrigated farming. Urinary schistosomiasis (due to *S. haematobium*) occurs throughout the inhabited parts of Africa and is particularly common in the Nile valley. It extends into irrigated and other parts of the Middle East, with small foci in South Asia and Europe (figure 32-1). *S. mansoni* is widespread in Africa and in Brazil and other countries of northeast South America, with a patchy distribution in the Caribbean (figure 32-2). Of the other intestinal schistosomiases, *S. intercalatum* has a restricted distribution in central West Africa, whereas *S. japonicum* occurs in the Philippines, Sulawesi, China, and other parts of Southeast Asia and was formerly important in Japan (figure 32-1). A closely related form is found along the Mekong River.

Within endemic areas the prevalence in schoolchildren of urinary schistosomiasis may often exceed 80 percent, and this level is reached at a later age for *S. mansoni*. Worm loads in a few people will be in the thousands, although the majority will be less heavily infected. The proportion going on to progressive disease will vary with intensity of infection. It may be locally up to 25 percent, but a lower proportion is usual, and of these only some will die of liver or urinary tract disease, most of them during early to middle adult life.

### *Infectious agents*

The schistosomes are digenetic trematodes in which the sexes are separate and differ in size and shape. The broader males are around 10 millimeters in length, and the slender female normally lies enclosed by the folded body of the male (figure 32-3). The several species that infect man are most readily distinguished by the shape of their eggs: those of *S. haematobium* and *S. intercalatum* have a terminal spine, whereas *S. mansoni* eggs have a lateral spine, and *S. japonicum* eggs are rounded with a small knob. Several more or less closely related blood flukes infect domestic animals and birds. The bird schistosomes can give rise to dermatitis in bathers in temperate climates.

### *Reservoirs*

Man is the effective reservoir of *S. haematobium* and *S. mansoni*. Though wild animals may become infected with *S. mansoni* and one small epidemic was traced to infected baboons, they may for practical purposes be disregarded. *S. japonicum* is a zoonosis, however, and in fact the Taiwan strain is noninfective to man and entirely transmitted between animals. Elsewhere, a variety of domestic animals—dogs, cattle, water buffaloes, and rats—act as reservoirs of infection, although man is still usually responsible for the majority of transmission, it is likely that it would continue, albeit at a lower level, in his absence.

### *Transmission*

Each paired female worm of *S. mansoni* and *S. haematobium* lays some hundreds of eggs daily, but *S. japonicum* lays thousands. The eggs are large, some 140 micrometers in length, and elongated. The proportion that escape, which may be as low as 20 percent, pass through the tissues into the urine (*S. haematobium*) or feces (other species). Although they take several days to

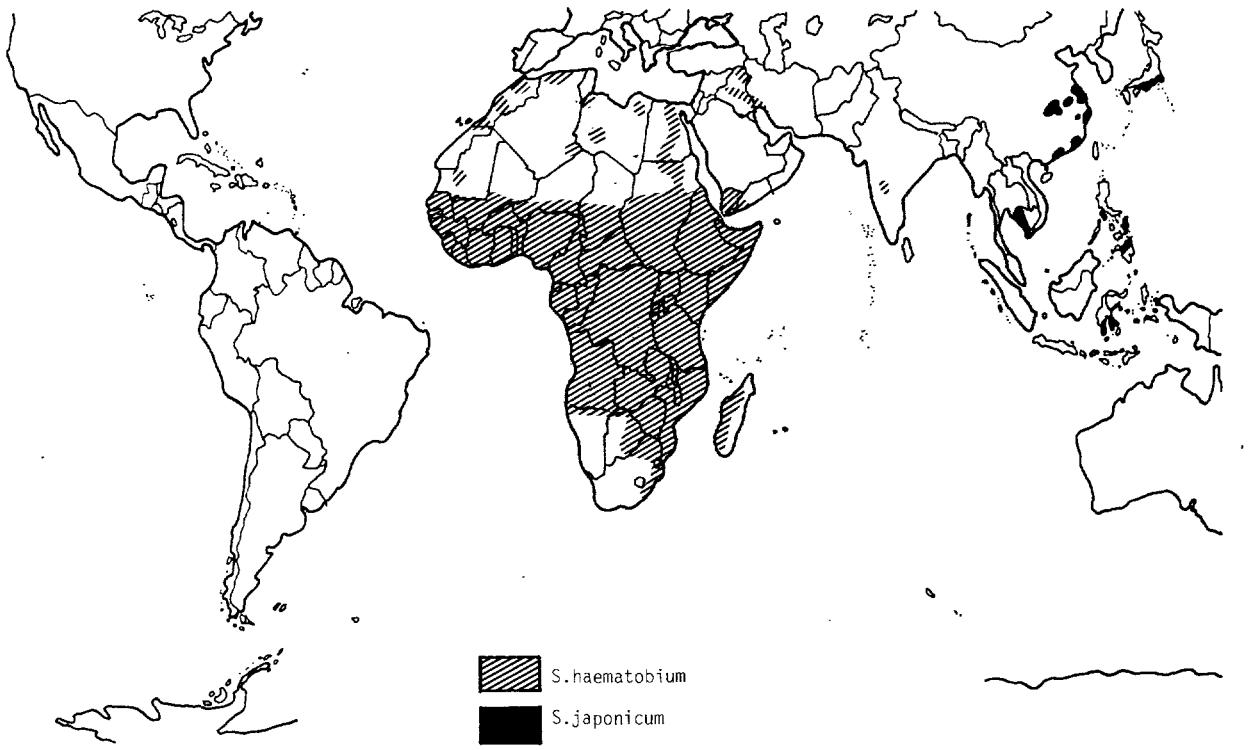


Figure 32-1. *Known geographical distribution of* Schistosoma haematobium *and* S. japonicum. The infections may occur in areas as yet unrecorded. *S. haematobium* transmission is most unlikely at altitudes above 1,500 meters

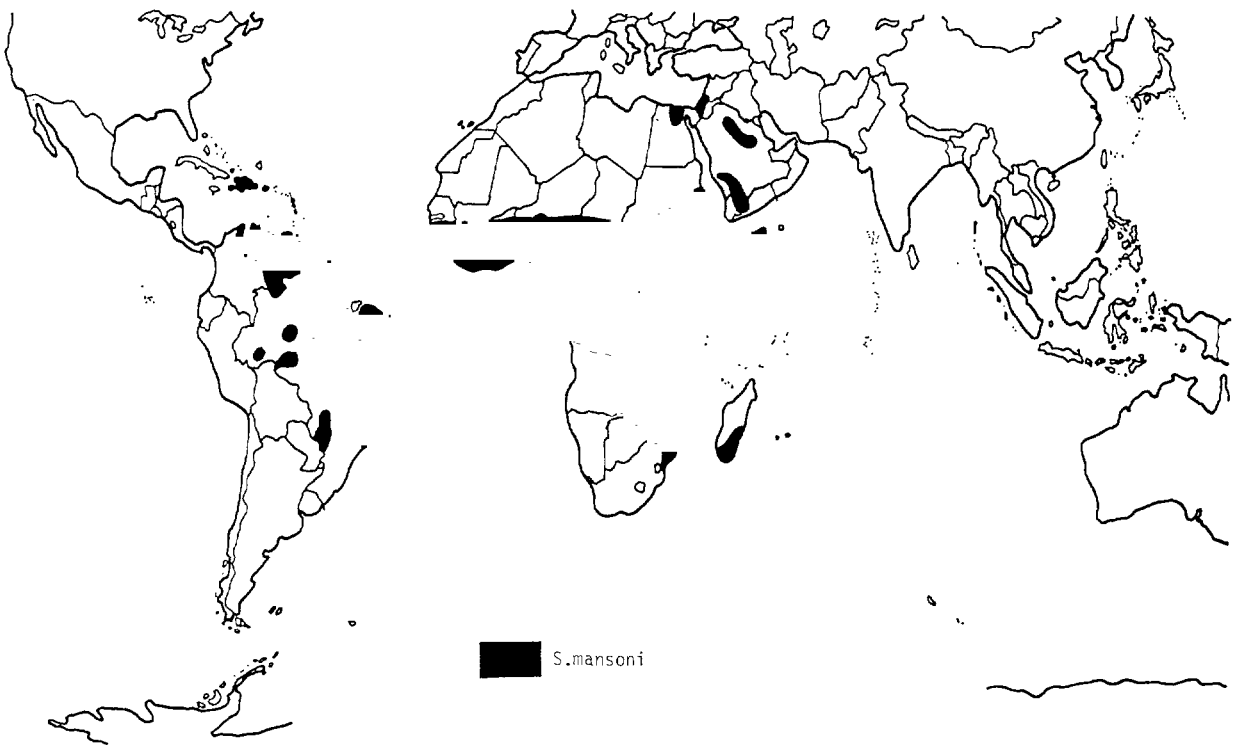


Figure 32-2. *Known geographical distribution of* S. manosni. The infection may occur in areas as yet unrecorded. *S. mansoni* transmission is most unlikely at altitudes above 2,000 meters

Figure 32-3. *A male and female* S. mansoni *under scanning electronmicroscopy.* The slender female (10–20 millimeters long and 0.16 millimeters wide) lies in a groove in the body of the sturdier male (6–12 millimeters long and 1.1 millimeters wide) and can be seen protruding at one end. Scale bar = 1 millimeter. (Photo: M. M. Wong, Primate Research Center, University of California, Davis, California, USA)

develop after being laid by the female, by the time they pass out of the body with the excreta they are mature and ready to hatch. After the worms have died or been killed by therapy, a few dead and calcified eggs may continue to be excreted over months or years, but these are not hatchable.

The eggs hatch when osmotic pressure of the surrounding medium falls, as when they reach water, and light and warmth speed hatching. The egg shell splits, and a motile ciliated larva called a miracidium emerges and scans the aquatic snail environment for up to 6 hours. On encountering a suitable species of aquatic snail, the larva penetrates it and undergoes a series of developmental stages. Between 1 month and 3 months later, depending on the temperature and on the species of snail, further aquatic larvae called cercariae begin to emerge from the snail. The pattern varies from less than a dozen each day for many months for *S. japonicum* in *Oncomelania quadrasi* to nearly a thousand daily from *S. mansoni* in *Biomphalaria glabrata*, though in the latter case the snail may only survive two weeks of shedding cercariae. These live up to 48 hours in the water, swimming to the surface and then slowly sinking during rest periods, but on encountering human skin they rapidly penetrate it and, after migrating to the lungs where they develop for some days, they move to the portal venous system of the liver to mature and pair before migrating to the intestinal and vesical blood vessels.

The snail hosts vary by region, and the schistosomes are very species specific. The principal genera of snails that act as host for the main species of schistosomes are:

| | |
|---|---|
| *Bulinus* | for *S. haematobium* |
| *Biomphalaria* | for *S. mansoni* |
| *Oncomelania* and *Tricula* | for *S. japonicum* |

The hosts of *S. japonicum* are amphibious and leave the water for muddy canal-banks from time to time. The snail hosts of other schistosomes are truly aquatic, although many, even if infected, can survive a dry season by burrowing into the drying mud of a seasonal pond. Still or gently flowing water favors the snails, as

does a high calcium content of water, neutral or alkaline pH, and aquatic vegetation. Where scattered families use separate ponds for water supplies the infection can be highly focal, but cercariae may be carried many meters in flowing water to infect downstream settlements.

Although people may become infected through the mucosa of the mouth when drinking water, the bulk of infections are acquired through water contact with the skin.

*Prepatent and incubation periods*

The shortest times recorded between cercarial penetration and the appearance of *S. mansoni* eggs in the excreta are just over a month, but 2 months is more usual, and a longer period is normal for the other schistosome species. An incubation period cannot be stated because symptoms may develop gradually, or not at all, depending on the number of schistosome worms infecting and the immune status of the host.

*Period of communicability*

Once mature worm pairs are established, they may persist and continue laying eggs for a long time, and worm survival for 30 years is documented. However, the majority of worms die sooner, and a half-life of 3 to 6 years is probable, though evidence of even shorter survival is appearing (for instance, Goddard and Jordan 1980). In endemic areas, the relative importances of prolonged worm survival and superinfection are not yet well defined. The eggs, once excreted, may persist for weeks or months, as discussed below, but hatch promptly in water and have only a few hours of life thereafter unless a snail is found.

*Resistance*

Most, if not all, people are susceptible to schistosomiasis, although some races may be more susceptible to the severer forms than others. Acquired resistance due to natural exposure to infection is well documented in several animal species and is clearly indicated to occur in human *S. haematobium* and *S. japonicum* infections. The evidence in *S. mansoni* is equivocal. Acquired immunity is certainly incomplete, and its importance in the natural history of the infections in communities is not well defined. Peak infection loads are usually seen around the age of 10 years in *S. haematobium* and a little later in *S. japonicum*; egg output declines thereafter even where water contact persists.

*Epidemiology*

The epidemiology of schistosomiasis is a complex and much studied subject, and space does not permit a full discussion here. In a given locality, schistosome dynamics depend upon both the macroecological effects of topography, hydrology, water quality, settlement patterns, agriculture, sanitation, human behavior, snail behavior, and the microecological factors of host-parasite relationships in man and in the snail. The total system is complex, and many gaps in scientific understanding remain, particularly on the role of immunity in natural infections. Attempts to construct mathematical models of transmission, and thus to predict the impact of alternate control strategies, have been mathematically sophisticated but of limited usefulness.

For successful transmission, man must live near to bodies of surface water that have the characteristics (temperature, chemistry, pH, plant life, velocity) necessary to support the appropriate species of snail. For transmission from man to snail to take place, fresh human excreta (urine for *S. haematobium* and feces for other species) must reach these bodies of water where the snail colonies are living. This may happen owing to promiscuous defecation, urination while near or in water, or to the discharge of untreated sewage into water. Finally for transmission from snail to man to take place, there must be a pattern of behavior in the community that causes people to enter regularly those surface waters that harbor the snails and have been polluted by the excreta.

In most communities where schistosomiasis is endemic, prevalence and intensity of infection are highest in the 5–20 age group. This age group is likely to be heavily exposed during play or bathing and possibly also when performing household tasks such as collecting water, tending water buffalo, fishing, or helping a parent in the fields. Adults are exposed to infection while working in irrigated fields, fishing, collecting water, washing clothes, bathing, or any other activity involving water contact.

Different patterns of work may cause differences in schistosomiasis prevalence. Thus, in a village in the Nile Delta (Egypt) women who consistently worked in the fields had a schistosomiasis prevalence similar to men, whereas women who worked exclusively around the home had an appreciably lower rate of infection (Abdel-Wahab and others 1980). Another study in Egypt (Farooq and others 1966) showed that, for males, there was an increased prevalence of *S. mansoni* among fishermen, water carriers, and washermen and an increase in *S. haematobium* infection among boatmen.

The transmission of schistosomiasis can be highly localized both in time and space. Regular seasonal variations can be due to temperature if there is a winter period when snail populations decrease and it is too cold for schistosomes to develop outside their human hosts. Seasonality of transmission can also be due to rainfall and surface water hydrology. High rainfall may wash out snail habitats or swell rivers to a point where water contact is reduced and cercarial concentrations are greatly diluted. More usually, dry periods eliminate ponds and streams completely. It is common in Africa for *S. haematobium* transmission to take place in ponds and waterholes in the wetter seasons, reaching a peak as water bodies shrink and man-water contact becomes more focal in the early dry season, but ceasing later when the ponds dry up and the snails estivate in the mud and await the next rains.

Focal transmission occurs if it is associated not with surface water bodies in general but with certain specific streams or ponds that are polluted by excreta, that support the correct snail species, and that are visited by people for play or work. Thus, as mentioned above, *S. haematobium* transmission may largely take place at one or two sites in or near a village. The correct identification of these sites is essential to the design of control programs.

Two aspects of human behavior are integrally linked with the epidemiology of schistosomiasis: water-contact behavior and excretion behavior. Water-contact behavior has been increasingly studied over the past decade, and these studies are a rare and encouraging example of the value of collaboration between sociologists and epidemiologists.

A water-contact study, in an area of northern Nigeria heavily infected with *S. haematobium*, showed that most contact with water at a dam site took place during fishing, bathing, swimming, and playing (Tayo, Pugh and Bradley 1980). The great majority (94 percent) of all water contacts observed involved males, because females were relatively secluded in this Muslim society. Schistosomiasis prevalences were much higher among males than females in the area. Peak water-contact activity occurred in the afternoon: the time of peak urinary egg output and peak cercarial shedding by infected snails.

Water-contact studies on the shore of Lake Volta (Ghana), another *S. haematobium* area, showed that women were most exposed during domestic tasks such as water fetching and clothes washing and that men were most exposed during swimming, bathing, and entering canoes (Dalton and Pole 1978). Overall, males had more water contact and higher intensities of schistosomiasis infection than females. It is pointed out that piped water supplies and clothes washing facilities in the villages might reduce water contact for females, but would not affect the recreational activities of males or the time they spend around canoes in the shallow water at the lake edge.

Excretion behavior studies are more difficult to carry out than water-contact studies, and there is, as yet, little information on this important aspect of schistosomiasis epidemiology. Studies in the Gezira irrigation scheme in the Sudan by Cheesmond and Fenwick (1981) found that 46 percent of all observed acts of excretion took place before 9.00 AM. Men and women squatted in the open to urinate, although women did not urinate in sight of men. Nearly all observed urination was onto soil, not into water, although children may have urinated unseen while immersed in water. Privacy was the prime determinant of defecation site. 93 percent of defecations took place in the fields in cotton, sorghum, or among trees. Only 31 percent of people washed themselves after excretion and only 7 percent washed their anal region directly into a water body. This study, unlike some others, found that privacy was more important than closeness to water in determining defecation sites and that most observed excretion could not lead to the entry of schistosome eggs from urine or feces into canals or other water bodies.

A study of excretion behavior was carried out in the Nile Delta (Egypt; Farooq and Mallah 1966). Children under 10 years played frequently in water and often urinated while doing so. Both sexes and most ages usually urinated and defecated within 2 meters of water. Boys commonly urinated directly into water. Adult males performed ablution after defecation by squatting close to the water's edge and splashing water and washing with the left hand. Adult females who defecated in the open did so early in the morning or after sunset and did not perform ablution. Although females had more frequent water contact than males, males were observed to contaminate water 5 times more frequently than females, and those girls polluting were mostly under the age of frequent schistosomal infection.

The contamination of surface waters by excreta, and the subsequent infection of snails, occur not only as a result of promiscuous defecation but also because of the discharge of inadequately treated sewage. In Minas Gerais (Brazil) the effluent from a septic tank was entering a stream, and 65 percent of *Biomphalaria glabrata* less than 10 meters from the sewage outfall, 15 percent of those snails between 90 and 100 meters from the outfall, and no snails 200 meters away were infected (Paulini 1964). In addition, snail density was much

greater near the outfall because some snail species have a preference for polluted waters (see also Watson 1958). A similar situation in South Africa is reported by Bayer (1954).

Although schistosomiasis is primarily a rural disease, urban communities are also infected. Transmission may take place in urban streams, borrow pits, or ponds, or it may be due to urban people leaving town for recreational or agricultural purposes and becoming infected in the countryside. A survey of residents in San Juan (Puerto Rico) showed that swimming and fishing were the main reasons for water contact and that these activities took place in streams throughout the island. Most water contact involved the 5–19 age group but was not associated with socioeconomic status (Lipes and Hiatt 1977). In a working class suburb of São Paulo (Brazil), where thirty locally acquired infections of children with *S. mansoni* and three infected *Biomphalaria tenagophila* were detected, the sites of transmission were two borrow pits used as communal bathing pools, chiefly by children (Rodriques and Ferreira 1966).

Schistosomiasis is closely related to surface-water hydrology and irrigated agriculture and is therefore sensitive to the development of manmade lakes and irrigation schemes. The typical experience has been that major irrigation and lake development projects in areas of endemic schistosomiasis have increased the prevalence and intensity of this infection among the local population. There are well documented cases of this from Egypt, Ghana, Iran, Nigeria, Sudan, Tanzania, Zambia, Zimbabwe, and elsewhere (Rosenfield and Bower 1979). The development of the lakes and the irrigation and drainage canals increases the number of habitats for snails; the development of irrigated agriculture and fishing increase the frequency and duration of water contact; the increased availability of surface water for recreation also increases water contact; population densities rise to take advantage of the new agroeconomic opportunities; and the fecal contamination of the surface water is assured by the general poverty of the local communities and the lack of concurrent sanitation programs. All these factors contribute to rising transmission rates leading to rising infection rates and, ultimately, to more frank disease.

In some areas, the ecological changes caused by lake and irrigation development may not only increase schistosomiasis transmission but may also affect the type of schistosomiasis that is dominant. Thus, in some villages in the Nile Delta (Egypt) the major changes in hydrology and agriculture that have followed from the construction of the Aswan High Dam have been associated with a rise in *S. mansoni* prevalence and a fall in *S. haematobium* prevalence (Abdel-Wahab and others 1979).

## Control Measures

Schistosomiasis control is at present in a state of flux. During the 1960s the only control method shown to be effective was application of molluscicides to host snails, but more recently control programs have used several methods simultaneously and at high cost. Chemotherapy is likely to play an increasing role in the future.

### *Individual*

Until recently, there was no drug suitable for mass chemotherapy on a large scale. The antimonial compounds used to treat schistosomiasis were toxic and required repeated intravenous or intramuscular injections spread over up to a month. Now there are several oral schistosomicides available, and others are undergoing trial. Metrifonate, only active against *S. haematobium*, causes negligible side effects and costs little. Two or three spaced doses are required. A single dose of oxamniquine treats *S. mansoni*, has few side effects, but is expensive. The chemotherapy of *S. japonicum* is unsatisfactory. A long course of niridazole is needed, with indifferent cure rates, but a very promising drug (praziquantel) is under field trial. Mass chemotherapy can now reduce prevalence and intensity of infection greatly; the duration of the reduction is limited (Costa, Katz and Dias 1980), and accompanying transmission control measures are necessary. Advantages of chemotherapy are its immediate effects on worm load and the disease in man. Cost is the chief defect, except for *S. haematobium*, and the cooperation of the population may be difficult to sustain in the long term.

### *Environmental*

The intermediate host snails may be controlled either by rendering the habitat unfavorable to them or by the use of molluscicides. Environmental control has been most dramatically used against hosts of *S. japonicum*: in Japan, where irrigation canals were lined with concrete; in China, where labor-intensive methods of resiting canals and burial of the snails in the

old canal were used; and in the Philippines, where it was shown that improved methods of irrigated field management both raised the production of rice and reduced the host snail populations in the fields. For the aquatic snail hosts of other schistosomes, such measures as channel straightening, weed clearance, and intermittent drying out of irrigation canals and drains have limited snail numbers. Biological control of snail populations by competitor species of snails has been shown in small specialized habitats and is claimed to have a significant effect on a larger scale, but as an operational control method has been little used.

Molluscicides have a long history, beginning with copper sulphate, but niclosamide (Bayluscide) and *n*-trityl-morpholine (Frescon) are the only ones in operational use now (McCullough and others 1980). They are relatively nontoxic to man, although they may harm fish and nontarget invertebrates. To achieve good snail kills the dosage needs careful control, and this can best be achieved where irrigation flows are appropriately managed. There is, as yet, no clear evidence of snail resistance to Bayluscide, even after prolonged application (Barnish and Prentice 1981). Although molluscicide can stop transmission, the long survival of adult worms in man implies continuing the program for many years to maintain control or combining the molluscicide program with mass chemotherapy and improvements in water supply and sanitation (Hiatt and others 1980). Apart from altering snail habitats, environmental approaches to control consist either of preventing infected excreta from reaching the snails or of preventing human contact with infected water. Two control programs (in Brazil and St. Lucia), which provided water supplies, bathing or laundry facilities, and attempted to reduce infected water contact, have reduced transmission considerably, whereas other successful programs have included water supply and sanitation (Barbosa, Pinto and Souza 1971; Jordan 1977; Jordan and others 1978; Jordan and Unrau 1978).

The few recorded attempts to control schistosomiasis by providing excreta disposal facilities alone have been unsuccessful (Scott and Barlow 1938). This has been ascribed to people's failure to use the facilities because they were wrongly located — in villages, while defecation took place in the fields — or offensive, or ill-adapted to the cultural tradition or to use by children. One epidemiological model of schistosomiasis transmission (Macdonald 1965) has been interpreted as showing that excreta management is inefficient as a control method, even when latrines are used. However, this results more from the structure and assumptions of the model, rather than being a robust conclusion. Empirical testing of excreta disposal, when facilities are used, as a sole means of schistosome control is lacking. In general, one would doubt its efficacy, but as a concomitant measure with mass chemotherapy to prevent rapid build-up of the worm population after treatment it may have value, provided that those who "escape" the treatment regimen are not those who also fail to use excreta disposal facilities. Children below school age, and males who have recently left school and become migrants in search of work, are groups of particular concern.

Three factors mitigate against the efficacy of improved sanitation in schistosomiasis control. First, a single stool or urination may contain many eggs, and a single miracidium reaching a snail may give rise to several thousand cercariae. Therefore, the contamination of surface waters by excreta may have to be reduced to an extremely low level. Second, although it is possible to improve defecation behavior in some communities and to reduce the contamination of the environment by feces, it is very difficult to modify urination behavior. Therefore, the impact of sanitation programs on *S. haematobium* is likely to be markedly less than their impact on *S. mansoni* and *S. japonicum*. In addition, *S. haematobium* egg output is at its maximum in the early afternoon (Stimmel and Scott 1956), a time when children are likely to be playing in water and adults to be working in water. Third, those people in the community excreting most eggs are in the age group of 5–20 years. This group is likely to be less affected by sanitation programs than adults.

In summary, schistosomiasis control depends upon a carefully designed mix of chemotherapy, snail control, water supply, sanitation, and health education (Sandbach 1975; WHO 1980). The nature of this mix will be different in different places and must arise from detailed study of the local epidemiology of schistosomiasis. Some bizarre control strategies have been suggested, such as the maintenance of crocodile and hippopotamus populations in Lake Sibaya (Natal, South Africa) to discourage water contact (Appleton and Bruton 1979).

Some poor countries have achieved notable progress in schistosomiasis control by the sustained application of integrated control measures and the mobilization of popular support and participation. An example is China (Anon. 1977; Cheng 1971; Chung 1977; Sandbach 1977). Other countries have achieved substantial levels of control by specific antischistosome measures in the context of rising incomes and improved socioeconomic conditions. Examples are Japan, Puerto Rico, and Venezuela (Bhajan and others 1978; Negrón-Aponte and Jobin 1979; WHO 1973).

## Occurrence and Survival in the Environment

The stages of schistosomes found in the environment are eggs, miracidia, and cercariae. Schistosome eggs are considerably less rugged and long lived than those of *Ascaris*, *Trichuris*, or *Taenia* worms. Schistosome miracidia and cercariae are fragile and must find a snail or vertebrate host within hours or they die.

### *In water*

Schistosome eggs hatch rapidly on reaching water, and light and warmth speed hatching (Faust and Hoffman 1934; Maldonado, Acosta Matienzo and Vélez Herrera 1950; Miyairi and Suzuki 1913; Standen 1951). *S. japonicum* eggs will not hatch at temperatures below 3°C or above 38°C, with temperatures of 13–28°C being most suitable for hatching (Ito 1953). Standen (1951) found the *S. mansoni* eggs had an optimal temperature for hatching of 28°C. Hatching of *S. mansoni* eggs is reduced at salinities as low as 0.05 percent and ceases completely at 0.6 percent (Standen 1951). *S. mansoni* egg hatching is also inhibited by low dissolved oxygen levels (Kawata and Krusé 1966).

Miracidia swim in the water; if they come close to a snail, they are attracted to it and penetrate. If they do not encounter a snail, they may live for up to 3 days but are probably unable to penetrate a snail after a few hours (Faust and Meleney 1924; Miyairi and Suzuki 1913; Porter 1938). Experiments on *S. douthitti* miracidia showed that mean longevity falls with increasing temperature, from 11 hours at 8°C, to 7 hours at 20°C, and 1.5 hours at 35°C (Farley 1962). Kawata and Krusé (1966) found that *S. mansoni* miracidia in water at 26°C survived for up to 18 hours, with a mean of 6 hours. Miracidial survival is enhanced at pH values of around 7.5.

Schistosome cercariae are shed from the snail into the water and must find an appropriate vertebrate host and penetrate. Cercarial survival in water seldom exceeds 2 days and is temperature dependent. *S. japonicum* cercariae survive for over 7 days at 5°C and under 4 hours at 40°C (Jones and Brady 1947). As the length of time in the water increases, the ability of a cercaria to penetrate decreases, and it is probable that nearly all cercariae in warm tropical waters lose their ability to infect after less than 24 hours. *S. japonicum* cercariae tolerate pH in the range 5.5 to 8.4 (Jones and Brady 1947).

Schistosome cercariae are readily removed from drinking water by chlorination (Coles and Mann 1971; Frick and Hillyer 1965; Wittenberg and Yofe 1938) or by storage for 2 days.

### *In feces and night soil*

The survival of *S. mansoni* and *S. japonicum* eggs in feces is of epidemiological importance. *S. mansoni* eggs in feces in South Africa all survived for 3 days, only half were hatchable after 6 days, and none after 8 days (Porter 1938). Experiments in Puerto Rico showed *S. mansoni* eggs survived for over 2 days in formed feces, but only 1 day in liquid feces, at 24–32°C. In formed feces at 7–10°C, survival was for over 7 days (Faust and Hoffman 1934).

*S. japonicum* eggs in feces may survive for longer than *S. mansoni* eggs. Early studies in Japan found that *S. japonicum* eggs in cow dung survived for up to 2–4 weeks (Miyairi and Suzuki 1913). Subsequent studies in Japan showed that *S. japonicum* eggs in wet rabbit feces survived for 20 days at 28°C, 113 days at 18°C, and 180 days at 8°C (Ito 1954*b*) *S. japonicum* eggs in the anaerobic fecal liquor of a biogas plant in China survived for up to 14 days in summer, 22 days in autumn, and 37 days in winter (McGarry and Stainforth 1978). The addition of urine to the feces, or drying to a moisture content of 5 percent, greatly reduces *S. japonicum* egg survival (National Schistosomiasis Research Committee 1959).

### *In urine*

For *S. haematobium* eggs it is survival in urine, rather than feces, that affects transmission. Studies on *S. haematobium* eggs in urine at room temperature in South Africa showed that 60 percent were hatchable after 2 days, 10 percent after 3 days, 4 percent after 5 days, and none after 8 days (Porter 1938). Survival times were prolonged at cooler temperatures. Ito (1954*b*) studied *S. japonicum* eggs in urine. At 28°C they were unhatchable within a day in rabbit urine, 2 days in cow urine, and 3 days in human urine. Survival times doubled at 18°C and quadrupled at 8°C.

### *In sewage*

Schistosomiasis is primarily an infection of poor people in rural areas, and such people typically produce no sewage. There are, however, some urban communities with flush toilets and sewerage systems where schistosomiasis prevalences are high enough to cause a detectable level of schistosome eggs in the sewage. An example was San Juan (Puerto Rico), where raw sewage contained 2 *S. mansoni* eggs per liter (Rowan and Gram 1959). Jones and others (1947) reported that *S. japonicum* eggs would not hatch in raw or settled sewage, but would hatch in raw sewage diluted to one-quarter strength in water or settled

sewage diluted to one-third strength. Sewage with a low oxygen content seriously inhibited hatching and reduced the viability of *S. mansoni* eggs (Kawata and Krusé 1966).

## Inactivation by Sewage Treatment Processes

Schistosome egg removal in sewage treatment processes has been little studied but is similar to *Ascaris* egg removal (chapter 23). The major difference is that many schistosome eggs will hatch during sewage treatment, especially in well-aerated environments such as activated sludge tanks or maturation ponds. Hatching promotes schistosome removal because the released miracidium is far more vulnerable than the egg and must find a suitable snail within a few hours or die.

Laboratory studies on *S. japonicum* eggs in sewage showed settling velocities of over 1 meter per hour for 73 percent of eggs (Jones and others 1947). Bench-scale trickling filter experiments showed higher removal of *S. japonicum* eggs at lower loading rates, and many eggs hatched during secondary sedimentation. Most eggs hatched after 24 hours aeration in a simulated activated sludge unit (Jones and others 1947).

Studies in Puerto Rico showed that 83 percent of *S. mansoni* eggs were removed during primary sedimentation and 99.5–100 percent by complete trickling filter or activated sludge plants (Rowan 1964*a*). It is possible that these very high removal rates were partly caused by some eggs hatching in the treatment plants and by the miracidia not being detected by the method used to detect eggs in the effluent. However large numbers of *Biomphalaria glabrata* snails were exposed for 3–6 hours to the plant effluents, but none became infected; although in an earlier study done at the same activated sludge plant when it was receiving a higher influent egg load *B. glabrata* snails did become infected by miracidia in the final effluent.

Schistosome eggs, miracidia, and cercariae should be completely removed by waste stabilization ponds. Laboratory experiments with *S. mansoni* eggs showed that hatching was inhibited, though not prevented, in anaerobic ponds and that hatching proceeded normally in facultative and maturation ponds. Miracidia survived for up to 6 hours (mean 2 hours) in an anaerobic pond and for up to 10 hours (mean 4 hours) in a maturation pond at 26°C. *Biomphalaria glabrata* snails survived for up to 42 days (mean 20 days) in an anaerobic pond and lived and reproduced normally in a maturation pond (Kawata and Krusé 1966).

Schistosome eggs entering a pond system will either die or hatch but will not be carried through to the effluent. Those that hatch will liberate miracidia that must find a suitable snail host rapidly or die. If the ponds are colonized by an appropriate snail species, some miracidia will encounter snails and penetrate. Subsequently numerous cercariae will be shed, but these must reach the outfall and find a human host within about 1 day. In a well-designed pond system, with an overall retention time of 15 or more days, cercariae will die long before they reach the outfall. Workers entering the ponds for maintenance purposes are at risk and need protective clothing.

Effluent chlorination to the level needed to have a satisfactory effect on excreted viruses (chapter 9) and bacteria (chapter 13) will also inactivate most schistosome eggs and all miracidia (Jones and Hummel 1947; Mercado-Burgos 1975; Rowan 1964*b*). Sand filtration of effluents will also remove schistosome eggs, but not all miracidia (Jones and others 1947; Newton, Figgat and Weibel 1948).

## Inactivation by Night Soil and Sludge Treatment Processes

As with all helminth eggs, schistosome eggs in sewage treatment processes become concentrated in the sludge. Schistosome eggs are not long lived in feces, sludge, or night soil compared with *Ascaris*, *Trichuris*, or *Taenia* eggs, and any process that removed these other worm eggs will guarantee schistosome egg destruction.

*S. japonicum* eggs did not survive 21 days in sludge at 16–24°C or 9 days at 29–32°C (Newton, Figgat and Weibel 1948). *S. japonicum* eggs in digesting sludge at 24–30°C survived less than 25 days (Jones and others 1947). Kawata and Krusé (1966) found that 91 percent of *S. mansoni* eggs would not hatch after only 4 hours in waste stabilization pond sludge at room temperature, again suggesting that *S. mansoni* eggs are considerably less robust in the environment than are *S. japonicum* eggs. Normal anaerobic sludge digestion processes should therefore eliminate schistosome eggs if operated on a batch basis.

Sludge drying processes do not normally achieve the very low moisture contents needed to kill schistosome eggs by desiccation. Three weeks of sludge drying in warm climates should eliminate schistosome eggs irrespective of the moisture content reached (Jones and others 1947).

Schistosome eggs are readily killed by heating and are therefore eliminated by well-managed thermophilic

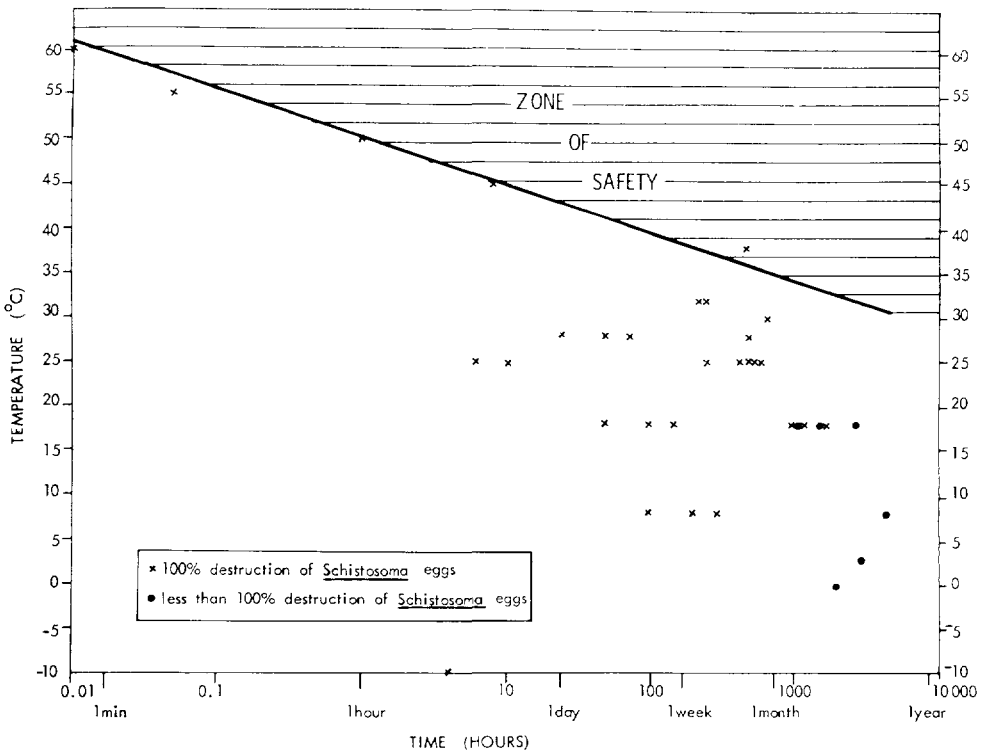


Figure 32-4. *The influence of time and temperature on* Schistosoma *eggs.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

composting processes. Data on the heat inactivation of schistosome eggs, from Ito (1954*a*) and other studies, are plotted on figure 32-4.

Work in China has shown that schistosome egg destruction in night soil may be promoted by the addition of urea, calcium cyanamide, or ammonium bicarbonate (Cheng 1971; National Schistosomiasis Research Committee 1959), and these add to the nitrogen value of the resulting fertilizer.

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# 33

# *Strongyloides* and Strongyloidiasis

SOME ASPECTS of the epidemiology and transmission of *Strongyloides* resemble those of the hookworms described in chapter 22. In other aspects, however, it is entirely different, and it must be considered quite separately from *Ancylostoma* and *Necator*.

## Description of Pathogen and Disease

The curious life cycle of *Strongyloides*, and the danger of very severe consequences of strongyloidiasis in immunodeficient or otherwise debilitated individuals, have generated considerable research interest in this worm. A recent review is provided by Carvalho Filho (1978).

### *Identification*

Strongyloidiasis is an infection of the small intestine by the nematode worm *Strongyloides stercoralis*. Symptoms are often vague or absent, but infection is potentially serious, particularly in malnourished or immunosuppressed individuals. Nonspecific symptoms such as diarrhea with abdominal discomfort, recurrent respiratory symptoms, and perhaps a rash are common. In a few patients, enteritis with a malabsorption syndrome, loss of elasticity in the gut, and emaciation occur. When the body's immune responses are deficient, disseminated strongyloidiasis may occur, with larvae attacking most organs of the body; such cases are usually fatal.

Diagnosis is by finding larvae in feces examined under a microscope. Clinically, watery diarrhea with mucus is suggestive of infection if accompanied by lesions on the buttocks, in the anal region, and an eosinophilia of up to 30 percent. Treatment is by oral drug therapy with thiabendazole or mebendazole.

### *Occurrence*

Strongyloidiasis occurs worldwide and particularly in warm, wet climates. In most areas strongyloidiasis is coextensive with hookworm but has a lower prevalence. It is probable that strongyloidiasis is everywhere more common than prevalence figures would indicate, since it is difficult to diagnose.

### *Infectious agent*

*Strongyloides stercoralis* is a minute nematode parasitizing man. The adult females are only 2–2.5 millimetres long and live embedded in the mucosa of the small intestine (figure 33-1). The eggs are ovoid and measure 50–60 by 30–35 micrometers but are seldom seen because larvae hatch out and are passed in the feces.

### *Reservoirs*

The reservoir of *S. stercoralis* is man, although dogs and apes have been found naturally infected.

Another species, *S. fuelleborni*, infects man in Cameroon, Central African Republic, Congo, Ethiopia, Malawi, Togo, Zaire, Zambia, Zimbabwe, and other African countries and in Papua New Guinea and West Irian (Indonesia). It is a common parasite of monkeys and baboons in Africa and Asia, probably also a natural parasite of man, and the predominant *Strongyloides* species infecting man in the rain-forest belt of Central Africa.

### *Transmission*

The mature parasitic female, which lives embedded in the mucosa of the small intestine, deposits several dozen partially embryonated eggs each day. These eggs hatch and liberate noninfective rhabditiform larvae that migrate into the lumen of the small intestine. The rhabditiform larvae either leave the host with the feces or develop into dwarfed filariform larvae that may invade the mucosa of the lower portion of the small intestine or large intestine and cause infection. The

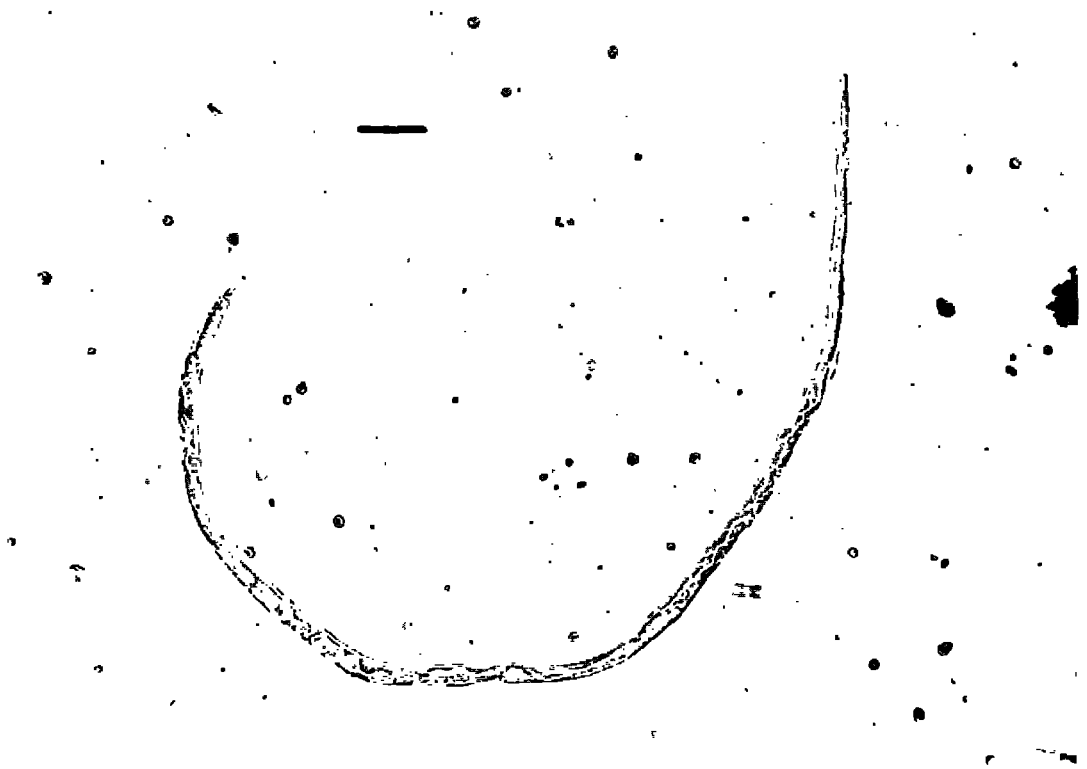

Figure 33-1. *An adult* Strongyloides stercoralis *under a light microscope.* Scale bar = 0.1 millimeters. (Photo: Wellcome Museum of Medical Science)

latter course is known as autoinfection. Autoinfection can also occur when rhabditiform larvae in feces are deposited on the perianal skin and develop into infective filariform larvae that penetrate the body through the skin. Thus, *Strongyloides* is the one human helminth that can increase its numbers within the intestine without any form of reinfection or external transmission.

The rhabditiform larvae passed in the feces continue their development in the soil. When conditions are favorable (high nutrient concentration and high moisture in soil), they develop into free-living adults. These adults continue a free-living life cycle in the soil as long as conditions allow. When conditions are unfavorable (low nutrient concentration and low soil moisture), the rhabditiform larvae develop into infective filariform larvae that can remain alive in moist soil for a few weeks. These infective filariform larvae penetrate the skin, usually of the foot, and are carried in the blood, through the heart, to the lungs. The larvae penetrate capillary walls around the alveoli (lung sacs), enter alveoli, ascend the bronchus and trachea to the epiglottis, and descend the digestive tract to reach the upper part of the small intestine where development of the adult parasite is completed.

A new infection can be initiated by the penetration of a single larva. An adult female worm can produce eggs without fertilization by a male worm (parthenogenesis), and male adults are rarely found in the intestine.

The transmission and life cycle of *S. fuelleborni* is similar to *S. stercoralis* except that eggs, rather than larvae, are shed in the feces.

### *Prepatent and incubation periods*

Worms become mature, and larvae appear in the feces, 17–28 days after skin penetration by filariform larvae. Symptoms may develop slowly or not at all.

### *Period of communicability*

As long as mature females are present in the small intestine, larvae will be passed in the feces. Because autoinfection is common, patients may pass larvae for many years. Larvae in soil live for less than 2 weeks, but free-living cycles can occur under favorable conditions to prolong contamination of soil for an unknown period.

*Resistance*

Susceptibility is general, but there is good evidence for limited immunity.

*Epidemiology*

The epidemiology of strongyloidiasis has been little studied, and most prevalance figures are probably underestimates because microscopic examination of feces for larvae detects only some infections. *Strongyloides* is usually, although not always, coextensive with hookworm infection. Promiscuous defecation, poverty (no shoes), and a wet humid climate are conditions favoring the transmission of both infections. Strongyloidiasis typically is rarer than hookworm infection, which is surprising in view of the greater egg production of the *Strongyloides* female and its free-living cycle, which may build up higher populations of infective larvae in the soil. Counter influences are that the *Strongyloides* rhabditiform larva in the feces is less rugged than the hookworm egg, and the infective filariform larva of *Strongyloides* is shorter lived than that of the hookworm.

Studies in Cali (Colombia) during 1956–61 showed an overall prevalence of strongyloidiasis of 14 percent, with a maximum of 30 percent among people 40–49 years old (Faust and Mugaburu 1965). As with hookworm, infection was more common in males than females. Other reports of strongyloidiasis in developing countries include those from Brazil (Asami, Enomoto and Miura 1970; Dias 1968), Cuba (Razón 1971), India (Nawalinski, Schad and Chowdhury 1978), Iran (Ghadirian and Amini 1970), and Tunisia (Dancesco and others 1971).

Strongyloidiasis is found in some areas of some industrialized countries. It occurs throughout the USA but is more common in the rural south (Blumenthal 1977; Burke 1978; Warren 1974). Infection rates are high in some institutions, especially in homes for the mentally retarded. Continuing autoinfection can maintain strongyloidiasis in individuals long after they leave endemic areas, and these infections may become serious and even fatal if resistance is lowered by other diseases or by certain types of drug therapy (Weller, Copland and Gabriel 1981). It has recently been shown in Britain (Gill and Bell 1980) and Australia (Grove 1980) that up to 28 percent of former prisoners of war of the Japanese have chronic strongyloidiasis, even 40 years after their supposed infection in eastern Asia. For British ex-prisoners, strongyloidiasis is significantly more common among those who worked on the Thai-Burma railway (21 percent) than among those who did not (9 percent). Other accounts of strongyloidiasis in developed countries include those from Japan (Tanaka 1968), Poland (Soroczan 1976), the USSR (Shablovskaya 1964; Stefanov 1970), and Yugoslavia (Bezjak and Breitenfeld 1969).

Accounts of *S. fuelleborni* infection in man include those from Papua New Guinea (Vince and others 1979), Zambia (Hira and Patel 1980), and elsewhere in central Africa (Pampiglione and Ricciardi 1971, 1972).

## Control Measures

Mass chemotherapy is an ingredient of any control program, but it is hampered by the limited effectiveness and possible side effects of the drugs available.

Environmental and educational control measures are similar to those for hookworm (chapter 22) and must emphasize excreta disposal, excreta treatment prior to agricultural application, and the wearing of shoes. The free-living *Strongyloides* cycle and the risk of continuing autoinfection, however, suggest that environmental and educational measures will be less effective against *Strongyloides* than against hookworm.

Successful reduction of strongyloidiasis in mental institutions by a combination of chemotherapy and environmental improvement has been reported from Hungary (Bánki and others 1963), the USA (Jeffery 1960), and the USSR (Shablovskaya and Smaga 1967).

There has been little work on the control of endemic strongyloidiasis in villages in developing countries. An exceptional report comes from Costa Rica (Arguedas and others 1975). In 1965, the inhabitants of the village of Palomo were given mass treatment with thiabendazole in a dose of 75 milligrams per kilogram, repeated after 6 weeks. A neighboring village, Purisil, was used as a control. For the first 2 years there was a dramatic drop in the prevalence of strongyloidiasis in Palomo from 19 to 1 percent, whereas Purisil showed a drop only from 18 to 10 percent. Hookworm, *Trichuris*, and *Ascaris* infection rates were not improved after 2 years. Seven years after the mass treatment, another survey was done in 1973. Meanwhile, sanitary and socioeconomic conditions had improved: latrines, piped water, and electricity had been installed. The prevalence of *Strongyloides* had dropped to 6 percent in Purisil but remained very low at 0.5 percent in Palomo. These findings suggest that mass treatment with thiabendazole, combined with concurrent social and environmental improvements, had a considerable long-term effect.

## Occurrence and Survival in the Environment

The eggs of *S. stercoralis* are not found in the environment, although those of *S. fuelleborni* are. The larvae of *S. stercoralis* may be expected in night soil and sewage in endemic areas and have been reported from sewage in the German Democratic Republic (Kalbe 1956).

*S. stercoralis* larvae typically live for less than 3 weeks, even in soil under optimal conditions. The optimal conditions for the infective filariform larvae are 20–25°C and high moisture. Larvae die rapidly in dry soil or at temperatures of over 46°C. Rhabditiform larvae are less able to withstand desiccation than filariform larvae, and at low temperatures they will not develop further (Kreis 1932; Little and Gutierrez 1968; Melashenko 1963).

It was observed in Colombia that most human feces were buried by dung beetles within a few hours after being deposited on the ground (Little and Gutierrez 1968). It was concluded that, although the burial of feces by dung beetles might have reduced the number of infective larvae developing in the soil, it probably also increased the chances of people coming in contact with the larvae that did develop. Since dung beetles kept the surface of the defecating sites relatively clean, people tended to return repeatedly to the same site to defecate and thus increased their chances of becoming infected. Studies in the Ukraine (USSR) showed that rhabditiform and filariform larvae of *S. stercoralis* did not move vertically in soil, but that free-living adults penetrated to a depth of 0.3 meters (Shablovskaya 1963).

A variety of alkalis and acids, especially hydrochloric acid and alcohol, are effective larvicides (Karbach 1966; Melashenko 1963; Rai 1935). Larvae are also susceptible to halogens, especially iodine (Thitasut 1961). Thiabendazole has also been used as a larvicide. Some plants and plant products (such as *Cymbopogon citratus*, *Eucalyptus globulus*, and *Mentha spicata*) have larvicidal properties (Goulart and others 1972).

## Inactivation by Sewage Treatment Processes

*S. stercoralis* exists in sewage as a delicate larva, not as a robust egg, and it is to be expected that complete elimination will take place during most sewage treatment processes. *S. fuelleborni* eggs may react to sewage treatment processes in a manner similar to hookworm eggs (chapter 22). No studies have been reported.

## Inactivation by Night Soil and Sludge Treatment Processes

*S. stercoralis* exists in night soil and sludge as a delicate larva, not as a robust egg, and it is to be expected that complete elimination will take place during most night soil and sludge treatment processes. *S. fuelleborni* eggs in night soil and sludge treatment processes may be eliminated in a manner similar to hookworm eggs (chapter 22). No studies have been reported.

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# 34

# *Taenia*, Taeniasis, and Cysticercosis

THE TAENIASES OF MAN, infection by the beef or pork tapeworms, are common in some areas but are typically not a major public health problem. They are, however, a major veterinary problem because infected cattle and pigs are not suitable for human consumption and there is considerable financial loss and wastage in endemic areas. The taeniases are of special interest to sanitary engineers because their transmission depends on the ingestion by cattle or pigs of inadequately treated human feces.

## Description of Pathogen and Disease

The medical and veterinary pathology, immunology, and therapy of taeniasis have been much studied, and a brief summary is given in the following sections. The epidemiology, in contrast, is poorly understood.

### *Identification*

Taeniasis is an infection with the adult stage of the beef tapeworm (*Taenia saginata*) or pork tapeworm (*T. solium*). The adult worm is attached to the wall of the small intestine and typically causes no symptoms. There may be irritation at the site of mucosal attachment and, rarely, abdominal pain, nausea, weakness, loss of weight, increased appetite, headache, and intestinal obstruction.

Cysticercosis describes an infection with the larval stage of *T. saginata* or *T. solium*; in man, only the larval stage of *T. solium* can infect. Human cysticercosis is a severe somatic disease involving many different organs and tissues in which encystment may occur. The manifestation of symptoms depends on the number of cysticerci and the tissues or organs involved. Cysticercosis is most common in muscles, the brain, and the heart.

Diagnosis of taeniasis is based on the recovery of gravid proglottids or eggs from the feces or perianal region. Differential diagnosis cannot readily be made between *T. saginata* and *T. solium* by the examination of *Taenia* eggs in the stool. Recovery of the gravid proglottids and a count of the main lateral arms of the uterus (7–13 on each side in the case of *T. solium* and 15–30 in the case of *T. saginata*) is the specific pretreatment diagnosis. Diagnosis of cysticercosis usually awaits excision of the larvae and microscopic examination, although serological techniques are available and radiology may be useful.

Treatment for taeniasis is by oral drug therapy, with niclosamide, praziquantel, or other suitable agent. Treatment of cysticercosis is usually surgical, by attempting to remove the cysts. Fenbendazole and praziquantel are under trial for the drug therapy of human cysticercosis.

### *Occurrence*

*T. saginata* and *T. solium* occur in almost all countries where beef or pork are eaten raw or undercooked (figures 34-1 and 34-2). *T. saginata* has its highest prevalence in East and Central Africa, in the Middle East, and in Latin America. *T. solium* is most frequently found in Southeast Asia and Latin America. Both are quite common in East Europe, where *T. saginata* and *T. solium* coexist, although the former is by far the more common. In some areas the prevalence of infection of man by adult worms is low (about 0.01 percent), whereas the prevalence of cysticercosis in cattle may be substantial (about 10 percent).

### *Infectious agent*

Worms of the genus *Taenia* are cestodes or tapeworms. The adult *Taenia* worm lives attached to the wall of the small intestine; its body winds back and forth in the lumen of the small bowel. *T. solium* is usually between 2–4 meters in length, whereas *T.*

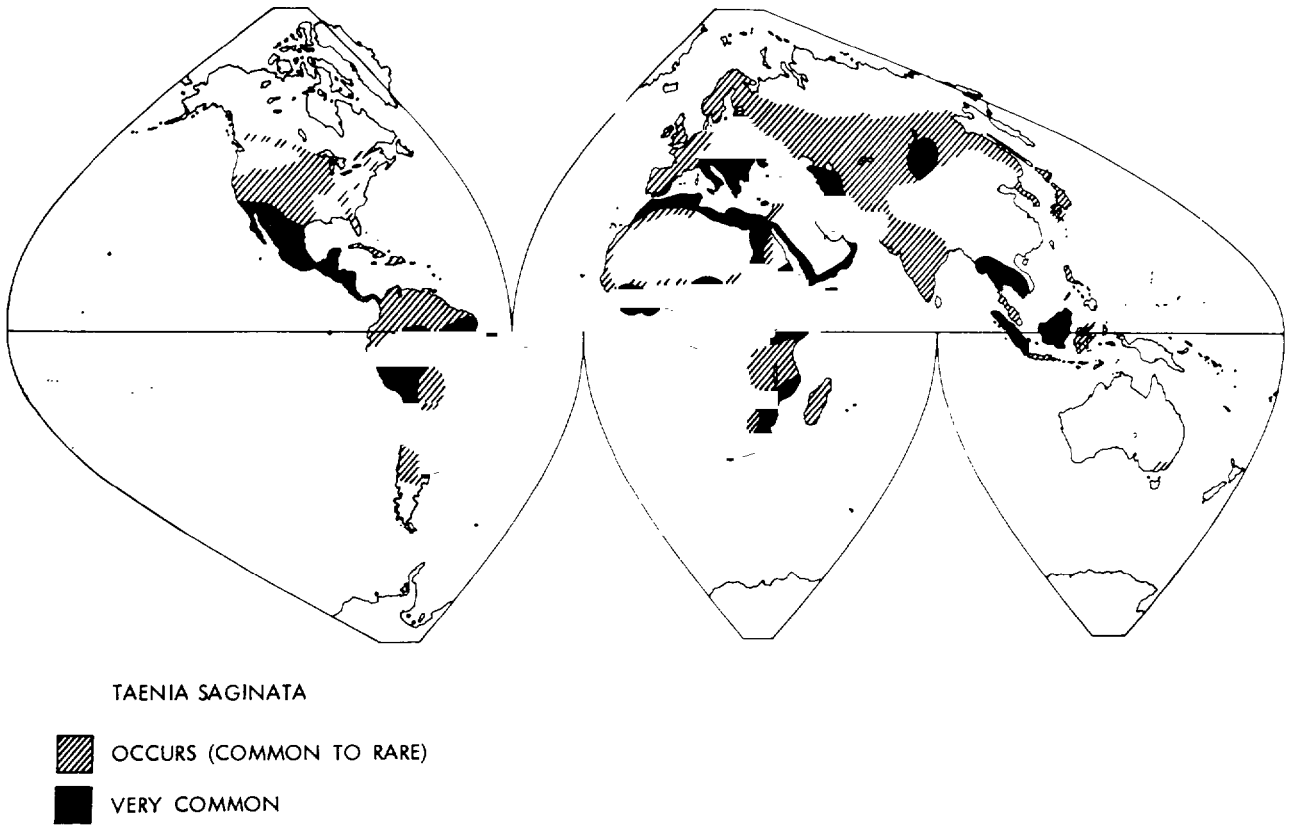


Figure 34-1. *Known geographical distribution of* Taenia saginata. The infection may occur in areas as yet unrecorded

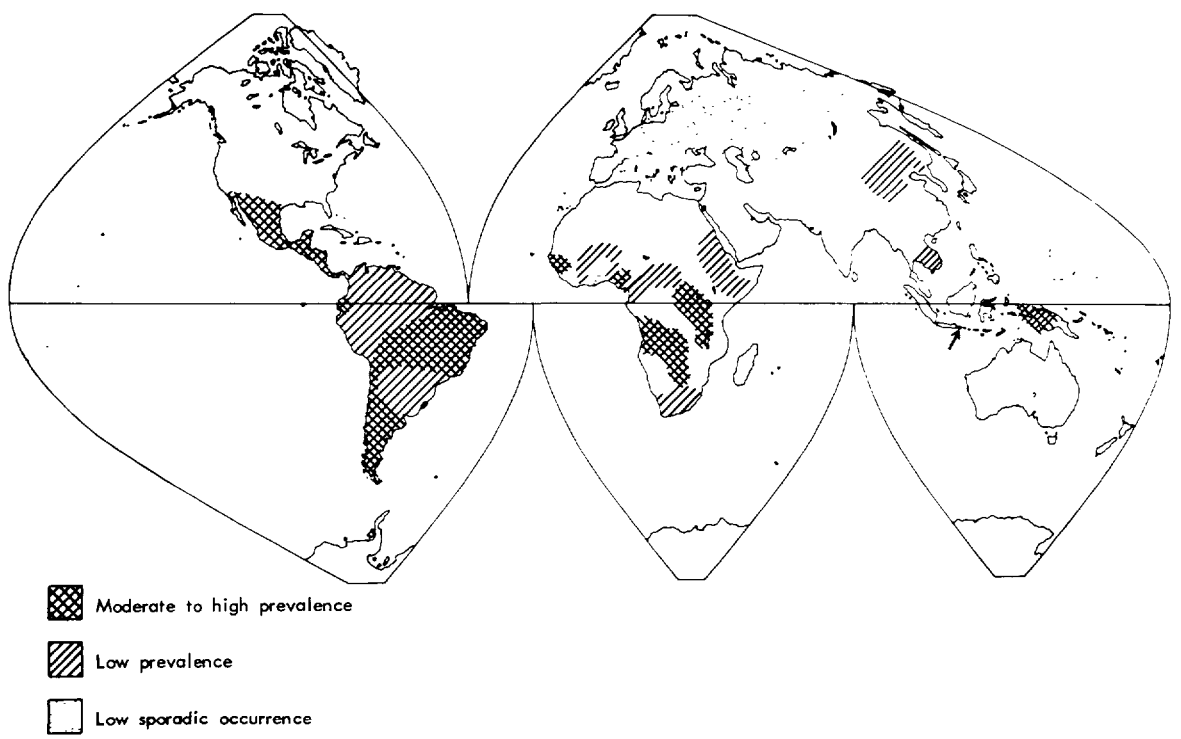


Figure 34-2. *Known geographical distribution of* T. solium. The infection may occur in areas as yet unrecorded

*saginata* under favorable conditions can reach a length of 15 meters or more but is usually not more than 6–10 meters long (figure 34-3).

The adult tapeworm consists of a scolex 1–2 millimeters in diameter, which bears four suckers that are attached to the gut wall (the scolex of *T. solium* also has a ring of hooks—see figure 34-3), and an area just behind the scolex that is a region of active cell division, the neck. From the neck the chain of proglottids (segments) is generated. The number of proglottids is 800–1,000 for *T. solium* and 1,000–2,000 for *T. saginata*. Each mature proglottid is roughly square and measures 10 by 12 millimeters. The mature proglottids have completely formed female and male sexual organs and are followed by the gravid proglottids, which consist essentially of a uterus distended with eggs. The gravid proglottids break off from the chain, usually pass out complete in the feces, and release the eggs in the soil. The number of eggs per proglottid can be $3 \times 10^4$–$9 \times 10^4$ for *T. solium* and $8 \times 10^4$–$1 \times 10^5$ for *T. saginata*. The eggs are roughly spherical and measure 30–70 micrometers in diameter.

### *Reservoirs*

The adult stage of *T. saginata* lives only in man; the larval stage lives in cattle and can possibly also infect buffalo, giraffe, llama and reindeer. The adult stage of *T. solium* lives only in man; its larval stage lives in pigs and can also infect man, apes, dogs, and possibly cats and sheep.

### *Transmission*

The adult tapeworm passes about $8 \times 10^5$–$1 \times 10^6$ eggs a day inside gravid segments. The eggs of the tapeworm that are passed in the stool are immediately infective to the intermediate host. The eggs of *T. saginata* cannot be distinguished readily from those of *T. solium*. When mature eggs are ingested by the intermediate host (cattle for *T. saginata* and pigs for *T. solium*) and reach the duedenum, hatching of the oncospheres occurs. An oncosphere is a term for the embryo within the egg. The embryo escapes from its shell, penetrates the intestinal wall, enters lymphatic or blood vessels, and is carried into the voluntary muscles where it develops into a mature bladder worm, known as a *Cysticercus bovis* (in the case of *T. saginata*) or *C. cellulosae* (in the case of *T. solium*) within 60–75 days.

The longevity of *T. saginata* cysticerci in the intermediate host depends on the host and on the type of tissue involved. In the liver, lung, and heart some cysticerci degenerate as early as 20 days after infection. It is usual to find living and dead cysticerci in the same host. Calves may differ from cattle in the maximal survival time of cysticerci, which may be 21–30 months.

When raw or undercooked infected beef or pork is eaten by man, the larval tapeworm attaches itself to the mucosa of the jejunum, where a mature worm develops in 5–12 weeks.

Human infection with *C. cellulosae* is caused by the ingestion of *T. solium* eggs. This may occur via contaminated food or water (heteroinfection) or via contaminated fingers when they are introduced into the mouth by patients who have the adult worm in their intestine (external autoinfection). Internal autoinfection—where the eggs are carried by reversed peristalsis back to the stomach and hatch—has been postulated, but there is no firm evidence that it occurs.

One cysticercus ingested may give rise to one adult hermaphroditic tapeworm and so may be enough to transmit the infection. One egg of *T. solium* ingested can give rise to one larva in the tissues. Although each egg is potentially infective to the animal host, there is an indication that a minimal dose is needed to cause cysticercosis. In previously unexposed calves 30–100 eggs developed 3–8 cysticerci, respectively, and 500 eggs produced 60–80 cysticerci (Jepsen and Roth 1949). The infective doses of *Taenia* eggs for cattle and pigs vary according to the previous history of infection, since immunity is developed in the intermediate host.

### *Prepatent and incubation periods*

*T. saginata* reaches maturity in the human intestine within 6–10 weeks from ingestion. *T. solium* reaches maturity in 5–12 weeks. An incubation period cannot be stated because symptoms may never develop.

### *Period of communicability*

The adult worms of *T. saginata* and *T. solium* can live in the human intestine up to 25 years or more. As long as the adult worms are present, infective eggs will be passed in the feces, and thus the possibility of transmission persists.

### *Resistance*

Man is universally susceptible. There is no evidence for the development of immunity against *Taenia* infections in the human host. Unlike the adult worm, which is weakly immunogenic, the larval stage of *Taenia* produces an active immunological response in cattle or pigs, and there is also an immunological response in man to infection by *Cysticercus cellulosae* (Flisser, Pérez-Montfort and Larralde 1979).

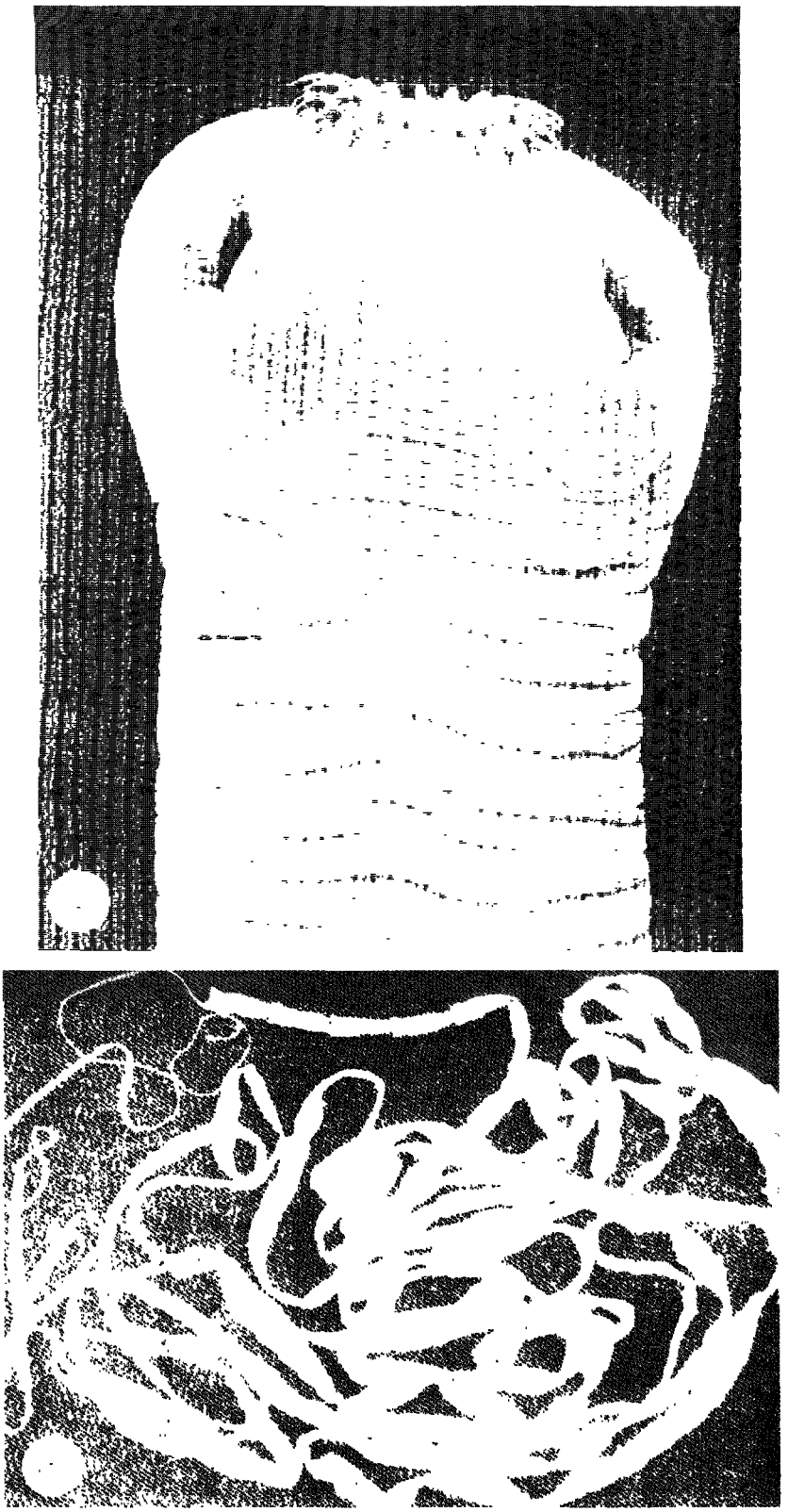

Figure 34-3. T. solium *and* T. saginata. (a) The head (scolex) and neck of *T. solium*-type under scanning electronmicroscopy, showing two of the four suckers and the two rows of hooks that aid attachment to the wall of the small intestine. *T. saginata* has the four suckers but no hooks. Scale bar = 0.1 millimeters. (Photo: A. Jones, Commonwealth Institute of Helminthology, St. Albans, UK.) (b) An adult *T. saginata*, several meters long. (Photo: Wellcome Museum of Medical Science)

*Epidemiology*

The epidemiology of taeniasis is not well documented. This is partly because it is not a major public health problem, even in areas where it is common, and also because it is a difficult infection to survey. Eggs are excreted intermittently in proglottids (Štěrba and Dyková 1979) and are not evenly distributed in the feces. Therefore, a single stool survey will grossly underestimate the prevalence of infection in man. The prevalence of infection in cattle or pigs is also difficult to measure. Serological techniques are under development; in the meantime, the only method is to slaughter and minutely inspect the carcases for cysts. Routine inspection of carcases at abattoirs underestimates the prevalence of infection (Rickard and Adolph 1977).

To maintain their life cycles, *T. saginata* and *T. solium* require two essential and specific conditions. First, cattle and pigs must eat human feces or fodder contaminated by human feces; second, beef and pork must be eaten raw or undercooked. The first condition is essential for transmission from man to animal and the second for transmission from animal to man. It follows that taeniasis is especially prevalent in communities where large herds of cattle or pigs are kept in close proximity to houses, where there is indiscriminate defecation by humans or the application of fecal products to pasture (or both), and where meat is not always thoroughly cooked. Simanjuntak and others (1977) described endemic taeniasis on Bali (Indonesia) and its relationship to the consumption of *lawar*, a dish of spiced minced pork often eaten uncooked, and the lack of adequate excreta disposal systems on the island. Fischer (1938) described how the outside pit latrines on farms in Germany in the 1930s were cold and unpleasant in winter and so encouraged the use of the warm cowsheds, often adjoining the house, for defecation by the farmer and his family. He recommended that latrines be made more accessible and more attractive than the cowsheds and that educational campaigns should encourage infected individuals to report for treatment and should discourage the eating of raw beef.

In developing countries, infection of cattle and pigs with *Cysticercus bovis* and *C. cellulosae*, respectively, is very common in communities where these animals have ready access to human excreta. Thus, 11 percent of slaughtered cattle in Sokoto State (Nigeria) had *C. bovis* infection (Dada and Belino 1979). Up to 80 percent of cattle in some East African herds are infected (WHO 1979).

In developed countries *T. saginata* infection remains common, especially in East Europe (Hajduk and others 1969; Sinnecker 1958), whereas *T. solium* is very rare. In the USA there are at least 200,000 cases of *T. saginata* infection in man, mainly in the west and northeast (Warren 1974). Taeniasis is more common in wealthy than in poor communities in the USA, presumably owing to higher meat consumption and more sophisticated recipes (steak tartare, for example). The prevalence of cysticercosis among slaughtered cattle in the USA is 0.05–0.08 percent.

Beef tapeworm has been attracting increased interest in Britain (Crewe and Owen 1978). It was very rare prior to 1945, but there is evidence of an increasing prevalence since that time. The overall prevalence of bovine cysticercosis is estimated at 0.1 percent, with a resultant economic loss to the beef industry of over US$1 million a year (Crewe and Owen 1978).

The modes of transmission of beef tapeworm in Britain and other industrialized countries remain uncertain and controversial. The link from cow to man is readily explained by the eating of undercooked beef; the link from man to cow is more difficult to explain in a society in which nearly everybody defecates into a sanitary toilet. Two main explanations have been propounded. First, that the increasingly widespread application of sewage sludge to pasture land provides the necessary opportunities for cattle to ingest fecal material of human origin. Second, that birds, especially seagulls, feeding on trickling filters and sludge drying beds pass the *Taenia* eggs or proglottids unharmed through their guts and excrete them later on pasture. Both these transmission mechanisms are theoretically possible, and *T. saginata* eggs have been isolated both from sludge applied to pasture and from gull droppings (Crewe 1967; Silverman and Griffiths 1955). It remains unresolved, however, which mechanism is the more important, and this uncertainty is a major constraint to policy formulation on sludge treatment prior to pasture application or other possible control strategies.

Human cysticercosis can be a very severe disease and occurs following the ingestion by man of *T. solium* eggs. Man accidentally takes the place of the pig in the normal pork tapeworm life cycle. Human cysticercosis occurs wherever there is endemic pork tapeworm infection (figure 34-2), and especially where taeniasis prevalances in man are high and sanitation and hygiene are poor. Thus cysticercosis is to be expected among poor people who keep pigs and eat pork.

The highlands of West Irian (Indonesia) and Papua New Guinea are ideal sites for endemic *T. solium* and consequent human cysticercosis. West Irian was free of *T. solium*, however, until the introduction of infected pigs from elsewhere in Indonesia during 1971. This

introduction caused an epidemic of taeniasis and cysticercosis in certain areas. This in turn led to an outbreak of severe burns from open fires sustained during epileptic fits caused by *Cysticercus cellulosae* cysts in the brain (Gajdusek 1978).

## Control Measures

Control rests upon denying cattle and pigs access to inadequately treated human excreta, meat inspection, and encouraging thorough cooking of beef and pork.

### *Individual*

There are no specific prophylactic drugs available for *Taenia* infections. Compulsory mass diagnosis and treatment campaigns carried out in Poland, Bulgaria, and the USSR have successfully reversed a rising incidence of taeniasis. Other measures, such as improved meat inspection and excreta disposal, have to be taken in conjunction with mass chemotherapy to prevent reinfection.

There is no immunological control technique against taeniasis in man. There is, however, a possibility of immunizing cattle against infection, and research is in progress.

A comprehensive system of meat inspection at abattoirs, and the discarding of parts or all of infected carcases, is an essential element in tapeworm control programs. Most industrialized countries, and some developing countries, have a meat inspection system, but effectiveness depends on the training and supervision of the inspectors, on their ability to withstand inducements to overlook infected carcases, and on the absence of an alternate meat distribution system that circumvents the registered abattoirs. These conditions may be very difficult to achieve.

### *Environmental*

Long-term reduction in transmission of *Taenia* depends on improved sanitation and sanitary education. Disposal of feces in a way that prevents any contact between the infective eggs and the intermediate host will break the life cycle and, together with chemotherapy and meat inspection, will help to eliminate the disease. The use of night soil or sludge as a fertilizer on pasture, or disposal of effluents into rivers that are a source of drinking water for cattle, necessitates adequate treatment. Educational programs should cover sanitary education, prevention of illegal slaughter and unsupervised meat distribution, meat inspection, and cooking habits.

## Occurrence and Survival in the Environment

The information available on *Taenia* eggs in the environment is less extensive than for *Ascaris* eggs and has recently been reviewed by Lawson and Gemmel (1983). Survival of *Taenia* eggs is dependent primarily on temperature and moisture, with greatly reduced survival times in hotter and dryer conditions. Survival times in various environments are less than those of *Ascaris* eggs (chapter 23).

### *In water*

*Taenia* eggs have been isolated from river water in the USSR (Bukh 1945; Usacheva 1951) and elsewhere. *Taenia* eggs have also been isolated from seawater near sewage outfalls in the USSR (Amirov and Salamov 1967) and from coastal and riverine beaches (Amirov and Salamov 1967; Iwańczuk 1969).

Survival in water is temperature dependent, with longer survival at lower temperatures. Laboratory experiments on *T. saginata* eggs in normal saline at 2–5°C showed survival of up to 168 days (Froyd 1962), between 95 and 116 days (Penfold, Penfold and Phillips 1937), and up to 335 days (Silverman 1956). In saline at room temperature, survival times were reduced to about 60 days (Silverman 1956). Suvorov (1965) reported that survival times of *T. saginata* eggs in water increased as temperature fell from 37°C to −4°C, at which temperature eggs remained viable for 63 days. Survival then decreased with lower temperatures and was 17 days at −30°C.

Jepsen and Roth (1949) demonstrated that *T. saginata* eggs were still infective to calves after storage in water for 33 days at 18°C, and Hajduk and others (1969) reported that *T. saginata* eggs survived in river water in the German Democratic Republic for 35 days. Livingstone (1978) reported that *Taenia* eggs survived in seawater for periods similar to *Ascaris* eggs.

Eggs in proglottids are more resistant to ovicidal chemicals than are free eggs (Gall and Wikerhauser 1968), but free eggs survive longer in water than eggs in proglottids (Suvorov 1965).

### *In sewage*

*Taenia* eggs are found in sewage deriving from any community with endemic taeniasis. They may be present only in very low concentrations, and they may be still retained in their proglottids, thus making detection difficult. *Taenia* eggs have been isolated from sewage in the German Democratic Republic (Kalbe

1956; Sinnecker 1958), Japan (Liebmann 1965), South Africa (Nupen and de Villiers 1975), the USA (Wang and Dunlop 1954), the USSR (Vassilkova 1941), and elsewhere.

Jepsen and Roth (1949) demonstrated that *T. saginata* eggs remained infective to calves after 16 days at 18°C in sewage, and Hajduk and others (1969) reported that *T. saginata* eggs survived in sewage in the German Democratic Republic for 20 days.

### *In sludge*

*Taenia* eggs are concentrated in the sludge of sewage treatment plants. Newton, Bennett and Figgat (1949) found that 30–46 percent of *Taenia* eggs survived for more than 6 months in sludge at 24–30°C. Hajduk and others (1969) reported that *T. saginata* eggs survived in dung from cowsheds in the German Democratic Republic for 71 days.

### *On pasture*

*T. saginata* is transmitted from man to cattle when cattle ingest infected human feces. This may occur when feces, sludge, or night soil are deposited accidentally or deliberately on pasture or are added to silage that is later fed to cattle. The survival of *T. saginata* eggs on pasture and in silage is therefore of epidemiological importance.

Some reported survival times are listed in table 34-1. Survival is inversely related to temperature above 0°C, and *T. saginata* eggs may survive for 6 months under cool, moist conditions. Under hot, dry conditions survival is unlikely to exceed 2 months.

## Inactivation by Sewage Treatment Processes

*Taenia* eggs respond to sewage treatment processes in the same way as *Ascaris* eggs, and the probable efficacy of various treatment technologies may be judged from chapter 23. Data from Britain (Silverman 1955; Silverman and Griffiths 1955), India (table 22-4), the USA (Greenberg and Dean 1958; Newton, Bennett and Figgat 1949), the USSR (Vassilkova 1936), Federal Republic of Germany (Liebmann 1963, 1964), and other countries confirm that most removal takes place by sedimentation and that tapeworm eggs are concentrated in the sludge. Waste stabilization pond effluent should contain no *Taenia* eggs.

Table 34-1. *Some studies on the survival of* Taenia *eggs in grass, silage, and soil*

| *Country* | *Site of eggs* | *Temperature or season* | *Survival (days)* | *Source* |
|---|---|---|---|---|
| Australia | Grass | July–September | 57 | Penfold, Penfold and Phillips (1937) |
| Denmark | Soil | February–July | 159 | Jepson and Roth (1949) |
| Dem. Rep. Germany | Grass | ND | 180 | Hajduk and others (1969) |
| USA | Hay | 1–30°C | 22 | Lucker and Douvres (1960) |
| USSR | | | | |
| Nakhichevan (Azerbaydzhan) | Grass | Winter (10 to −16°C) | 30 | Abbasov (1965) |
| | Grass | Summer | 60 | |
| Samarkand (Uzbekistan) | Soil | Autumn and winter | 180–210 | Babaeva (1966) |
| | Soil | Spring | 45–105 | |
| | Soil | Summer | "Several days" | |
| Vologodskaya Province | Hay | Winter | 210 | Shepelev (1961) |
| | Soil | Summer | 180 | |
| Fed. Rep. of Germany | silage | 10°C | 80 | Enigk, Stoye and Zimmer (1969) |

ND No data.

*Note:* All experiments used *T. saginata* eggs except those of Abbasov (1965) and Shepelev (1961), which used *T. hydatigena* eggs.

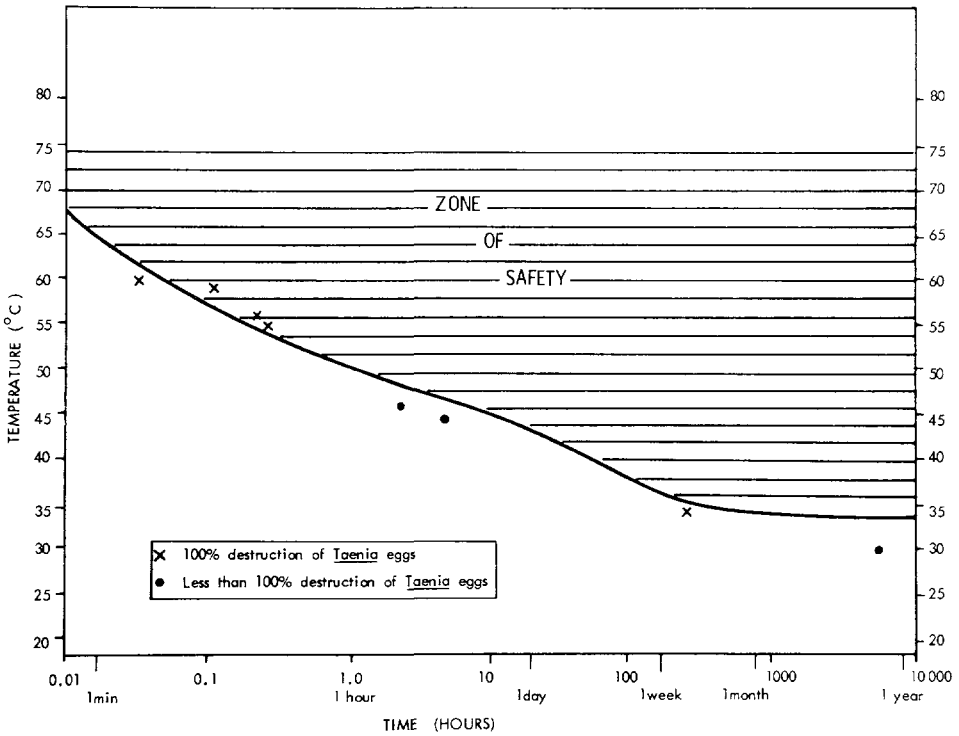


Figure 34-4. *The influence of time and temperature on* Taenia *eggs.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

## Inactivation by Night Soil and Sludge Treatment Processes

Time, temperature, and desiccation are the principal lethal factors acting on *Taenia* eggs during the treatment of night soil or sludge. Mesophilic digestion is generally thought not to eliminate *Taenia* eggs (Liebmann 1964; Pawłowski and Schultz 1972), although contrary evidence is given by Silverman and Guiver (1960).

For elimination of Taenia eggs, night soil and sludge must be stored for a protracted period or be heat-treated by thermophilic composting. Necessary storage times depend on ambient temperatures; 1 year may be required in temperate regions, whereas 6 months is probably adequate in the tropics. If the stored sludge becomes extremely dry (moisture content $<10$ per cent), *Taenia* egg destruction is hastened.

Time-temperature requirements for the inactivation of *Taenia* eggs have been reported by Allen (1947), Silverman (1956), and other workers cited above. These data are plotted on figure 34-4, and comparison with figure 23-2 shows that *Taenia* eggs are more readily destroyed than *Ascaris* eggs. Any night soil or sludge treatment process that destroys *Ascaris* eggs may be assumed also to destroy *Taenia* eggs.

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# 35

# *Trichuris* and Trichuriasis

THERE ARE THREE intestinal worm infections of man having cosmopolitan distributions and producing severe clinical consequences in heavily infected individuals. These are hookworm infection (chapter 22), ascariasis (chapter 23), and trichuriasis (this chapter). Trichuriasis is often considered jointly with ascariasis because they are usually endemic in the same communities and their life cycles, modes of transmission, and epidemiologies are similar.

## Description of Pathogen and Disease

Trichuriasis is an infection of man by the human whipworm *Trichuris trichiura.* It is commonly referred to as whipworm infection and, rarely, as trichocephaliasis. Recent reviews include Chanco and Vidad (1978), Mahmoud (1979), and Wolfe (1978).

### *Identification*

Trichuriasis is a helminthic infection of the large intestine and cecum. Most infections in adults are symptomless, but there may be slight abdominal pain and some diarrhea. In malnourished children heavy infection can cause anemia, bloody diarrhea, and occasionally, prolapse of the rectum (Jung and Beaver 1951; Jung and Jelliffe 1952; Kamath 1973). Diagnosis is by identification of *Trichuris* eggs in the feces. Safe and effective drugs—for instance, mebendazole—are now available for treating trichuriasis, and some of these are effective in mixed infections with hookworm and *Ascaris.*

### *Occurrence*

Trichuriasis occurs throughout the world and is very common in some areas with warm and humid climates. Local prevalences of 50–99 percent are reported, although 25–40 percent is more usual. Prevalence and intensity of infection are highest among children 5–15 years old.

### *Infectious agent*

*Trichuris trichiura* (formerly called *Trichocephalus dispar, Tricho. trichiura,* and *Tricho. hominis*), a nematode, is the human whipworm. Adult female worms are 25–50 millimeters in length; males measure 30–45 millimeters, with a tightly coiled posterior end (figure 35-1). The eggs are lemon-shaped with plug-like, translucent prominences at each end. The eggs are 50–55 micrometers long and 22 micrometers wide. The pig whipworm, *T. suis,* is very similar and can also infect man; routine stool examination does not distinguish between eggs of *T. trichiura* and those of *T. suis.* Closely related species infect other animals; for instance, *T. ovis* in sheep, *T. vulpes* in dogs and foxes, and *T. muris* in mice.

### *Reservoir*

The reservoir of *T. trichiura* is man, who may also act as a minor reservoir of *T. suis.* It is possible that pigs, lemurs, and monkeys may also act as a reservoir of *T. trichiura.*

### *Transmission*

Female worms lay 2,000–10,000 eggs per day. The unsegmented fertilized eggs are discharged in the feces and take 2–5 weeks to develop into the infective stage in a moist, warm environment (usually soil or the perineum). Development time is temperature dependent and may be 4–6 months at 15°C, 3–4 weeks at 26°C, 17 days at 30°C, and 11 days at 35°C. The eggs of *Trichuris* are less resistant than those of *Ascaris,* but even so they may survive for several months in shaded, moist soil. Once ingested—from contaminated hands,

Figure 35-1. *A male (left) and female (right)* Trichuris trichiura *under a light microscope.* The worms are 30–50 millimeters in length. (Photo: Wellcome Museum of Medical Science)

food, or soil—infective eggs hatch in the intestine to liberate small larvae, which feed and grow in the small intestine before passing to the cecum to become mature adults. Adult worms anchor themselves to the large bowel mucosa by means of a spear-like projection at their anterior end. This process of maturity takes about 2 months, and the adult worm can live for 3–5 years in man. Heavy infections are associated with egg counts in excess of 30,000 per gram of feces.

*Prepatent and incubation periods*

A female worm may mature and start producing eggs about 2 months after the ingestion of infective eggs. Clinical symptoms may never develop or may develop gradually as a result of continuing reinfection and increasing worm burdens.

*Period of communicability*

Eggs are discharged in the feces as long as there is an adult fertilized female worm living in the cecum. The life span of an adult worm may be up to 8 years but is typically around 3 years. The eggs may develop and remain infective for several months in a moist environment.

*Resistance*

Little is known of the immunology of trichuriasis. Susceptibility is general, and the age distribution of infection may be due to the varying degrees of exposure at different ages.

*Epidemiology*

Trichuriasis is extremely common in some areas of the world, especially where the climate is wet and humid and where there is extreme poverty. Surveys of children 1–5 years old in Guatemala revealed trichuriasis prevalences of 33 percent among rural children, 9 percent among poor urban children, and 4 percent among more wealthy urban children (Pierce and others 1962). In one village in Guatemala, 48 percent of children became infected by *Trichuris* between birth and 3 years (Mata and others 1977).

A survey of children 4–6 years old in Kuala Lumpur (Malaysia) found a prevalence of trichuriasis of 84

percent among poor children and 8 percent among "upper middle class" children (Yan and others 1978). A study of rural schoolchildren (6–12 years old) 72 kilometers from Kuala Lumpur found an 85 percent prevalence of trichuriasis (Lo and others 1979).

In the late 1960s, trichuriasis was the most common intestinal worm infection in South Korea, with prevalence rates of 72 percent in Seoul and 75 percent in rural areas (Seo and others 1969). *Trichuris* was also the most common intestinal worm in Cali (Colombia) during 1956–61, when the prevalence among children 5–9 years old was 91 percent (Faust and Mugaburu 1965). Similarly, *Trichuris* was more prevalent than *Ascaris* or hookworm in Haiti, with an 85 percent prevalence among children 10–14 years old (Raccurt, Vial and Pierre-Louis 1977).

There are major unexplained variations among communities in the prevalence of trichuriasis and in the relative prevalence of *Trichuris*, *Ascaris*, and hookworm infections. It is often asserted that areas of high rainfall are associated with more *Trichuris* and less *Ascaris*, whereas in drier regions *Ascaris* may be expected to be the more prevalent worm (Spindler, 1929). Some data support this, and high prevalences are frequently found in hot wet climates, especially in areas of East Asia where night soil is widely used in agriculture. In Iran, the prevalence of trichuriasis was 6 percent in the dry southwest (Massoud and others 1980); compared with up to 60 percent in the wetter area bordering the Caspian Sea (Ghadirian, Croll and Gyorkos 1979). Low prevalences are reported, however, from areas with seasonally wet climates, such as Delhi (Biswas and others 1978) and Bengal (Nawalinski, Schad and Chowdhury 1978) in India and from areas with perennially wet climates, such as Papua New Guinea (Jones 1976).

As with ascariasis (chapter 23), both the prevalence and intensity of trichuriasis infection typically peak in the 5–15 age group. At older ages intensities decline, and prevalences either decline similarly or maintain fairly constant levels throughout later life.

Trichuriasis is common in some developed countries. There are an estimated 2.2 million people infected in the USA, and the disease is especially common in the southeast (Fulmer and Huempfner 1965; Warren 1974). Male homosexuality is associated with risk of trichuriasis in some developed countries (McMillan 1978). A survey of trichuriasis among mentally subnormal patients at seventeen hospitals in the UK revealed that the overall prevalence was 13 percent, with markedly more infection in southern England than northern England and no infection in Scotland (Lynch and others 1972).

The epidemiology of trichuriasis is closely similar to that of ascariasis, and the remarks made on epidemiology in chapter 23 are applicable. A major difference is the greater longevity of the adult *Trichuris* worm, which provides greater opportunity for accumulating high worm burdens and delays the effects of any control program.

Students of trichuriasis epidemiology, and of the comparative epidemiology of ascariasis, trichuriasis, and hookworm infection, should read the early accounts of these infections from China, Panama, and the USA (Cort, Otto and Spindler 1930; Cort, Schapiro and Stoll 1929; Cort and Stoll 1931; Cort and others 1929; Otto 1932; Otto, Cort and Keller 1931).

## Control Measures

Only environmental and behavioral changes can have a sustained impact on trichuriasis, but mass chemotherapy may be used to reduce infection rates in the short term.

### *Individual*

No prophylactic drugs or vaccines are available. Mass chemotherapy has not been as successful in controlling trichuriasis as it has been with ascariasis and hookworm infection, because until recently safe and effective drugs were not available. With the advent of new drugs, especially mebendazole and oxantel-pyrantel, mass chemotherapy has become an important control strategy.

### *Environmental*

The remarks made about the environmental control of ascariasis (chapter 23) apply to trichuriasis. The major difference is that the longer life span of *Trichuris* in the human host results in prevalence rates falling more slowly following a successful campaign to control transmission.

Control rests upon major improvements in excreta disposal, especially for children, and changes in behavior associated with defecation. In areas where night soil is used in agriculture, it is necessary to treat the night soil thoroughly before application to the fields. Environmental measures that interrupt transmission will reduce prevalence rates slowly, and it may be many months before measurable reductions in trichuriasis in the community are obtained. Therefore environmental and behavioral modifications should be

combined with periodic mass chemotherapy in any trichuriasis control program. This integrated approach to control has been successful in several countries; for instance in South Korea (Soh 1973).

Some studies on environmental interventions and trichuriasis are listed in table 35-1. The discussion of table 23-2 applies fully to these findings.

In the city of Kermanshah (Iran), Ghadirian and others (1973) recorded trichuriasis prevalences of 72 percent in a low-income area near an open sewer and 58 percent in a high-income area far from the sewer. This latter high prevalence in a more wealthy section of the city was attributed to the irrigation of vegetables with night soil and sewage. In South Africa it was reported that Xhosa children (6–9 years old) living in Cape Town (piped water and sewer connections to every house) had a trichuriasis prevalence of 89 percent, whereas rural Xhosa children in villages in the Transkei (water collected from streams and sanitation comprising pit latrines or indiscriminate defecation) had a prevalence of only 3 percent (van Niekerk and others 1979). The reason for this strange finding was not known but may be connected with the wetter climate of Cape Town. Both the Iranian and South African data strongly suggest that limited environmental improvements may not be sufficient to control *Trichuris* transmission.

## Occurrence and Survival in the Environment

There is considerably more information on the occurrence and survival of *Ascaris* eggs in the environment than on the survival of *Trichuris* eggs. This is partly because, worldwide, *Ascaris* is probably the more common worm and also because *Ascaris* eggs are more resistant to hostile environments than *Trichuris* eggs. *Ascaris* eggs therefore provide a better parasitological indicator than *Trichuris* eggs in areas where both are endemic. *Ascaris* eggs are better able to survive both warm and cold temperatures than *Trichuris* eggs (Nolf 1932); and the latter require a higher moisture level in soil to develop than *Ascaris* eggs (Spindler 1929).

A few studies on *Trichuris* eggs in the environment are mentioned below. It may be assumed that the comments on *Ascaris* eggs in the environment made in chapter 23 apply to *Trichuris*, except that *Trichuris* eggs will typically remain viable for shorter periods.

### *In water*

Yarulin (1955) isolated *Trichuris* eggs from 3 percent of Caspian Sea water samples near a sewage outfall in the USSR. Usacheva (1951) isolated *Trichuris* eggs from river water and river sediment in the USSR. Iwańczuk (1969) found *Trichuris* eggs in greater numbers than *Ascaris* eggs on public beaches on the River Vistula (Poland), but these probably derived from promiscuous defecation by visitors rather than from the river water. Livingstone (1978) reported that *Trichuris* eggs rapidly swelled and died in seawater.

### *In feces and night soil*

Some *Trichuris* eggs in pit latrines remained viable for over 18 months but died sooner than *Ascaris* eggs (Biziulevicius 1965).

### *In sewage*

Reported concentrations of *Trichuris* eggs per liter of sewage include 10–20 in Tokyo (Japan; Liebmann 1965) and 41 in San Juan (Puerto Rico; Rowan and Gram 1959). Many accounts of *Trichuris* eggs in

Table 35-1. *Some studies on environmental influences on trichuriasis*

| *Country* | *Result* | *Source* |
|---|---|---|
| Costa Rica | Trichuriasis prevalence was the same among individuals with or without a latrine but was lower among those having a septic tank system. | Moore, de la Cruz and Vargas-Mendez (1965) |
| Japan | Night soil treatment with thiabendazole reduced trichuriasis prevalence from 65 percent to 47 percent over 2 years. | Kutsumi (1969) |
| Singapore | Poor families rehoused for 1 year in modern flats had a trichuriasis prevalence of 28 percent; in comparison squatter families had a 58 percent prevalence. | Kleevens (1966) |
| USA | *Trichuris* transmission was interrupted when adult female mental patients were moved from old unsanitary quarters to a modern building. | Jeffery (1960) |

sewage in the German Democratic Republic (for instance, Kalbe 1956; Sinnecker 1958), and the USSR (for instance, Vassilkova 1936) have been published.

*In sludge*

*Trichuris* eggs, like *Ascaris* eggs, tend to settle in primary and secondary sedimentation tanks and are therefore concentrated in the sludge from sewage treatment plants. *Trichuris* eggs have been found in sludges in Czechoslovakia (Králová and Šafránek 1957) and the USA (Theis, Bolton and Storm 1978), and may be expected at every sewage treatment plant serving a population with endemic trichuriasis.

*In soil*

*Trichuris* eggs, like *Ascaris* eggs, can remain alive in soil for extended periods, especially if conditions are moist, cool, and shaded. A study at a hospital near London (England) showed that 21 percent of *T. trichiura* eggs were still potentially infective after 18 months in "clay-flint" soil (Burden and others 1976).

*On crops*

Where trichuriasis is endemic and fecal materials (sewage, night soil, or sludge) are used in agriculture, *Trichuris* eggs may be found on crops, and this may play some role in transmission. *Trichuris* eggs on vegetables have been extensively studied in the USSR (Barchenko 1953; Biziulevicius 1954; Khaustov 1935; Romanenko 1971; Vassilkova 1941) and other East European countries. Sinnecker (1958) isolated *Trichuris* eggs from sewage-irrigated lettuce in the German Democratic Republic and reported that prevalences of trichuriasis were higher (20 percent) among irrigation workers than among sewermen (8 percent) or sewage treatment plant operators (0 percent). *Trichuris* contamination of vegetables is especially common in areas of East Asia—for instance, in South Korea (Choi 1970), where untreated night soil is a commonly used fertilizer.

The prominence of contaminated vegetables in trichuriasis transmission is partly dependent on the level of domestic hygiene and sanitation, and thus on the degree to which yard transmission among children is taking place. In more wealthy communities where hygiene and sanitation are good, it is possible that contaminated vegetables may be a major transmission route. This was thought to be the situation among the Jewish residents of Jerusalem (Israel), where trichuriasis prevalence fell from 13 percent in 1947 to 5 percent in 1960, possibly owing to the cessation of the supply of sewage-irrigated vegetables from Jordan (Ben-Ari 1962).

Crops may be decontaminated by soaking in an ovicidal chemical, although *Trichuris* eggs are more resistant to iodine than are *Ascaris* eggs (Thitasut 1961). It will usually be more reliable and appropriate to immerse the vegetables in warm water (60°C) for 10 minutes (see chapter 23).

## Inactivation by Sewage Treatment Processes

Little information is available on *Trichuris* egg removal by sewage treatment processes because most researchers have focussed on *Ascaris* egg removal. The data presented for *Ascaris* egg removal (chapter 23) may be assumed to apply to *Trichuris* egg removal.

Indian data assembled by Panicker and Krishnamoorthi (1978) are presented in table 22-4 and show that *Trichuris* and *Ascaris* removals are similar. This is also shown by studies on sedimentation and activated sludge treatment in Calcutta (Bhaskaran and others 1956). Correctly designed and operated waste stabilization ponds remove all *Trichuris* eggs (table 22-4 and Lakshminarayana and Abdulappa 1972). Vassilkova (1936) studied *Trichuris* eggs in sewage treatment plants near Moscow (USSR), and Plyushcheva (1974) experimented with ionizing radiation to kill *Trichuris* eggs in sewage.

## Inactivation by Night Soil and Sludge Treatment Processes

*Trichuris* eggs tend to be concentrated in the sludge of all sewage treatment processes, and high concentrations may be expected in night soil in areas where trichuriasis is endemic. The data presented on *Ascaris* egg removal (chapter 23) may be assumed to apply to *Trichuris* egg removal, except that *Trichuris* eggs are probably eliminated somewhat earlier than *Ascaris* eggs during storage, digestion, or composting.

Reports on *Trichuris* eggs in sludge following various forms of treatment are available from the USA (Wright, Cram and Nolan 1942), the USSR (Vassilkova 1936), China (Szechwan Research Institute 1974), and other countries. Most research has concentrated on *Ascaris* eggs in sludge and night soil treatment processes because they are usually more numerous and are believed to be more persistent than *Trichuris* eggs. Contrary evidence is provided by Enigk

and others (1975), who found that *Trichuris* eggs survived thermophilic digestion for up to 5 days, whereas *Ascaris* eggs were eliminated within 3 days.

*Trichuris* eggs in sludge and night soil can be eliminated by the addition of appropriate ovicides (table 23-4). Kutsumi and Komiya (1965) experimented with thiabendazole and found that, under similar conditions, *Trichuris* eggs were killed by the application of concentrations one-sixth to one-sixteenth of those required for *Ascaris* egg destruction.

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# *Section V.*
# *Insects and Excreta*

# 36

# *Culex pipiens* Mosquitoes and the Transmission of Bancroftian Filariasis

CHAPTERS 36 AND 37 are different from the other chapters in Part Two in that they deal not with excreted infections *per se* but with insects that may transmit both excreted and nonexcreted infections and that breed in or visit excreta or sewage. This chapter describes the *Culex pipiens* mosquitoes, which breed in sewage and sullage and are a vector of Bancroftian filariasis. Because of the very different nature of the subject matter, the standard headings adopted for chapters 9–35 have not been used in this chapter and chapter 37.

## The Biology of *Culex pipiens* Mosquitoes

The immature stages (eggs, larvae, and pupae) of mosquitoes live in water. Among the many mosquitoes able to transmit human disease, three groups of species are particularly important. One group, the *Culex pipiens* complex, has the most relevance to sanitation methods because it favors polluted water for breeding. Of the other two groups, *Anopheles* species (vectors of malaria) breed in stretches of fairly clean water, such as flood or irrigation water, and *Aedes aegypti* (vector of yellow fever, dengue, and dengue hemorrhagic fever viruses) breeds especially in clean water stored in pots, cisterns, and the like, unless these are carefully screened to prevent their access.

The *Culex pipiens* complex consists of several closely related forms such as *C. p. pipiens*, *C. quinquefasciatus*[1] (previously called *C. p. fatigans*, *C. p. quinquefasciatus*, and *C. fatigans*), *C. p. molestus*, and *C. p. pallens*. These vary in such physiological characters as ability to hibernate or lay their first batch of eggs without taking a blood meal. One or another of the members of the *C. pipiens* complex live anywhere that man accidentally creates suitable conditions for them within the geographical range shown in figure 36-1.

The eggs of *Culex pipiens* mosquitoes are laid in clumps ("rafts") of 50–250. The larvae hatch after 1–2 days and then pass through four stages (instars), lasting a total of 1–2 weeks at tropical temperatures, during which they breathe air through a siphon at the posterior end while hanging diagonally from the surface film of the water (figure 36-2) and feed on detritus. The comma-shaped pupae swim about, like the larvae, and give rise to the adult mosquito after about two days (figure 36-2). The female adults mate once only in the first few days of life and, as with other mosquitoes, only the females feed on blood. One blood meal is taken between the laying of each egg raft. Feeding takes place at night, and the favored blood sources are man and birds.

## Bancroftian Filariasis

In some parts of the world, especially in continental Asia, *Culex pipiens* mosquitoes are the vectors of the causative agent of Bancroftian filariasis, the nematode worm *Wuchereria bancrofti* (figure 36-1). *W. bancrofti* is transmitted by quite different mosquito species in different parts of the world. In some places the vector is *Anopheles* (the malaria-carrying mosquito genus), whereas elsewhere the vectors are *Aedes* mosquitoes.

The adult worms live in the lymphatic ducts of man. Embryos (microfilariae) are shed in vast numbers into the blood stream. In the periodic form of the disease, which is the only form transmitted by *C. pipiens*, the microfilariae only occur in the blood at night, and the routine method of diagnosing the infection is by examination of blood samples taken at night for the presence of microfilariae. If a susceptible mosquito

1. This description was proposed by Sirivanakarn and White (1978) and later adopted by the World Health Organization.

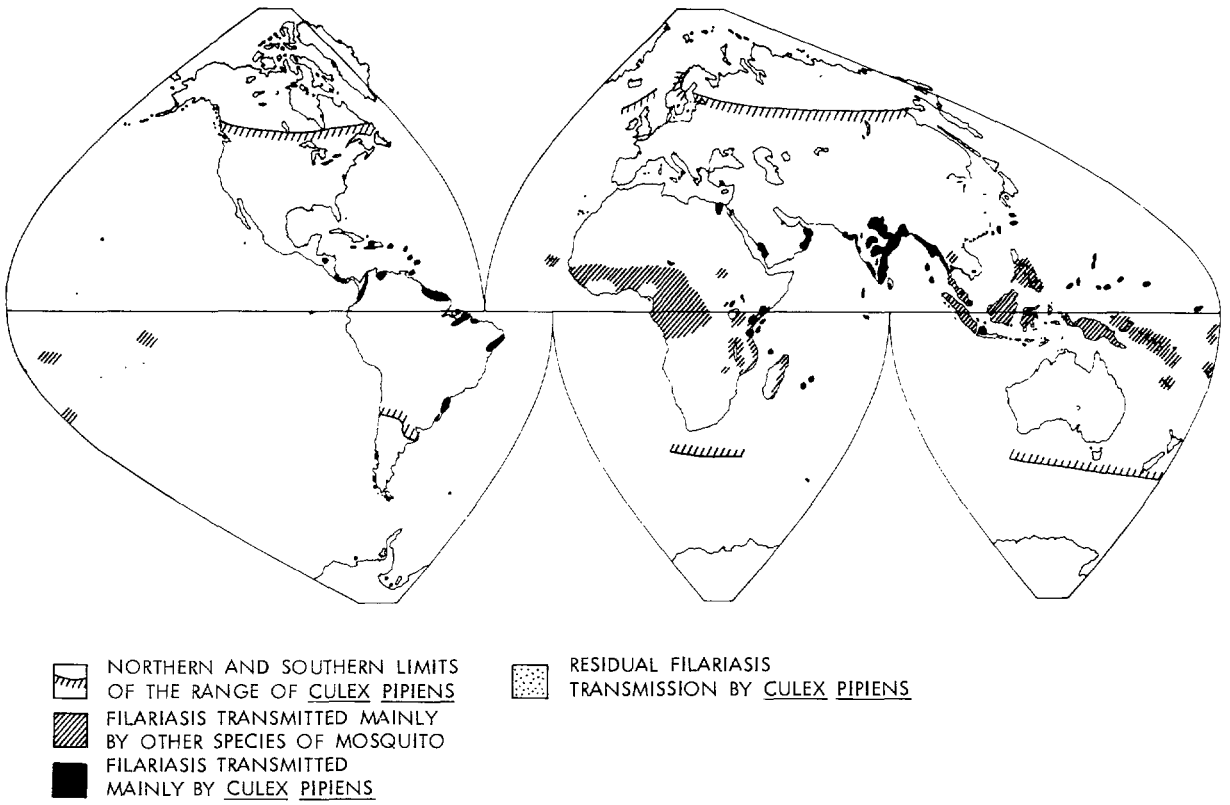



Figure 36-1. *Known geographical distribution of* Culex pipiens *mosquitoes and Bancroftian filariasis*

ingests microfilariae during a blood meal, they develop inside the mosquito over a period of 10–15 days to become infective larvae. When the mosquito feeds again, they may be reintroduced into another person where male and female worms establish themselves in the lymphatic system and recommence the cycle of microfilaria production.

Several years (often 20 or more) after the adult worms have established themselves, a reaction by the tissues of the infected person may block the lymphatic vessels and so prevent the return of the lymph. This may lead to swellings of the genitalia, legs, or arms. The first is known as hydrocele, whereas the stage of gross deformity of the legs or arms is called elephantiasis. Filariasis is not a lethal disease. The economic impact of the disablement that it causes has not been assessed, but the disease is much feared in areas where it is endemic.

There is no vaccine available as yet against filariasis, but the drug diethylcarbamazine is effective against the filarial worms while they are still alive and before severe symptoms of the disease appear. The drug may cause unpleasant side effects if used at the dosages required for effectiveness in a short course of treatment. Nevertheless, drug treatment has been effective in some areas—for example, Japan, Western Samoa, China, and French Polynesia. The other approach to filariasis control, by control of the vector mosquitoes, is dealt with below after further consideration of their ecology.

Figure 36-1 shows the areas in the world in which Bancroftian filariasis is known to occur. More extensive surveys may reveal further infected areas; for example, in India the known infected areas have steadily expanded as surveys have increased in thoroughness. Recent estimates in India are that 8 million people are infected, and that a population of 136 million is at risk from the disease. (ICMR 1971).

## *Culex pipiens* as a Vector and as a Nuisance

The member of the *Culex pipiens* complex most widespread as a vector of Bancroftian filariasis is *Culex quinquefasciatus*. *C. quinquefasciatus* is the principal vector in South America, on the coast of East Africa, and through much of Asia (see figure 36-1). However, in China and Japan the vector is *C. p. pallens*, whereas in Egypt it is *C. p. molestus* (Southgate 1979). *C. p. molestus* is also the vector of Rift Valley fever in Egypt,

Figure 36-2. *Larval and adult* Culex pipiens *mosquitoes.* (a) Two larvae and one pupa of *C. pipiens* mosquitoes suspended from the water surface. (b) An adult *C. pipiens* taking a blood meal. (Photos: R. Page, Department of Medical Entomology, London School of Hygiene and Tropical Medicine, London, UK)

where there were explosive epidemics during 1977 and 1978 (Hoogstraal and others 1979).

As indicated in figure 36-1, there are large areas of the world where *Culex pipiens* exists but there is no Bancroftian filariasis or where other species of mosquito are the main vectors of filariasis. *C. quinquefasciatus* has increased substantially in density in West African towns over the last 30 years, but at present filariasis is mainly a rural disease transmitted by *Anopheles* species in the region. There is evidence that West African strains of *C. quinquefasciatus* have a relatively low susceptibility to *W. bancrofti*, in contrast to Indian strains of *C. quinquefasciatus*. However, there is no cause for complacency about the danger of high *C. quinquefasciatus* populations in West Africa because of the risk that the local *W. bancrofti* will adapt to the new potential vector that has become available in recent years.

Not only in West Africa, but throughout the tropics in recent years there has been rapid urbanization without adequate excreta or sullage disposal systems, and this has led to massive increases in *C. quinquefasciatus* numbers. It is feared that this is leading to corresponding increases in filariasis incidence. Quite apart from their role as disease vectors, *C. quinquefasciatus* are the main nuisance mosquitoes of tropical urbanized areas. They can be extremely unpleasant, especially where most of the population sleep out of doors or in inadequately mosquito-proofed houses. In Calcutta (India), over 700 *C. quinquefasciatus* bites per person per night have been recorded (Gubler and Bhattacharya 1974). In Pondicherry (India) in January, over 9 million *Culex quinquefasciatus* mosquitoes are emerging per day, and 90 percent of these are breeding in drains (Menon and Rajagopalan 1980).

## The Association of *Culex pipiens* with Polluted Water

The breeding places of *C. pipiens* are mostly associated with sewage or sullage; stagnant open drains are perhaps the most prolific source. In many cases these drains were installed originally for storm water but, with the increased human population and inadequacies in the sewage disposal system, they have become the repository for sewage and sullage and tend to block and stagnate. Even if excreta are not disposed of in open drains, the use of such drains for sullage water creates equally important *C. pipiens* breeding places if the water is allowed to stagnate.

Pit latrines are another important breeding place, although this problem can be avoided if it is possible to dig the latrines so that they do not reach the water table. The installation of pit latrines in some villages in East Africa has been associated with invasion of these rural areas by *C. quinquefasciatus*. Where septic tanks and soakage pits are not made mosquito-proof, or are cracked, they too can become major breeding places. Examples of the relationship between *Culex* breeding and the availability of polluted water are given below.

It is thought that before 1926 Bancroftian filariasis transmitted by *C. quinquefasciatus* was limited in Sri Lanka to two towns. However, between 1926 and 1946 there was compulsory introduction of a system of bucket latrines in the southwestern part of the island and 30,000 were installed, each with an associated cement-lined pit to receive water used for ablution after defecation. These pits provided breeding sites for *C.*

*quinquefasciatus* and appeared to be associated with a major spread of filariasis. As part of a control program, 10,874 of the catch pits were converted to the water-seal type with a subsidy of 60 rupees each. Additional control measures included the use of organophosphate insecticides and campaigns to dispose of water-filled receptacles. In areas where the control program was in progress, the proportion of mosquitoes infected with *Wuchereria bancrofti* declined from 12–24 percent in 1949 to 0.7–3.3 percent in 1962 (Abdulcader 1967).

Afridi and Abdul Majid (1938) reported that measures against *C. quinquefasciatus* larvae had been taken throughout the inhabited area of New Delhi and for half a mile around it, but a severe *C. quinquefasciatus* biting nuisance was experienced during the months of April and May. It was thought that the breeding source was a suburban sewage farm in which very large numbers of larvae were found when sewage was pumped onto fields of grass and into stagnant connecting channels. Evidence that the city's problem did originate from this source came from trapping data that showed a gradation in mosquito density as one proceeded for several kilometers away from the sewage farm. Also, in the traps nearer the farm a higher proportion of males was obtained, which is generally an index of nearness to the breeding source. About 138,000 mosquitoes marked with silver dust were released at the sewage farm. Eleven marked mosquitoes were recaptured among the many thousands caught in the traps. Two of the recaptures were at more than 3.5 kilometers from the release point, the farthest being at 5 kilometers. These data indicate that *C. quinquefasciatus* from one massive breeding source could infest the whole city and emphasize the great dispersal power of this species when conditions are favorable.

Subra and Hebrard (1975) studied the ecology of *C. quinquefasciatus* larvae in the Comoro Islands, where this mosquito is the main vector of Bancroftian filariasis. Its main breeding places were pit latrines, soakage pits used for washing water, water-filled receptacles, and streams that became blocked by sand bars and stagnated in the dry season. Of the three main ethnic groups on the islands, the Sakalava (Malagasys) had soakage pits but no latrines in their villages; the Anjouan had pit latrines but no soakage pits; and the Moharais had both latrines and soakage pits. In semiurbanized areas the proportion of houses with pit latrines, soakage pits, or both was greater than in rural areas. Pit latrines did not contain water or become breeding places unless ground water entered them, which only occurred in deep pits and in the wet season when the water table rose. Thus, in villages with deep pit latrines *C. quinquefasciatus* biting densities rose dramatically in the wet season, whereas in villages with soakage pits only there was little seasonal variation. Chlorpyrifos (Dursban) was an effective and persistent larvicide in the pit latrines but has high mammalian toxicity. Drinking water wells were often dug close to pit latrines, and there was serious risk of diffusion of chlorpyrifos from flooded pit latrines to drinking water. Digging pit latrines less deeply so as not to reach the water table was considered the most effective control method. Temephos (Abate), with low mammalian toxicity and lower persistence than chlorpyrifos, was recommended for use as a larvicide in soakage pits.

Goettel, Toohey and Pillai (1980) studied *Aedes* and *Culex* breeding in Suva (Fiji). *C. quinquefasciatus* larvae were found in half of all septic tanks sampled. *C. quinquefasciatus* breeding, unlike that of *Aedes* species, showed no seasonal trend, and this suggests that breeding was associated more with permanent water bodies created by human water use than with ephemeral water bodies created by rainfall.

Bang, Sabuni and Tonn (1973) recorded the changes in mosquito populations between 1954 and 1971 in Dar es Salaam (Tanzania). Whereas urbanization and routine control measures had led to a reduction in malaria vectors, there had been a steady increase, despite control measures, in the population of *C. quinquefasciatus*. Part of the change was attributed to urbanization, which had led to a reduction of the clean pools suitable for *Anopheles* and to an increase in polluted water sources suitable for *C. quinquefasciatus*. Several permanent swamps known for decades as sources of malaria vectors had become *Culex* breeding sites owing to pollution from new human settlements. In an uncontrolled area of Dar es Salaam, for example, the *C. quinquefasciatus* population had increased since 1954 at an annual rate of 0.97 females per room.

The *Culex* breeding problem in Asian towns and cities has been reviewed by Singh (1967). Many towns in Asia are growing rapidly, and the sewerage arrangements are not keeping pace; there is a consequent increase in exposed polluted water that is available for breeding by *C. quinquefasciatus*. The situation is exemplified by Rangoon (Burma), where breeding is intense in drains, swampy ground near bucket latrines, pools polluted by effluent from the overloaded and defective waterborne sewage systems, unprotected septic tanks, and pit latrines in swampy ground. Both Hyderabad and Bangalore (India) were free of *C. quinquefasciatus* in the 1940s, but with increases in population and industrialization with poor sanitation the mosquito is now widespread, and

filariasis transmission is occurring. The presence of extensive *C. quinquefasciatus* breeding does not necessarily lead to intense filariasis transmission, since this is only possible where the adult mosquito life span is long enough to allow maturation of the parasite. In the humid parts of south India the mosquito life span is long throughout the year, but in northwest India it is only long enough to allow transmission from July to October.

Useful reviews of *Culex* breeding and filariasis in Asia and Africa have also been prepared by Gratz (1973) and Hamon and others (1967). From this and other literature, several matters of grave concern emerge. First, rapid urbanization unaccompanied by adequate excreta disposal and drainage infrastructure causes substantial increases in the *Culex* population, and these may cause an increase in filariasis transmission, prevalence, and intensity of infection. Second, *C. quinquefasciatus* is becoming increasingly numerous in urban areas of Africa where, at present, the vector of filariasis is the *Anopheles* mosquito. If *W. bancrofti* became adapted to transmission by *C. quinquefasciatus* in these areas of Africa, the consequences would be most serious. Third, there is some evidence that *Anopheles* and *Aedes* mosquitoes, commonly known as clean water breeders, are adapting to take advantage of the breeding opportunities provided by the proliferation of contaminated surface water bodies in fast-growing tropical cities. Chinery (1969) reported that *Anopheles gambiae* and *Aedes aegypti* were found breeding in earth drains, concrete drains, septic tanks, soakaways, and pit latrines in Accra (Ghana). Yao (1975) found *Anopheles stephensi* larvae in puddles of sewage effluent outside Lahore (Pakistan).

It should be pointed out that not all recent increases in *Culex pipiens* populations are associated with sewage and sullage. In villages near Delhi (India) large numbers of seasonally disused irrigation wells, which had been dug as part of the "green revolution" in agriculture, are major sources of *C. quinquefasciatus* breeding (Yasuno 1974). In Egypt a very substantial increase in the *C. p. molestus* population over the past 20 years has been promoted by poorly maintained wells, stagnant pools of spilled water near public taps and handpumps, and by the drastically altered irrigation practices and rising water tables that have followed the opening of the Aswan High Dam in 1971. The increased vector population, together with other factors, is responsible for a marked increase in the prevalence and intensity of infection with Bancroftian filariasis in Egypt and a widening geographical range (Southgate 1979).

## *Culex pipiens* Breeding in Waste Stabilization ponds

Under certain circumstances, waste stabilization ponds can become important breeding places for *Culex* mosquitoes. Beadle and Harmston (1958) studied twenty-six stabilization ponds in the USA. The main mosquito species found was *Culex tarsalis*, which is the vector of western equine encephalitis virus. The presence or absence of mosquito breeding in the ponds was closely correlated with the presence or absence of emergent or overhanging vegetation. Smith and Enns (1967) also studied stabilization ponds in the USA. Two ponds receiving animal waste were overloaded, frequently anaerobic, and had overhanging or emergent vegetation. They produced large numbers of *Culex pipiens* and other *Culex* species (30,000–60,000 larvae in a standard sample). This contrasted with 20–600 larvae in comparable samples from well-planned and well-maintained municipal sewage ponds.

Steelman and Colmer (1970) studied two ponds, also in the USA. One was newly dug, and the other had been used for 6 years for the reception of effluent from a pig farm. For the first 9 months of its life the new pond was filled with rainwater and from wells, but subsequently effluent from the farm began to be introduced. In the old pond large populations of *C. quinquefasciatus* larvae were found at each survey. In the new pond no *C. quinquefasciatus* was found, and the insect larvae before and for a few months after the introduction of effluent differed markedly from those in the old pond. In the old pond 7,000 to 13,000 coliform bacteria were found per milliliter. None was found in the new pond before introduction of effluent, but 7,000 per milliliter were found 5 months after this introduction, and at about this time *C. quinquefasciatus* larvae began to be found in the new pond. After 2 years the bacterial and insect populations of the two ponds had become very similar. Laboratory cage studies showed that suspensions of *Escherichia coli*, *E. freundii*, and *E. intermedia* were more attractive as oviposition sites for *C. quinquefasciatus* than were suspensions of *Aerobacter aerogenes*, and that all these suspensions were much more attractive than sterile water.

Yao (1975) reviewed limited data on mosquito breeding in waste stabilization ponds in India and concluded that pond colonization by vegetation was the main factor predisposing to breeding. In his own experiment outside Lahore (Pakistan), Yao (1975) studied four ponds (each 46 meters by 18 meters in plan and 1.2 meters deep) receiving domestic sewage and an experimental irrigation system. The ponds were lined with bricks, were in good operating order, and had no

emerging or encroaching vegetation. Two of the ponds showed no evidence of mosquito breeding, whereas the other two had minimal breeding in one month out of five. Many *C. quinquefasciatus* larvae were found in a roughly vegetated earth pond receiving effluent. Breeding ceased, however, when the vegetation was cleared. Vegetated puddles of effluent in the irrigation canals were also the sites of prolific breeding of *C. quinquefasciatus*, *C. theileri*, and *Anopheles stephensi*.

These studies have shown that the most important measure to prevent mosquito breeding is avoidance of vegetation hanging into or emerging through the surface of the ponds. Other ecological factors associated with the presence or absence of mosquito breeding in stabilization ponds have been studied in the USA, and studies of this subject in tropical countries would be very valuable. Little is known about the relationships between water pollution and female oviposition behaviour or about the ability of mosquito eggs to develop through their larval and pupal stages to become adults. The literature, reviewed by Darwall (1979), suggests that location, salt concentration, organic pollution, dissolved oxygen, surface tension, larval nutrients, and aquatic flora and fauna may be important determining factors. Even after 70 years of research, however, these relationships are poorly understood, and more research is urgently required.

## Methods for *Culex pipiens* Control

There are two approaches to *Culex* control, and successful control programs will almost always apply a combination of both of them. The two approaches, which are clear from the discussion above on preferred breeding sites, are modifications of the physical environment and the use of insecticides or other chemicals. Before describing these approaches, it is instructive to report a few case studies of control efforts in various environments.

### *Case studies in control*

White (1971) reviewed the *C. quinquefasciatus* control practices in East Africa and pointed out that malathion is unsuitable as a larvicide in polluted water because it is rapidly destroyed at acid or alkaline pH. Oiling of breeding places is frequently used, but in sullage pits detergents emulsify the oil and reduce its efficiency. Experiments with 110 milliliters of Flit mosquito larvicide oil per pit latrine gave poor control, but dosages of 450 milliliters of used engine oil per pit gave satisfactory results. A granular formulation of the organophosphate chlorfenvinphos (Birlane) was tested at a dosage of 2.5–5 grams per pit latrine. This completely prevented breeding for 9–10 days. One man can apply granules to thirty pits per hour, and it was estimated that the labor costs of a program would be 20 percent of those for oiling. Chlorfenvinphos has fairly high acute mammalian toxicity, and the possibility of seepage from pit latrines into streams must be considered.

In Guyana the breeding place of *C. quinquefasciatus* is principally pit latrines and secondarily clean water in drums and tanks. Spraying of latrines with oil was carried out at a dosage of about 650 grams each, which prevented breeding for 4–6 weeks. The cost of the oil for a monthly program of spraying over 2,000 latrines was US$71. In addition, all unused drums containing water were turned over, but some were used for drinking water and could not be emptied or sprayed. The campaign produced a dramatic reduction in the density of mosquitoes in houses. In addition, the filaricidal drug diethylcarbamazine was issued to all the inhabitants of the area. The result of the program was that the proportion of filaria-infected mosquitoes was reduced from 17.7 to 2.2 percent (Burton 1967).

Graham and others (1972) reported a field trial in Rangoon (Burma) of *C. quinquefasciatus* control in an area of 4 square kilometers. Breeding of the mosquitoes was mainly in open drains, which frequently became blocked by debris and the activities of rats. The drains had originally been built for stormwater, but they were now used for sewage because of the deterioration of the underground waterborne sewage system and the increase in population of the city. Additional breeding sources included soakage pits for sullage, improperly maintained septic tanks, and latrines. Much of the *C. quinquefasciatus* population rests and feeds out of doors, so that a house-spraying program against adult mosquitoes would have been inappropriate, as would the use of organochlorine insecticides because of resistance. Among the organophosphate insecticides, fenthion has been found to be one of the most effective and persistent as a larvicide in polluted water, and this chemical was used for the trial in the form of emulsifiable concentrate applied with a compression sprayer to larval breeding sites, especially drains and septic tanks. No resistance to fenthion appeared over the 3-year period of the trial. The overall effect of the 3-year program was an average reduction by 97 percent in the man-biting rate in the trial area compared with a comparison area, where the municipality's routine program of oiling breeding sources was in progress. Apart from the much greater effectiveness of the

fenthion program, it was considered that the higher labor and transport costs of applying the necessary large volumes of oil made oiling more expensive than the use of fenthion. The authors considered this field trial to be a model for what could be done in other tropical cities.

*Culex* control in Singapore was described by Chan Kai-Lok (1973). Surface flood water drains had originally been built as an *Anopheles* control measure, but these were now used for sullage and had become breeding places for *C. quinquefasciatus*. About 78 percent of the *C. quinquefasciatus* breeding in the city was in these concrete drains. Much of the remainder was in cracked septic tanks. A survey indicated that 6 percent of the septic tanks in the city were cracked and had *C. quinquefasciatus* breeding. Control measures used in the drains consisted of cleansing to improve the flow rate and oiling with Flit MLO refined light oil. Guppy fish (*Poecilia reticulata*) had been established in some concrete drains and canals and had had a significant effect in controlling *C. quinquefasciatus*. Sealing of inspection slabs on septic tanks reduced breeding.

An experiment to eradicate Bancroftian filariasis by vector control in a village in Nagasaki Prefecture (Japan) was recorded by Omori, Wada and Oda (1972). *Culex pipiens pallens* bred in ditches and polluted pools in the village and was the primary vector of Bancroftian filariasis. A program of vector control was carried out for 9 years using diazinon as a house spray against adult mosquitoes and for treatment of breeding sites. The latter treatments were carried out about twenty times per year during the mosquito breeding season, the target dosage being 1 milligram per liter. No filaricidal drugs were given, but all members of the village population (about 500) were examined annually for microfilariae. Entomological observations showed partial suppression of the *C. p. pallens* density in the first two years of treatment relative to the pretreatment year. During the last 7 years of treatment there was almost complete suppression of the vector. The prevalence of human microfilaria carriers declined from 14 percent before the trial to 0.5 percent at the end of it. This experiment is one of the very few demonstrations that filariasis in some areas can be drastically reduced solely by vector control if this is continued long enough for the adult worms in the human population to die out.

### *Control by modifying the physical environment*

Control methods based on environment modification must either eliminate the breeding site or make it impossible for the egg-laying female to reach the site. The particular strategies that might succeed in a particular place can only be designed after a detailed survey of *Culex* breeding in that area. In other words, control methods should be sharply focussed upon eliminating, or restricting access to, the known primary breeding sites. The main methods of environmental control include:

- Avoidance, if at all possible, of using open drains for sewage or sullage
- If the use of open drains for sewage or sullage is absolutely unavoidable for the time being, prevention of stagnation in the drains by frequent cleaning and the provision of adequate garbage disposal services
- Fitting and maintenance of mosquito-proof netting over ventilation pipes on septic tanks, aquaprivies and improved pit latrines
- Sealing and repairing covers on septic tanks, soakage pits, and improved pit latrines
- Fitting of water seals on pit latrines
- Clearance of vegetation from waste stabilization ponds.

Pit latrines are becoming increasingly commonplace in poor urban and rural communities. If they are well constructed and well maintained, they provide an acceptable excreta disposal method for those families that cannot afford more elaborate systems. But if the pits are wet, owing to a high water table or the addition of excess washing water, they provide an ideal breeding ground for *Culex pipiens* mosquitoes. Four approaches to overcoming this problem are recognized. First, to replace the drop-hole slab by a pour-flush slab, thus providing a water seal that prevents the entry or exit of mosquitoes to the pit. Second, to install a ventilation pipe with mosquito-proof gauze at the top (see figure 36-3). Third, to install exit traps over the squat holes. Fourth, to spray the pits with oil or insecticide.

The role of vent pipes (as on ventilated improved pit—VIP—latrines) in controlling *Culex* breeding was investigated in Dar es Salaam (Tanzania) and Gaborone (Botswana) by Curtis and Hawkins (1982). It was found that 67 percent of *Culex* mosquitoes emerging from the pits did so via the vent pipe rather than the squat slab. If the vent pipe was properly screened at the top, these mosquitoes could not escape. The effect of vent pipes on mosquito production was considerably less than their effect on blowfly production, of which about 90 percent went up the vent. Some latrines produced over 1,000 *Culex* per night through the squat slab, despite having a vent pipe.

Insect traps placed over the squat hole can be used on pit latrines with or without vent pipes (figure 36-3). The traps can be cheaply made of a box or tin partly covered in mosquito proof gauze and with a gauze cone in the bottom surface. Mosquitoes and flies enter the trap through this cone, presumably attracted upward from the pit by light and/or fresh air. They do not readily find their way out again, but if some do they go back into the pit. Insects caught in the traps die there in a day or two. No provision is made for removing the corpses, but ants have been seen removing dead mosquitoes. In future a lizard might be placed in each trap to eat captured insects. Provision is made, with appropriately placed flaps, to block other easy exit routes, but a precision fit is not necessary because insects emerging from pits take the obvious route into the well-lit and aerated trap rather than follow the more tortuous alternatives. This is one of the advantages of a trap as compared with a lid—mosquitoes will eventually find small imperfections in the fitting of a lid.

The principle of the trap is readily understood, and catches of mosquitoes or flies obtained from heavily infested pits are impressive and informative in showing the householder where an insect pest problem originates and that it can be stopped at source by the "self-help" measure of always ensuring that the trap is put back in place after using the pit. Initial trials in Dar es Salaam and Gaborone (Curtis 1980; Curtis and Hawkins 1982) on a variety of pit latrines, cess pits, and a soakage pit produced large catches of *C. quinquefasciatus* and blowflies. There were uniformly favorable reactions of householders to the traps, which were always found in place when checks were made. It remains to be seen whether such cooperation can be achieved over the long term by an appropriate initial program of public information followed by periodic checking on the traps for correct usage and for the repair of any damage. It also remains to be demonstrated that the area-wide use of traps is effective in suppressing the density of *C. quinquefasciatus* and blowflies in houses.

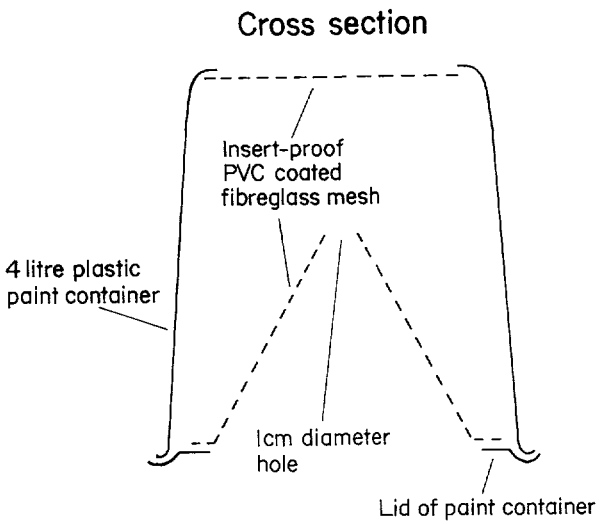
### *Control by insecticides and oils*

Control of *C. pipiens* breeding by environmental management methods alone is often not feasible, and insecticidal methods must also be considered. *C. pipiens* mosquitoes are now almost universally resistant to organochlorine insecticides such as DDT and dieldrin (Hamon and Mouchet 1967). Gas oil or used lubricating oil are routinely used on mosquito breeding places in many tropical cities as a control method. The method works by interfering with larval respiration, and studies in Guyana showed that it can be a very effective method in pit latrines if conscientiously applied (Burton 1967). Light oils with high spreading pressures have been developed especially for mosquito control ("Flit MLO") and are routinely used—for example, in Singapore—but relatively large amounts of transport and labor are required for an effective oiling program.

Organophosphate insecticides such as temephos (Abate), chlorfenvinphos (Birlane), chlorpyrifos (Dursban), diazinon (Basudin), and fenthion (Baytex) are lethal to mosquito larvae at extremely low concentrations (much less than 1 milligram per liter) and can be applied as emulsifiable concentrate or absorbed into granules, which greatly reduces the transport and labor required compared with oiling. The chemicals are fairly expensive, but temephos, chlorpyrifos, and fenthion remain effective for several weeks in stagnant polluted water, so that very frequent treatment is not necessary. Large-scale urban trials have shown substantial suppression of adult biting populations as a result of prolonged application of fenthion in Rangoon (Burma; Graham and others 1972), chlorpyrifos and temephos in Bobo-Dioulasso (Upper Volta; Subra, Bouchite and Gayral 1970), chlorpyrifos in Dar es Salaam (Tanzania; Bang, Sabuni and Tonn 1975), chlorpyrifos in Morogoro (Tanzania; Mrope, Bang and Tonn 1974), and chlorfenvinphos in Tanga (Tanzania; White 1971). It has been shown in Japan that applications of diazinon to breeding places several times each year for 9 years suppressed *C. p. pallens* populations and reduced the incidence of filariasis virtually to extinction (Omori, Wada and Oda 1972).

Despite the success achieved with organophosphate insecticides, it would be unwise to rely on them exclusively. It seems possible that they might adversely affect the essential microbial activity in waste stabilization ponds, septic tanks, and aquaprivies, and this question demands investigations of the kind already conducted with regard to the use of kerosene as an insecticide (Razeghi, Lawrence and King 1972). In addition, there are reports from many parts of the world of the evolution of resistance to organophosphates in *C. pipiens* mosquitoes (see, for instance, Curtis and Pasteur 1981; Hamon and Mouchet 1967).

Recent experience in Tanzania provides a good example of the problems caused by the development of organophosphate resistance in *C. quinquefasciatus*. Following the trials in Dar es Salaam and Morogoro described above, chlorpyrifos was adopted in the early 1970s for routine spraying (every 10 weeks) of all pit



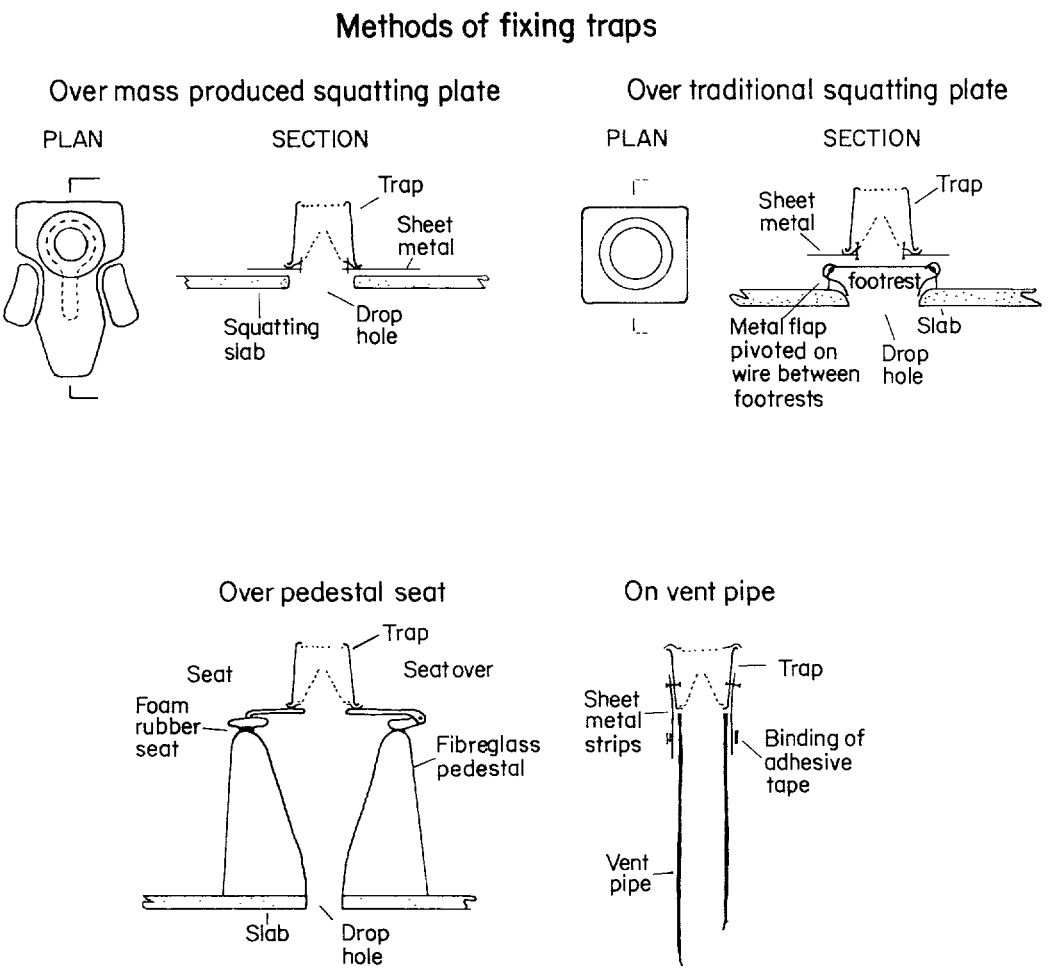




Figure 36-3. *Insect traps for pit latrines.* The traps shown can be fitted over the squat hole or seat of a latrine, or over the vent pipe, to catch flies and mosquitoes emerging from the pit. (from Curtis and Hawkins 1982; reproduced by permission of the Royal Society of Tropical Medicine and Hygiene)

latrines and soakage pits in Dar es Salaam. When the program was initiated, the insecticide residue remaining up to 10 weeks after a spraying was sufficient to give continuous control of larvae. However, unpublished data of Curtis and others indicate that the resistance that has evolved in Tanzania is now seriously interfering with the effectiveness of the program. Freshly sprayed chlorpyrifos is able to kill the resistant larvae, but after the insecticide residue has been degraded, diluted, or both, for 1 to 4 weeks it is no longer able to kill them. Thus, mosquito breeding can proceed in the sprayed pits for a large fraction of the 10-week spraying cycle. The program already costs about US$135,000 per year for the importation of insecticide, and increasing the frequency of spraying would require considerable additional expenditure, as well as the employment of more spray men.

It may be that practical alternatives to conventional insecticides will eventually become widely applicable using insect growth regulators, lipid monolayers (Levy and others 1980), insoluble foams, biological control by pollution-tolerant fish, algae (Ilyaletdinova 1978), microorganisms, or genetic systems based on cytoplasmic incompatibility and genes conferring nonsusceptibility to filaria. All of these methods, however, should be viewed only as ancillary to the installation and maintenance of well-designed excreta and sullage disposal systems that minimize the breeding opportunities available to *C. pipiens*.

## Conclusions

It is clear from this review that water supply projects and on-site sanitation systems have the potential for greatly increasing the population of *C. pipiens* mosquitoes in tropical towns and cities. Of special concern are inadequately maintained open drains, flooded pit latrines, soakage pits, and septic tanks. In villages, the construction of pit latrines in areas with high water table, and increased water usage leading to ponded sullage, may bring the traditionally urban problem of *C. quinquefasciatus* to these rural sites.

Minimizing this problem depends on a carefully thought-out combination of correct design features for drainage and on-site sanitation, of appropriate self-help action by the community (for intance, using exit traps on pit latrines and keeping open drains free from garbage), and of larvicide-spraying programs. It is necessary for those designing and implementing water supply and sanitation schemes to discuss their work with entomologists having detailed local knowledge, and sullage disposal systems that will minimize the mosquito-control activities. This is especially important in those parts of the world where *C. pipiens* mosquitoes are the vectors of Bancroftian filariasis, and it will become even more crucial if *Anopheles* and *Aedes* species adapt locally to use polluted water as breeding sites in urban areas.

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# 37

# Flies, Cockroaches, and Excreta

MANY INSECTS are associated with excreta. Those of importance to health, however, are mainly from two groups of insects: the two-winged flies (Diptera) and the cockroaches (Dictyoptera). There are many species of Diptera, including the mosquitoes (dealt with in the preceding chapter), so that this chapter concentrates on other flies and the cockroaches.

Patches of animal feces are worlds unto themselves, with parasites, predators and dung feeders living as a community together (Laurence 1977). Manmade environments with innovations such as sanitation and waste disposal units are invaded and colonized from more natural breeding places. Waste disposal provides two attractive materials for the development of insects—rich organic material and water. The kinds of insect found breeding in human waste disposal systems are, in consequence, those that breed in various forms of decaying organic material, including feces, or those that breed in freshwater and yet tolerate organic pollution. Relatively few species are able to take advantage of the new opportunities for breeding provided by waste disposal, but these often appear in very large numbers. The balance of a more varied fauna found in the natural breeding places may be lacking in the new manmade habitats. Consequently, the numbers of insects may build up to a level sufficient to cause a definite nuisance or endanger health. The presence of animal life in waste treatment systems, where there is a mixed culture of organisms (including the insects), is also part of the process of purification and breakdown of the organic material (Lloyd 1945; Usinger and Kellen 1955).

## Flies Associated with Waste Disposal

The most common flies associated with waste disposal are found in eleven families (Laurence 1977). The most important families are the flies that transmit disease organisms from man to man: the mosquitoes, or family Culicidae; and the flies that breed in fecal material and also come into contact with man as adults by feeding on his food (the housefly and the blowfly, families Muscidae and Calliphoridae). Other flies cause a nuisance by invading human habitation (families Psychodidae, Chironomidae, and Anisopodidae), may land in food, and are known to cause contact and inhalation allergy in sensitized persons. In addition, other families of fly (Stratiomyidae, Syrphidae, Sepsidae, Ephydridae, Phoridae, and Sphaeroceridae) are very common on sewage installations or in cess pits, but normally have little contact with man and are less of a problem except that they can cause confusion in the identification of the more important sewage-breeding nuisances.

Among the flies that breed in or feed on excreta, two families are implicated in particular in the carriage of fecal material: the Muscidae and the Calliphoridae. The most important species are the housefly (*Musca domestica*) and species of the tropical green blowfly (*Chrysomya*). The adults of these flies will enter houses and shops readily and are attracted to human food as well as to feces and garbage around the home. They are medium to large flies, 7–10 millimeters long, and have greyish (*Musca*) (figure 37-1) or green (*Chrysomya*) body color. The larvae are maggot-like and 10–12 millimeters long when fully grown. The three larval stages are found in excreta or mixtures of excreta and decaying vegetable matter. For the breeding of the housefly, *Musca domestica*, solid, moist, and fermenting matter are required; other species of *Musca* breed in wetter materials. Human feces are attractive to houseflies mainly in their solid state. The presence of another fly, *Hermetia*, in latrines renders the contents soupy and less attractive to houseflies. Reduction of the water content, by drainage or by killing the *Hermetia* with insecticides, causes an increase in housefly breeding (Kilpatrick and Schoof 1959). In contrast, the larvae of the blowfly, *Chrysomya*, are found in more liquid feces and may liquify masses of fecal material.

Figure 37-1. *The common housefly*, Musca domestica. (Photo: Wellcome Museum of Medical Science)

The housefly will develop from egg to adult in 10 to 20 days over the temperature range 20–30°C (the higher the temperature, the shorter the period of development), with a minimum developmental period of about 1 week. No development takes place below 12°C, and eggs, larvae, and pupae are killed at 47°C (Keiding 1976). *Chrysomya* (blowfly) larvae develop from egg to adult in Sri Lanka at room temperature in 8.5 days.

A survey of the insect fauna in some latrines in east Africa and Taiwan was conducted in 1978 by B. R. Laurence. The results are summarized in table 37-1, which shows that *Chrysomya* was ubiquitous. In east Africa *Chrysomya putoria* was the species involved, whereas in Taiwan it was *Chrysomya megacephala*. Similarly, Lien and Chen (1974) reported that the majority of flies breeding in vaults in Taipei (Taiwan) were *Chrysomya megacephala*. Pit latrines are notorious as breeding places for blowflies. Raybould (1966) reported that, at Amani in Tanzania, ninety pit latrines produced 3.5 kilograms of *Chrysomya putoria* in a single month (one adult *Chrysomya* weighs about 50 milligrams).

## Cockroaches

Cockroaches are attracted by the moisture of waste disposal systems of various kinds. They are also potential carriers of fecal pathogens, and they live in and around human dwellings. They often visit human food when they emerge at night from their noisome daytime hiding places. Cockroaches, in contrast to the very rapid turnover of the fly population, take several months to more than a year to develop to the adult stage, but large populations of adult and young cockroaches may be found together in the same habitat. Also, young cockroaches resemble the adult insect, whereas fly larvae have a very different appearance from the adult fly. Flies are able to breed in much more transient habitats than cockroaches.

## Flies, Cockroaches, and Health

Disregarding *Culex* mosquitoes, which are discussed in chapter 36, there are two ways in which insects related to excreta may affect man. First, large

Table 37-1. *Insect fauna of "dry" latrines*

| *Location* | *Type of toilet* | *Insect fauna* |
|---|---|---|
| Tanzania, Kenya | Pit latrines including Reed Odorless Earth Closets (ROECs) | *Chrysomya*, cockroaches<br>*Culex quinquefasciatus*, if flooded |
| Tanzania | Continuous 'multrum' type composting toilets | *Chrysomya* in feces and vegetable fiber<br>*Musca* in dry compost<br>*Telmatoscopus*, *Hermetia* and *Eristalis* in wet compost<br>Phorid flies, cockroaches |
| Tanzania | Batch double-vault composting toilets | *Chrysomya* in feces<br>Phorid flies, cockroaches |
| Taiwan | Latrine vaults | *Chrysomya* Phorid flies *Telmatoscopus*, *Hermetia*, *Eristalis* |

*Note:* *Chrysomya* = blow fly; *Eristalis* = rat tailed maggot; *Hermetia* = soldier fly; *Musca* = housefly; *Telmatoscopus* = moth fly.
*Source:* B. R. Laurence, unpublished data.

numbers of flies will breed in the various environments associated with waste disposal systems. Some of these may have a close association with man (the so-called synanthropic species) and can cause a nuisance. More seriously, they can cause allergy, with sensitization reactions (skin rash and asthma) as a response to the presence of the bodies of the flies (see, for instance, Ordman 1946; Phanichyakarn, Dockhorn and Kirkpatrick 1969). Second, and of greater importance, is the potential role of flies and cockroaches, which either breed in excreta or eat excreta, in disseminating fecal pathogens. It is on this aspect that we concentrate here.

Any insect that breeds in excreta or visits excreta to feed may carry particles of feces from place to place. This may be done either on the legs or other parts of the external body surface or by the insect vomiting or depositing pathogenic organisms, previously ingested, in the feces. Either of these mechanisms can assist the dissemination of human fecal pathogens in the environment.

## Transmission of Excreted Pathogens

Many studies have shown that insects that breed in excreta, or feed on it, may carry human pathogens on their bodies or in their gut. The massive literature on this subject shows that practically every excreted pathogen has been isolated at some time in a viable state from a fly or cockroach. This includes the excreted viruses (see, for instance, Melnick and Dow 1953), the excreted bacteria (see, for instance, Bidawid and others 1978; Burgess and Chetwyn 1978; Cox, Lewis and Glynn 1912; Steinhaus and Brinley 1957), the excreted protozoal cysts (see, for instance, Frye and Meleney 1936, Gupta and others 1972; Pipkin 1949; Rendtorff and Holt 1954; Root 1921), and the excreted helminth eggs and larvae (see, for instance, Gupta and others 1972; Oyerinde 1976; Round 1961). The literature on insectborne pathogens is very extensive and has been reviewed by Greenberg (1971, 1973) for flies and by Cornwell (1968, 1976) and Roth and Willis (1960) for cockroaches.

The medical significance of the carriage of excreted pathogens by insects depends in part upon the behavior of the insect and in part upon the other modes of transmission of these pathogens. Insects that enter human dwellings and visit human food are especially likely to promote disease transmission, and it is for this reason that the flies *Musca* and *Chrysomya* and cockroaches are so often implicated. Given that these insects are moving fecal pathogens into houses and onto food, however, it remains unclear what their contribution is to the spread of particular infections. A feces-fingers-food cycle is more direct and more probable than a feces-insect-food cycle.

The main epidemiological evidence of the importance of flies and cockroaches in the transmission of enteric infections comes from the results of surveys of infection in the human population before and during insecticidal control programs. Decrease of human enteric infection (especially *Shigella* infections) has sometimes been recorded in the sprayed areas (see, for instance, Abdel-Gawaad and El-Gayar 1972; Mackie and others 1956; Watt and Lindsay 1948; Wolff, van

Zijl and Roy 1969). In some experiments a rise in human infection accompanied the development of insecticide resistance in the flies (see, for instance, Lindsay, Stewart and Watt 1953).

## Methods of Fly and Cockroach Control

Whatever their exact role in disease transmission, it is always desirable to control any fly or other insect nuisance associated with excreta disposal. Control of human excreta alone, however, will not be sufficient because the insects will with some certainty be breeding elsewhere (in the excreta of other animals and in other materials), and the flies will disperse quickly into the controlled areas. For instance, surveys in three cities in the USA (Charleston, Phoenix, and Topeka) showed that the primary site for housefly and blowfly breeding was garbage (Schoof, Mail and Savage 1954). A survey of garbage disposal pits in an army camp in the USA also showed them to be a major breeding site (Mathis, Schoof and Mullenix 1969). In Dar es Salaam (Tanzania), the majority of flies emerging from pit latrines were *Chrysomya putoria*, whereas 9 percent of flies caught in kitchens were *Musca domestica*, thus clearly indicating that major breeding activity was taking place in other sites (Bang, Sabuni and Tonn 1975).

The control of fly and cockroach breeding in association with human excreta may not, therefore, have any measurable effect on the total fly and cockroach population. However, such control will reduce the population of flies and cockroaches that have been in contact with human excreta, and—because many excreted pathogens are found exclusively, or almost exclusively, in human excreta (for instance, poliovirus, hepatitis A virus, *Shigella*, *Vibrio cholerae*, *Entamoeba histolytica*, and the eggs of human roundworms and hookworms)—this reduction could be epidemiologically important (McCabe and Haines 1957).

### *Modifying the physical environment*

An ideal sanitary unit should exclude flies and other insects and prevent access to the feces but should not inhibit people from using it. The success of fly-proofing methods (self-closing lids, screening, darkness, proper coverage of the fecal material) depends upon the acceptance and maintenance of these methods by the local population. An enclosed, dark and fly-proof box that overheats would clearly not be acceptable as a toilet to the residents of tropical countries, and would probably also attract cockroaches. Hence, there is no easy, universal solution.

The feces of different animals attract different associations of insect species, and this reflects the differing consistencies of the feces. Sanitary measures designed to prevent housefly breeding, such as bored hole latrines, may not be successful against the blowfly. Wherever possible, improvement in sanitation design should aim to protect the feces from the visiting insects and provide conditions of disposal unsuitable for the development of the larval stages in the fecal material.

Housefly eggs are killed at 42°C, larvae at 47°C, and pupae at 45°C (Keiding 1976). Hence, composting should aim at maintaining the lethal temperature throughout the consolidated mass of excreta. Low temperatures at the edges will still permit larval development to adult fly.

A variety of design suggestions for controlling fly breeding in latrines, animal manure, and garbage are given by Busvine (1982). Fly control in garbage disposal plants using composting and sanitary landfill is described by Alvarez, Blanton and Putnam (1972) and Black and Barnes (1956), respectively. Data on fly control in composting plants for excreta plus garbage in China are given by Scott (1952).

The most important advances in thinking on the control of fly breeding in pit latrines concern the role of pit ventilation and the use of exit traps. Modern concepts of pit latrine design include a pit vent pipe or chimney, one major role of which is to exhaust foul gases and thus make the latrine odor-free and pleasant (see, for instance, Feachem and Cairncross 1978). Experiments conducted in Botswana, Tanzania, and Zimbabwe have shown that vent pipes also play an important role in reducing fly production by pit latrines. In Zimbabwe (Morgan 1977), four pit latrines were built in a row, two with vent pipes and two without, and were used for 6 months prior to the start of the experiment. During 2.5 months, 13,953 flies were trapped from the unvented pits, but only 146 were trapped from the vented pits. Most flies were *Chrysomya*. The studies in Botswana and Tanzania (Curtis and Hawkins 1982) found that, in vented pit latrines with their doors kept closed, about 90 percent of the emerging flies (mainly *Chrysomya putoria*) went up the vent pipe and were caught by the gauze at the top. When the doors were left open, only about 50 percent of flies attempted to exit via the vent pipe; the rest left through the drop hole. This suggests that keeping the latrine dark by closing the door encourages the newly emerged flies to go toward the

main light source, which comes down the vent pipe. The studies in Botswana and Tanzania also showed that female flies attempting to enter latrines to lay eggs were strongly attracted by the fecal odors from the vent pipes and therefore tried to enter the pits by flying down the vent pipes. They were prevented from doing this by the gauze at the top end of the vent pipe. Thus, screened vent pipes reduce the numbers of gravid female flies gaining access to the pits and prevent a considerable proportion of young flies from leaving the pits. [See note on page 81.]

The second important new approach to fly control in pit latrines is the use of an exit trap on the squatting hole instead of a lid. This is described in chapter 36 and by Curtis (1980) and Curtis and Hawkins (1982) and requires large-scale field testing.

*Insecticides and other chemicals*

The ideal cheap and nonbulky compound that reduces the attractiveness of feces to flies has yet to be found. Addition to feces of diesel oil, chloride of lime, borax, paradichlorbenzene, and thiourea, as well as other larvicides—some toxic to man—has been used. Such chemicals should be used with caution whenever the excreta will subsequently undergo some biological treatment process, such as digestion or composting, that could be adversely affected by the added chemicals. Caution is also required if the excreta may be reused in agriculture or aquaculture.

Fly control programs with the use of chlorinated hydrocarbon insecticides, such as DDT and BHC, have produced widespread resistance in the housefly, and resistance to other insecticides has also developed. Resistance to insecticides has also been recorded in *Chrysomya*. In some control programs resistance of flies to insecticides has developed extremely rapidly—for instance, in about 3 months (Lindsay, Stewart and Watt 1953). In Georgia (USA) the use of dieldrin, BHC, or chlordane in pit latrines greatly increased the breeding of insecticide-resistant *Musca domestica* because it reduced the population of *Hermetia illucens* larvae, which were making the pit contents too liquid for housefly breeding (Kilpatrick and Schoof 1959).

Flypapers, insecticide-impregnated cords and plastic blocks, baited fly traps, and poison baits have all been used against the adult fly. Distribution of this type of equipment is a possibility given the organization, understanding, and cooperation of the local population.

The control of insects other than houseflies and blowflies presents similar problems. As with the flies, widespread insecticide resistance is known in cockroaches. Fumigation of sewers, the use of residual insecticides as sprays or lacquer paints, and poison baits have been found to be effective, but the control of cockroach infestation in excreta disposal units is part of a wider problem of cockroach control in the human community. A combination of water, food, and darkness is ideal for renewed cockroach infestation in the tropics. A comprehensive review of cockroach control strategies is given by Cornwell (1968, 1976), and a recent survey of approaches to cockroach control in Hungary is presented by Bajomi and Elek (1979).

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