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) Average	Range	Source[b]
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*

Constituent	*Approximate composition (percent of dry weight)* 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*

				BOD_5 content						
Population	*Assumed adult fecal weight (grams daily)*[a]	*Assumed adult urine weight (kilograms daily)*	*Estimated water in feces (percent)*	*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)						
		Enterobacteria[a,b]	*Enterococci*[b]	*Lactobacilli*	*Clostridia*	*Bacteroides*	*Bifidobacteria*	*Eubacteria*
Largely carbohydrate	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}	2.5×10^{11}	5,000
Bacteria					
Pathogenic *E. coli*[e]	?	10^{8}	10^{10}	?	?
Salmonella spp.	7	10^{6}	10^{8}	3.5×10^{11}	7,000
Shigella spp.	7	10^{6}	10^{8}	3.5×10^{11}	7,000
Vibrio cholerae	1	10^{6}	10^{8}	5×10^{10}	1,000
Protozoa					
Entamoeba histolytica	30	15×10^{4}	15×10^{6}	2.25×10^{11}	4,500
Helminths					
Ascaris lumbricoides	60	10^{4}[f]	10^{6}	3×10^{10}	600
Hookworms[g]	40	800[f]	8×10^{4}	1.6×10^{9}	32
Schistosoma mansoni	25	40[f]	4×10^{3}	5×10^{7}	1
Taenia saginata	1	10^{4}	10^{6}	5×10^{8}	10
Trichuris trichiura	60	2×10^{3}[f]	2×10^{5}	6×10^{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 (bathroom and laundry)	20
Laundry (percent)	22	0		
Drinking				
Animals (percent)	2	8	0	0
Humans (percent)	45 (humans and kitchen)	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 1.9×10^7 per 100 milliliters irrespective of garbage content. After 24 hours of storage, this count had increased to 5.4×10^8, 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–(8.2×10^3)	220	1–(2.5×10^3)	44	1–(7×10^4)
Clothing washwater	18,000	85–(8.9×10^5)	1,400	9–(1.6×10^4)	210	1–(1.3×10^6)
Clothing rinsewater	5,300	190–(1.5×10^5)	320	35–(7.1×10^3)	75	1–(2.3×10^5)

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.

Literature Cited

Balasegaram, M. and Burkitt, D. P. (1976). Stool characteristics and Western diseases. *Lancet*, **1**, 152.

Bennett, E. R., Linstedt, K. D. and Felton, J. T. (1974). Rural home wastewater characteristics. In *Proceedings of the National Home Sewage Disposal Symposium, Chicago, Dec. 9–10, 1974*, pp. 74–78. St. Joseph, Mich.: American Society of Agricultural Engineers.

Burkitt, D. P., Walker, A. R. P. and Painter, N. S. (1972). Effect of dietary fibre on stools and transit-times, and its role in the causation of disease. *Lancet*, **2,** 1408–1412.

——— (1974). Dietary fiber and disease. *Journal of the American Medical Association*, **229,** 1068–1074.

Connell, A. M. and Smith, C. L. (1974). The effect of dietary fibre on transit times. In *Proceedings of the Fourth International Symposium on Gastrointestinal Motility, Banff, Alberta, 1973*; eds. Daniel, E. E. and others, pp. 365–368. Vancouver: Mitchell Press.

Cort, W. W., Otto, G. F. and Spindler, L. A. (1930). Investigations on *Ascaris lumbricoides* and the associated intestinal helminths of man in southwestern Virginia. *American Journal of Hygiene*, **11,** 1–55.

Cranston, D. and Burkitt, D. P. (1975). Diet, bowel behaviour and disease. *Lancet*, **2,** 37.

Crofts, T. J. (1975). Bowel-transit times and diet. *Lancet*, **1,** 801.

Crowther, J. S., Drasar, B. S., Hill, M. J., MacLennan, R., Magnin, D., Peach, S. and Teoh-Chan, C. H. (1976). Faecal steroids and bacteria and large bowel cancer in Hong Hong by socio-economic groups. *British Journal of Cancer*, **34,** 191–198.

Drasar, B. S. (1974). Some factors associated with geographical variations in the intestinal microflora. In *Normal Microbiological Flora of Man*, Society for Applied Bacteriology Symposium no. 3; eds. Skinner, F. A. and Carr, J. G., pp. 187–196. London: Academic Press.

Evison, L. M. and James, A. (1977). Microbiological criteria for tropical water quality. In *Water, Wastes and Health in Hot Climates*; eds. Feachem, R. G. A., McGarry, M. G. and Mara, D. D., pp. 30–51. London: John Wiley.

Feachem, R. G. A. (1977). Environmental health engineering as human ecology: an example from New Guinea. In *Subsistence and Survival—Rural Ecology in the Pacific*, eds. Bayliss Smith, T. and Feachem, R. G. A., pp. 129–182. London: Academic Press.

Feachem, R. G. A., Burns, E., Cairncross, A., Cronin, A., Cross, P., Curtis, D., Khalid Khan, M., Lamb, D. and Southall, H. (1978). *Water, Health and Development: An Interdisciplinary Evaluation*. London: Tri-Med Books.

Fuchs, H.-M., Dorfman, S. and Floch, M. H. (1976). The effect of dietary fiber supplementation in man. II. Alteration in fecal physiology and bacterial flora. *American Journal of Clinical Nutrition*, **29,** 1443–1447.

Goldsmith, H. S. and Burkitt, D. P. (1975). Stool characteristics of black and white Americans. *Lancet*, **2,** 407.

Gotaas, H. B. (1956). *Composting: Sanitary Disposal and Reclamation of Organic Wastes*. Monograph Series 31. Geneva: World Health Organization.

Goy, J. A. E., Eastwood, M. A., Mitchell, W. D., Britchard, J. L. and Smith, A. N. (1976) Fecal characteristics contrasted in the irritable bowel syndrome and diverticular disease. *American Journal of Clinical Nutrition*, **29,** 1480–1484.

Gray, H. and Tainter, M. L. (1941). Colloid laxatives available for clinical use. *American Journal of Digestive Diseases*, **8,** 130–139.

Greenberg, C. J. (1976). *Studies on the Fibre in Human Diets*. Ph.D. dissertation. Cambridge: University of Cambridge.

Hindhaugh, G. M. A. (1973). Nightsoil treatment. *The Consulting Engineer*, **37, 47** and **49**.

Hoogstraal, H., Meegan, J. M. and Khalil, G. M. (1979). The Rift Valley fever epizootic in Egypt, 1977–78: ecological and entomological studies. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **73,** 624–629.

Huponu-Wusu, O. O. and Daniel, S. O. (1977). Public health aspects of housing in Nigeria: observations on the human factors in refuse disposal in Lagos. *Health and Hygiene*, **1,** 153–158.

Hypes, W. D. (1974). Characterization of typical household gray water. In *Manual of Gray Water Treatment Practice*, ed. Winneberger, J. H. T., pp. 79–88. Ann Arbor, Mich.: Ann Arbor Science Publishers.

International Agency for Research on Cancer, Instestinal Microecology Group (1977). Dietary fibre, transit time, faecal bacteria, steroids, and colon cancer in two Scandinavian populations. *Lancet*, **2,** 207–211.

Kalbermatten, J. M., Julius, D. S., Gunnerson, C. G. and Mara, D. D. (1982). *Appropriate Sanitation Alternatives: A Planning and Design Manual*. World Bank Studies in Water Supply and Sanitation **2**. Baltimore, Md.: Johns Hopkins University Press.

Laak, R. (1974). Relative pollution strength of undiluted waste materials discharged in households and the dilution waters used for each. In *Manual of Grey Water Treatment Practice*, ed. Winneberger, J. H. T., pp. 68–78. Ann Arbor, Mich.: Ann Arbor Science Publishers.

McGarry, M. G. and Stainforth, J. (1978). *Compost, Fertilizer, and Biogas Production from Human and Farm Wastes in the People's Republic of China*. Ottawa: International Development Research Centre.

Mata, L. J., Carrillo, C. and Villatoro, E. (1969). Fecal microflora in healthy persons in a preindustrial region. *Applied Microbiology*, **17,** 596–602.

Otto, G. F., Cort, W. W. and Keller, A. E. (1931). Environmental studies of families in Tennessee infested with *Ascaris*, *Trichuris* and hookworm. *American Journal of Hygiene*, **14,** 156–193.

Otto, G. F. and Spindler, L. A. (1930). Effect of partial sanitation on infestation with intestinal parasites in southwest Virginia. *Southern Medical Journal*, **23,** 556–560.

Pescod, M. B. (1971). Wastewater solids utilization and disposal in tropical developing countries. In *Water Supply and Wastewater Disposal in Developing Countries: Proceedings of a Water Supply and Sanitation Seminar, Bangkok, January 19–23, 1970*, eds. Pescod, M. B. and Okun, D. A., pp. 208–226. Bangkok: Asian Institute of Technology.

Pradt, L. A. (1971). Some recent developments in nightsoil treatment. *Water Research*, **5,** 507–521.

Small Scale Waste Management Project (1978). *Management* *of Small Waste Flows*. Report EPA-600/2-78-173. Cincinnati, Ohio: US Environmental Protection Agency.

Southgate, B. A. (1979). Bancroftian filariasis in Egypt. *Tropical Diseases Bulletin*, **76**, 1045–1068.

Tandon, R. K. and Tandon, B. N. (1975). Stool weights in North Indians. *Lancet*, **2**, 560–561.

Walker, A. R. P. (1975). Effect of high crude fiber intake on transit time and the absorption of nutrients in South African Negro schoolchildren. *American Journal of Clinical Nutrition*, **28**, 1161–1169.

White, A. U. (1977). Patterns of domestic water use in low-income communities. In *Water, Wastes and Health in Hot Climates*, eds. Feachem, R. G. A., McGarry, M. G. and Mara, D. D., pp. 96–112. London: John Wiley.

White, G. F., Bradley, D. J. and White, A. U. (1972). *Drawers of Water: Domestic Water Use in East Africa*. Chicago: University of Chicago Press.

Winfield, G. F. (1937). Studies on the control of faecal-borne diseases in North China. III. Family environmental factors affecting the spread of *Ascaris lumbricoides* in a rural population. *Chinese Medical Journal*, **51**, 643–658.

Winneberger, J. H. T. (1974). *Manual of Gray Water Treatment Practice*. Ann Arbor, Mich.: Ann Arbor Science Publishers.

Witt, M., Siegrist, R. and Boyle, W. C. (1974). Rural household wastewater characteristics. In *Proceedings of the National Home Sewage Disposal Symposium, Chicago, December 9–10, 1974*, pp. 79–88. St. Joseph, Mich.: American Society of Agricultural Engineers.

WHO (1970). *Sewerage Planning in the Greater Taipei Area: A Master Plan Report*, Report no. WHO/UNDP/SF/CHA-27. Geneva: World Health Organization.

——(1979). *Human Viruses in Water, Wastewater and Soil*. Technical Report Series no. 639. Geneva: World Health Organization.