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 1.5×10^7 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

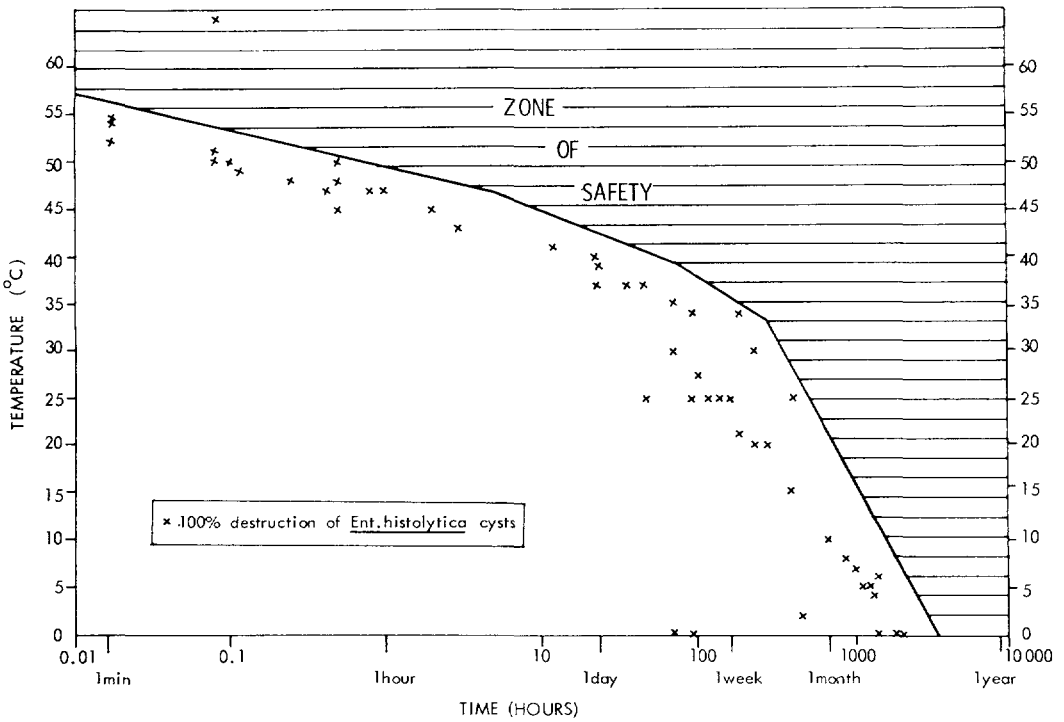

Figure 20-2. *The influence of time and temperature on* Ent. histolytica *cysts.* The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

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.

Literature Cited

Andrews, J. (1934). The retention of *Endamoeba histolytica* cysts under finger-nails. *American Journal of Tropical Medicine*, **14**, 439–441.

Arafa, M. S., El-Ridi, A. M. S., Ezzat, H. O. A. and Makhlouf, L. M. (1978). A seroparasitological study of *Entamoeba histolytica* in Egypt. *Journal of the Egyptian Society of Parasitology*, **8**, 329–332.

Arceivala, S. J., Lakshminarayana, J. S. S., Alagarsamy, S. R. and Sastry, C. A. (1970). Health aspects. In *Waste Stabilization Ponds: Design, Construction and Operation in India*, pp. 87–95. Nagpur, India: Central Public Health Engineering Research Institute.

Beaver, P. C. and Deschamps, G. (1949*a*). The viability of *E. histolytica* cysts in soil. *American Journal of Tropical Medicine*, **29**, 189–191.

———(1949*b*). The effect of acetic acid on the viability of *Endamoeba histolytica* cysts. *American Journal of Tropical Medicine*, **29**, 193–197.

Bray, R. S. and Harris, W. G. (1977). The epidemiology of infection with *Entamoeba histolytica* in The Gambia, West Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **71**, 401–407.

Brooke, M. M., Donaldson, A. W. and Brown, E. (1954). An amebiasis survey in a Veterans Administration Hospital. Chamblee, Georgia, with comparison of technics. *American Journal of Tropical Medicine and Hygiene*, **3**, 615–620.

Brooke, M. M., Healy, G. R., Levy, P., Kaiser, R. L. and Bunch, W. L. (1963). A sample survey of selected areas in and near Little Rock, Arkansas, to assess the prevalence of *Entamoeba histolytica*. *Bulletin of the World Health Organization*, **29**, 813–822.

Brooke, M. M., Melvin, D. M., Sappenfield, R., Payne, F., Carter, F. R. N., Offutt, A. C. and Frye, W. W. (1955). Studies of a water-borne outbreak of amebiasis, South Bend, Indiana. III. Investigation of family contacts. *American Journal of Hygiene*, **62**, 214–226.

Chandler, A. C. (1954). A comparison of helminthic and protozoan infections in two Egyptian villages two years after the installation of sanitary improvements in one of them. *American Journal of Tropical Medicine and Hygiene*, **3**, 59–73.

Chang, S. L. (1943). Studies on *Endamoeba histolytica*. II. Observations concerning encystation, maturation and excystation of *E. histolytica*, and on the longevity of culture-induced cysts in various fluids and at different temperatures. *Journal of Infectious Diseases*, **72**, 232–241.

———(1945). Sedimentation in water and the specific gravity of cysts of *Entamoeba histolytica*. *American Journal of Hygiene*, **41**, 156–163.

———(1950). Kinetics in the thermodestruction of cysts of *Endamoeba histolytica* in water. *American Journal of Hygiene*, **52**, 82–90.

———(1955). Survival of cysts of *Endamoeba histolytica* in human feces under low-temperature conditions. *American Journal of Hygiene*, **61**, 103–120.

Chang, S. L. and Fair, G. M. (1941). Viability and destruction of the cysts of *Endamoeba histolytica*. *Journal of the American Water Works Association*, **33**, 1705–1715.

Cram, E. B. (1943). The effect of various treatment processes on the survival of helminth ova and protozoan cysts in sewage. *Sewage Works Journal*, **15**, 1119–1138.

Dobell, C. (1928). Further observations and experiments on the cultivation of *Entamoeba histolytica* from cysts. *Parasitology*, **19**, 288–313.

Elsdon-Dew, R. (1968). The epidemiology of amoebiasis. *Advances in Parasitology*, **6**, 1–62.

———(1978). *Entamoeba histolytica* and *Homo sapiens*. *Drugs*, **15**(Supplement 1), 1–9.

Engbaek, K. and Larsen, S. O. (1979). A comparative socio-epidemiological study of families with *Giardia lamblia*, *Entamoeba coli* and *Trichuris trichiura*. *Ugeskrift for Laeger*, **141**, 1128–1131.

Eyles, D. E., Jones, F. E. and Smith, C. S. (1953). A study of *Endamoeba histolytica* and other intestinal parasites in a rural West Tennessee Community. *American Journal of Tropical Medicine and Hygiene*, **2**, 173–190.

Frye, W. W. and Meleney, H. E. (1936). The viability of *Endamoeba histolytica* cysts after passage through the cockroach. *Journal of Parasitology*, **22**, 221–222.

Gordon, E. I. (1941). Purification of sewage from cysts of intestinal protozoa. *[illegible] Parazitologiya i Parasitarnye Bolezni*, **10**, 236–243.

Gupta, S. R., Rao, C. K., Biswas, H., Krishnaswami, A., K., Wattal, B. L. and Raghavan, N. G. S. (1972). Role of the housefly in the transmission of intestinal parasitic cysts/ova. *Indian Journal of Medical Research*, **60**, 1120–1125.

Jeffery, G. M. (1960). A three-year epidemiologic study of intestinal parasites in a selected group of mental patients. *American Journal of Hygiene*, **71**, 1–8.

Jones, M. F. and Newton, W. L. (1950). The survival of cysts of *Endamoeba histolytica* in water at temperatures between 45°C and 55°C. *American Journal of Tropical Medicine*, **30**, 53–58.

Jones, T. C. (1979). *Entamoeba histolytica* (amebiasis). In *Principles and Practice of Infectious Diseases*, eds. Mandell, G. L., Douglas, R. G. Jr and Bennett, J. E., pp. 2087–2094. New York: John Wiley.

Kawata, K., Cramer, W. N. and Burge, W. D. (1977). Composting destroys pathogens. *Water and Sewage Works*, **124**, 76–79.

Kheissin, E. M. and Dmitrieva, E. K. (1935). The influence of salts on the cysts of *Entamoeba histolytica*. *Trudy Leningradskogo Instituta Epidemiologii i Bakteriologii imeni Pastera*, **2**, 22–31.

Knight, R. (1975). Surveys for amoebiasis: interpretation of data and their implications. *Annals of Tropical Medicine and Parasitology*, **69**, 35–48.

Kott, H. and Kott, Y. (1967). Detection and viability of *Endamoeba histolytica* cysts in sewage effluents. *Water and Sewage Works*, **140**, 177–180.

LeMaistre, C. A., Sappenfield, R., Culbertson, C., Carter, F. R. N., Offutt, A., Black, H. and Brooke, M. M. (1956).

Studies of a water-borne outbreak of amebiasis, South Bend, Indiana. I. Epidemiological aspects. *American Journal of Hygiene*, **64**, 30–45.

Mackie, T. T., Mackie, J. W., Vaughn, C. M., Gleason, N. N., Greenberg, B. G., Nenninger, E. S., Lunde, M. N., Moore, L. L. A., Kluttz, J. and Taliafero, M. O. (1956). Intestinal parasitic infections in Forsyth County, North Carolina. IV. Domestic environmental sanitation and the prevalence of *Entamoeba histolytica*. *American Journal of Tropical Medicine and Hygiene*, **5**, 29–39.

Mata, L. J., Kromal, R. A., Urrutia, J. J. and Garcia, B. (1977). Effect of infection on food intake and the nutritional state: perspectives as viewed from the village. *American Journal of Clinical Nutrition*, **30**, 1215–1227.

Mathur, T. N. and Kaur, J. (1972*a*). Epidemiology of amoebiasis with particular reference to amoebiasis as a familial infection. *Journal of the Association of Physicians of India*, **20**, 693–696.

———(1972*b*). The epidemiology of amoebiasis in an urban area. *Indian Journal of Medical Research*, **60**, 1134–1137.

Melvin, D. M. and Brooke, M. M. (1962). Parasitologic surveys on Indian reservations in Montana, South Dakota, New Mexico, Arizona, and Wisconsin. *American Journal of Tropical Medicine and Hygiene*, **11**, 765–772.

Moore, H. A., de la Cruz, E. and Vargas-Mendez, O. (1965). Diarrheal disease studies in Costa Rica. IV. The influence of sanitation upon the prevalence of intestinal infection and diarrheal disease. *American Journal of Epidemiology*, **82**, 162–184.

Morton, T. C., Stamm, W. P. and Seidelin, R. (1952). Indigenous amoebiasis: a recent outbreak in England. *British Medical Journal*, **2**, 114–116.

Nnochiri, E. (1965). Observations on childhood amoebiasis in urban family units in Nigeria. *Journal of Tropical Medicine and Hygiene*, **68**, 231–236.

Panicker, P. V. R. C. and Krishnamoorthi, K. P. (1978). Elimination of enteric parasites during sewage treatment processes. *Indian Association for Water Pollution Control Technical Annual*, **5**, 130–138.

Pierce, V., Ascoli, W., de Leon, R. and Gordon, J. E. (1962). Studies of diarrheal disease in Central America. III. Specific etiology in endemic diarrhea and dysentery in Guatemalan children. *American Journal of Tropical Medicine and Hygiene*, **11**, 395–400.

Pipkin, A. C. (1949). Experimental studies on the role of filth flies in the transmission of *Endamoeba histolytica*. *American Journal of Hygiene*, **49**, 255–275.

Reardon, L. V., Verder, E. and Rees, C. W. (1952). The cultural requirements of *Endamoeba coli* and the comparative effects of drying on the cysts of *E. coli* and *E. histolytica*. *American Journal of Tropical Medicine and Hygiene*, **1**, 155–161.

Rendtorff, R. C. (1954). The experimental transmission of human intestinal protozoan parasites. I. *Endamoeba coli* cysts given in capsules. *American Journal of Hygiene*, **59**, 196–208.

Rendtorff, R. C. and Holt, C. J. (1954*a*). The experimental transmission of human intestinal protozoan parasites. III. Attempts to transmit *Endamoeba coli* and *Giardia lamblia* cysts by flies. *American Journal of Hygiene*, **60**, 320–326.

———(1954*b*). The experimental transmission of human intestinal protozoan parasites. IV. Attempts to transmit *Endamoeba coli* and *Giardia lamblia* cysts by water. *American Journal of Hygiene*, **60**, 327–338.

Root, F. M. (1921). Experiments on the carriage of intestinal protozoa of man by flies. *American Journal of Hygiene*, **1**, 131–153.

Rudolfs, W., Falk, L. L. and Ragotzkie, R. A. (1950). Literature review on the occurrence and survival of enteric, pathogenic and related organisms in soil, water, sewage and sludges, and on vegetation. II. Animal parasites. *Sewage and Industrial Wastes*, **22**, 1417–1427.

———(1951). Contamination of vegetables grown in polluted soil. II. Field and laboratory studies on *Entamoeba* cysts. *Sewage and Industrial Wastes*, **23**, 478–485.

Sargeaunt, P. G., Williams, J. E., Kumate, J. and Jimenez, E. (1980). The epidemiology of *Entamoeba histolytica* in Mexico City. A pilot survey. I. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **74**, 653–656.

Schmerin, M. J., Gelston, A. and Jones, T. C. (1977). Amebiasis: an increasing problem among homosexuals in New York City. *Journal of the American Medical Association*, **238**, 1386–1387.

Schoenleber, A. W. (1940). The food handler as a transmitter of amebiasis. *American Journal of Tropical Medicine*, **20**, 99–106.

Scott, J. C. (1952). *Health and Agriculture in China: a Fundamental Approach to some of the Problems of World Hunger*. London: Faber and Faber.

Sexton, D. J., Krogstad, D. J., Spencer, H. C. Jr. Healy, G. R., Sinclair, S., Sledge, C. E. and Schultz, M. G. (1974). Amebiasis in a mental institution: serologic and epidemiologic studies. *American Journal of Epidemiology*, **100**, 414–423.

Simitch, T., Petrovitch, Z. and Chibalitch, D. (1954). Viability of cysts of *Entamoeba histolytica* outside the host. *Archives de l'Institut Pasteur d'Algérie*, **32**, 223–231.

Sole, T. D. and Croll, N. A. (1980). Intestinal parasites in man in Labrador, Canada. *American Journal of Tropical Medicine and Hygiene*. **29**, 364–368.

Spector, B. K., Baylis, J. R. and Gullans, O. (1934). Effectiveness of filtration in removing from water, and of chlorine in killing, the causative organism of amoebic dysentery. *Public Health Reports*, **49**, 786–800.

Spector, B. K. and Buky, F. (1934). Viability of *Endamoeba histolytica* and *Endamoeba coli:* effect of drying. *Public Health Reports*, **49**, 379–385.

Spencer, H. C. Jr, Hermos, J. A., Healy, G. R., Melvin, D. M. and Shmunes, E. (1976). Endemic amebiasis in an Arkansas community. *American Journal of Epidemiology*, **104**, 93–99.

Spencer, H. C., Muchnik, C., Sexton, D. J., Dodson, P. and Walls, K. W. (1977). Endemic amebiasis in an extended family. *American Journal of Tropical Medicine and Hygiene*, **26**, 628–635.

van Zijl, W. J. (1966). Studies on diarrhoeal diseases in seven

countries by the WHO Diarrhoeal Diseases Advisory Team. *Bulletin of the World Health Organization*, **35,** 249–261.

Wachs, A. (1961). *Studies on Sewage Stabilization Ponds in Israel: I. Oxidation Ponds at Herzhya.* Haifa: Technion Sanitary Engineering Laboratories.

Wang, W.-L. L. and Dunlop, S. G. (1954). Animal parasites in sewage and irrigation water. *Sewage and Industrial Wastes*, **26,** 1020–1032.

WHO Scientific Working Group (1980). Parasite-related diarrhoeas. *Bulletin of the World Health Organization*, **58,** 819–830.

Wykoff. D. E., Fonseca, J. R. C. and Richie, L. S. (1955). Epidemiology of amebiasis: possible influence of water supply, coincident with diverse features of terrain, on the occurrence of intestinal protozoa. *American Journal of Tropical Medicine and Hygiene*, **4,** 465–471.

Wykoff, D. E. and Ritchie, L. S. (1960). Intenstinal protozoa in family groups of four Japanese villages. *US Armed Forces Medical Journal*, **11,** 1106–1116.