16

Shigella and Shigellosis

DYSENTERY, the frequent passing of bloody stools, has been recognized and feared throughout history as a serious and sometimes fatal condition. One major cause of dysentery is infection by members of the bacterial genus *Shigella* (the other major cause is infection by the protozoon *Entamoeba histolytica*—described in chapter 20).

Description of Pathogen and Disease

Shigellae, and the infections they cause in man, are well documented in many countries. Together with *Salmonella* species (chapter 15) and *Vibrio cholerae* (chapter 17), they are the classic bacterial agents of intestinal infection.

Identification

Shigellosis (bacillary dysentery) is an acute diarrheal disease caused by bacteria of the genus *Shigella*. The disease, which primarily involves the large intestine, may be asymptomatic or may have symptoms ranging from mild diarrhea to a severe disease accompanied by fever, vomiting, cramps, and tenesmus, with blood, mucus, and pus in the stools. The typical case is of short duration (about 4 days), but in exceptional cases the symptoms may last for up to 2 weeks. The severity of the illness and the mortality rate depend on the nutritional state and age of the patient, on the serotype of the organism, and on the infecting dose.

The severe form of the disease, bacillary dysentery, is often due to *Shigella dysenteriae*. Disease due to type I (Shiga's bacillus) is particularly serious. Mortality of untreated cases of bacillary dysentery may be as high as 25 percent but is usually much lower. Diagnosis is by isolation of the bacteria from feces or rectal swabs. The presence of many pus cells in the stool is highly suggestive of this diagnosis.

Occurrence

Shigellosis has a worldwide distribution, with the highest incidence in communities where hygiene is poor. Children aged 1–4 years are the most affected, and 60 percent of cases and most fatalities are children under 10 years of age.

The different species of *Shigella* vary in their relative importance in different parts of the world. In Asia, South America, and Africa, *Shigella dysenteriae* is often responsible for severe disease, but all species are common. Shigellosis in the developed countries is most commonly caused by *Shigella sonnei*. In England and Wales 43,285 cases were reported in 1960 and 10,765 cases in 1970. At present some 98 percent of the infections in England and Wales are caused by *Sh. sonnei*, and the majority of known cases of infection with *Sh. flexneri*, *Sh. boydii*, or *Sh. dysenteriae* are persons recently returned from developing countries.

All types of shigellae are found in areas of inadequate sanitation and poverty; *Sh. dysenteriae*, *Sh. flexneri*, and *Sh. boydii* are the most frequently identified, and *Sh. sonnei* infections are relatively rare. As hygiene improves, *Sh. sonnei* becomes the dominant species, *Sh. flexneri* the next most common, and *Sh. boydii* and *Sh. dysenteriae* become rare. When conditions of hygiene degenerate, such as with an army in the field, infections with species other than *Sh. sonnei* again become common.

Infectious agent

Shigellae are Gram-negative, nonmotile rods belonging to the family Enterobacteriaceae and closely resembling *Escherichia coli* and *Salmonella* (figures 13-1a and 15-1). Four major serological groups have been described, with some forty serotypes making up these

groups. The group and type antigens are all cell-wall antigens, but group antigens are difficult to demonstrate.

- group A, *Sh. dysenteriae,* includes at least ten serotypes, with three others provisionally recognized. Phage-typing systems are also used.
- group B, *Sh. flexneri,* includes nine serotypes that are related not only by a common group antigen but also by various shared type antigens. Phage-typing systems are also used.
- group C, *Sh. boydii,* includes fifteen serotypes and two additional, provisional serotypes. Phage-typing systems are also used.
- group D, *Sh. sonnei,* includes only one serotype, and colicin-typing and phage-typing are used to subdivide the group.

Reservoir

Shigellae have no natural hosts other than the higher primates. Although experimental infections can be produced in other primates, man is the only effective source of infection. Unlike *Salmonella* and *Escherichia* infections, animal feces are not a source of inocula, though animals may become contaminated by the ingestion of human feces.

Transmission

Shigellae are transmitted from man to man, from ill persons, healthy convalescents, or symptomless carriers to susceptible persons. The organisms, which are excreted in the feces, are usually transmitted by the direct fecal-oral route. Infected persons with diarrhea typically excrete 10^5–10^9 shigellae per gram of wet feces, while symptomless carriers may excrete 10^2–10^6 per gram (Dale and Mata 1968; Thomson 1955). Food may be contaminated through the contaminated fingers of patients or carriers. Foodborne and waterborne outbreaks occur. However, contamination of the environment (such as seats, door handles, and water-flushing devices in toilets) by infected feces and their transfer to the mouth seems to be the usual mode of infection. Transfer of shigellae by flies breeding on feces has been of crucial importance in some outbreaks.

The infective dose for *Shigella* is reported to be lower than for the other main diarrhea-causing bacterial pathogens (*Salmonella, Vibrio cholerae,* and *E. coli*). The median infective dose (ID_{50}) for *Shigella* may be around 10^4 in healthy adults; for the other bacterial pathogens listed above, it is 10^7 or higher. Dupont and others (1972) found that a dose of 10^4 *Sh. flexneri* produced disease (oral temperature $\geqslant 37.8°C$, with four or more watery stools per 24 hours) in 59 percent of eighty-eight adult male volunteers, whereas a dose of 180 produced disease in 22 percent of thirty-six volunteers. Levine and others (1973) induced disease (oral temperature $\geqslant 37.8°C$, with three or more watery stools per 24 hours) in one out of ten adult male volunteers with only 10 virulent *Sh. dysenteriae* type 1. By contrast, Shaughnessy and others (1946) required massive doses of *Sh. flexneri* (10^8 organisms) to induce disease in volunteers who had previously ingested 2 grams of sodium bicarbonate to lower their gastric acidity.

Incubation period

The incubation period ranges from 36–72 hours; frank dysentery usually appears within 2 days.

Period of communicability

Patients recovering from an acute attack of shigellosis may continue to excrete bacilli in their stools. In general, this excretion lasts for only a week or so, but a small proportion become persistent carriers. DuPont and others (1970) found that 75 percent of 542 children who contracted shigellosis at an institution for the mentally retarded in New York (USA) excreted *Shigella* for less than 1 month, but that 7 percent continued to excrete the organism for over a year. There is also evidence of the presence of shigellae in completely symptomless carriers, and carriage for periods of years may be common under conditions of poor hygiene. In this context the carriage of shigellae for long periods by breast-fed infants should be noted. In such infants overt disease is seldom seen until breast feeding ceases.

Resistance

Children are especially susceptible, and natural resistance to *Shigella* infection has not been reported. Individuals in closed communities may have numerous repeated infections with a single serological type of *Shigella.* Such immunity as does occur is probably group specific and may involve the local production of short-lived antibodies in the colonic mucosa.

Epidemiology

Shigellosis is endemic and common in almost all communities where living standards are low and water and excreta disposal facilities inadequate. It may also be endemic in some institutions, such as schools and

geriatric wards, where poor hygiene occurs. Children, especially weanlings, suffer the highest incidence of infection and mortality, and malnourished children are especially vulnerable.

Shigella infections among poor people in developing countries are often very common. Over a 1-year period in an Egyptian village, for instance, 51 percent of eighty-two children between 6 months and 5 years old had one episode, 17 percent had two episodes, and 5 percent had three or more episodes (Higgins and Floyd 1955). Several studies have shown that up to about 18 percent of young children in poor communities may be excreting *Shigella* at any time. Beck, Munoz and Scrimshaw (1957) reported that the point prevalence of *Shigella* excretion among children under 10 years old in twelve communities in Guatemala varied between 3.7 and 16.2 percent. Richardson and others (1968) recorded that 13 percent of African school children (7–16 years old) in the Western Transvaal (South Africa) were excreting *Shigella* (mainly *Sh. flexneri*) in summer. In the USA, *Shigella* excretion prevalences of up to 10 percent among children have been recorded in some samples of farm labor families in California and mining families in Kentucky (Hollister and others 1955; Schliessmann and others 1958; Watt and others 1953). It has been estimated that the prevalence of *Shigella* excretion among the whole population is 0.46 percent in the USA, 0.33 percent in England and Wales, and 2.4 percent in rural Sri Lanka (Geldreich 1972).

Khan and Mosley (1968) studied shigellosis in Rayer Bazar, a village on the outskirts of Dacca (Bangladesh). Shigellosis occurred throughout the year with a peak during the monsoon (June–October). Seventy-one percent of shigellosis cases identified were children under 5 years old. The proportion of all recorded diarrheas that were associated with *Shigella* was 4.4 percent. Khan, Curlin and Huq (1979) studied the families of forty-seven index cases with diarrhea due to *Sh. dysenteriae* type 1 in Dacca. Twenty percent (49 of 240) of family contacts of index cases were infected with *Shigella* at some time during the 10 days following the reporting of the index case. The proportion of infected family contacts was highest (31 percent) among children 0–4 years old. Of the forty-nine infected family contacts, five (10 percent) required hospitalization and twenty-seven (55 percent) developed mild to moderate diarrhea. The equivalent proportions among the 0–4 age group were 27 percent and 73 percent—in other words, there were no asymptomatic infections in this age group. Poor families living in one-room houses with "open latrines" and unprotected water sources had higher secondary infection rates than other families. In another study in Dacca, Khan and Shahidullah (1980) found similar infection and diarrhea rates among family contacts of index cases having *Sh. dysenteriae* type 1 infection. Among family contacts of index cases having *Sh. flexneri* infection, 20 percent were infected, but the proportion of those infected who experienced diarrhea was only 20 percent.

Reller, Gangarosa and Brachman (1969) reviewed shigellosis in the USA over the period 1964–68. Approximately 10,000 cases per year were reported (very many will have gone unreported), and the highest attack rate occurred in the 0–4 age group. Peak incidence was in the late summer of each year. Residents of mental institutions, Indian reservations, and urban slums were identified as being especially at risk from shigellosis. Fifty-four percent of isolations were *Sh. sonnei* and 40 percent were *Sh. flexneri*. Only 6.6 percent of reported cases of shigellosis were associated with either foodborne or waterborne outbreaks (Black, Craun and Blake 1978).

The transmission of *Sh. sonnei* among school children in England was studied by Hutchinson (1956), who isolated the organism from toilet seats, toilet floors, chamber pots, clothes, bedding, toys, and floors. Eleven out of thirty-four toilet seats were found to be contaminated in a school during an epidemic. When heavily infected, loose, bulky stools were flushed away it was found that contamination of the seat could occur, but this did not appear to happen with solid stools. As many as 50 percent of children were found to be hand carriers after visiting a toilet at the peak of an outbreak. Observations at a nursery school showed that, of thirty-seven children, half handled the seat when settling themselves on it, and one-third of these then either handled their face or mouth or sucked their fingers. On the skin of the fingers *Sh. sonnei* remained alive for over 3 hours. Tests with five types of toilet paper showed that, when double thicknesses of paper were used, organisms from fluid or semisolid feces passed through the paper to the fingers each time. With solid feces the organisms passed through four out of five of the papers.

There is general agreement in the literature that the maintenance of endemic shigellosis has little or no relationship to water quality, but that it is strongly related to water availability and associated hygienic behavior. However, there will always be specific exceptions to this; for instance, Sultanov and Solodovnikov (1977) considered that the maintenance of dysentery in Dagestan (USSR) during 1959–73 was due to the widespread use of polluted surface water for domestic purposes.

Shigellosis occurs in epidemics in addition to being endemic. In 1969 and 1970 an epidemic due to Shiga's

bacillus (*Sh. dysenteriae* type 1) occurred in Central America and Mexico in which there were an estimated 112,000 cases and 8,300 deaths in Guatemala alone in the first 10 months of 1969 (Gangarosa and others 1970). Major outbreaks of Shiga dysentery also occurred in Bangladesh in 1973 (Rahaman and others 1975) and in Somalia in 1963–64 (Cahill, Davies and Johnson 1966). *Shigella* strains with plasmid-mediated multiple drug resistance were involved in Central America and Bangladesh.

Some epidemics are waterborne. An outbreak of some 2,000 cases of shigellosis due to *Sh. sonnei* occurred in 1966 in Montrose (Scotland) when the chlorination plant on the town's water supply broke down (Green and others 1968). During 1961–75, thirty-eight waterborne outbreaks (comprising 5,893 cases) of shigellosis were reported in the USA (Black, Craun and Blake 1978). Most of these outbreaks involved semipublic or individual water systems and were usually the result of inadequate or interrupted chlorination of water contaminated by feces. An outbreak due to *Sh. sonnei* and linked with bathing in a polluted section of the Mississippi River in Iowa (USA) has been reported (Rosenberg and others 1976).

Some epidemics are foodborne. An outbreak of at least 600 cases occurred on the island of Maui (Hawaii, USA) in 1970. *Shigella sonnei* was the causative bacterium, and the outbreak was spread by the contamination of poi (ground taro root) produced at a single factory and distributed around the island (Lewis and others 1972). It was shown that *Sh. sonnei* in poi could undergo a tenfold increase in concentration in 1 day at room temperature. An outbreak of at least 140 cases (culture-positive for *Sh. sonnei*) occurred in 1979 among hospital staff in the USA and was linked to the contamination of tuna salad by an infected cafeteria employee (Bowen 1980). During 1961–75, seventy-two foodborne outbreaks (comprising 10,648 cases) of shigellosis were reported in the USA (Black, Craun and Blake 1978). Most of these outbreaks were caused by contaminated salads associated with poor hygiene among food handlers.

Control Measures

Shigella bacteria are transmitted from person to person, especially where hygiene is poor and the domestic environment is fecally contaminated. Effective control depends upon personal hygiene and the sanitary disposal of excreta.

Individual

Antimicrobial prophylaxis has little to recommend it, although individual agents have been used for the control of institutional outbreaks. Sulphonamide and tetracycline have been used widely in some communities, but their use has a dubious effect. It must be remembered that resistance transfer factors were first described in *Shigella* and that the worldwide resistance of these bacteria to sulphonamides (and often also to other antibiotics including tetracycline, streptomycin, chloramphenicol, and ampicillin) is the result of the spread of such factors.

Killed vaccines are ineffective. Live oral vaccines are still at an experimental stage. They are type specific and protect for short periods against the disease. They are difficult to prepare and administer and are therefore of limited use.

Scrupulous personal hygiene is the most effective means of individual protection. Breast feeding considerably reduces the risk of disease in infants.

Environmental

There is probably more information available on the effect of environmental improvements in reducing shigellosis than on any other infection described in this book. Studies have been conducted into the spread and control of shigellosis in institutions in developed countries (such as the work of Hutchinson, summarized above), and other investigations have examined the role of environmental modifications in controlling shigellosis in poor communities (see table 2-1). These later studies have either used diarrhea rates or the prevalence of *Shigella* isolations from rectal swabs from a sample of the community, often children.

In towns in Georgia (USA) Stewart and others (1955) found that *Shigella* infection was related to poor water supply, poor excreta disposal facilities, high fly counts, and to poor housing in general. More specifically it was found that, among otherwise similar households, those with water close to the house had a lower incidence than those who fetched water from further away, but that the type of water source (well or tap) did not affect shigellosis incidence. A subsequent study in Georgia (McCabe and Haines 1957) recorded that a latrine program (bored hole latrines, 2.5 meters deep) in the town of Boston was associated with a reduction in the detection of *Shigella* from 4.7 percent to 2.8 percent of rectal swabs. Rates in the control towns did not fall over the period. After completion of the latrine program, the rate of reported diarrhea was half that in the control towns. Although the housefly

population was not reduced, the breeding of flies in excreta was much reduced.

Schliessmann and others (1958) investigated environmental influences on shigellosis in 11 mining camps in the eastern coalfields of Kentucky (USA) during 1954 to 1957. Reported diarrheal disease rates ranged from 9.4 to 53.6 per 100 persons per year in the different study areas. More than half the total cases were children between 0 and 4 years old, and more than one-quarter were under 2 years old. The highest incidence occurred in August and September. *Shigella* isolation rates, obtained by rectal swabbing of preschool children, ranged between 0.7 percent and 10 percent in individual study areas. Seventy-six percent of *Shigella* isolates were *Sh. flexneri*. Shigellosis was the major cause of acute diarrhea in the areas with poor sanitation but was not a primary cause in the areas with the best sanitation. Housefly populations were generally low and were not associated with *Shigella* prevalence. Water quality was not related to the incidence of diarrhea or to the prevalence of *Shigella* excretion. Those having flush toilets and inside water had an incidence of diarrhea of 14 per 100 persons per year and a *Shigella* excretion prevalence of 1.1 percent; those having inside water but an outside latrine had figures of 24 per 100 per year and 2.4 percent; and those having outside water and an outside latrine had an incidence of 36 per 100 per year and a *Shigella* prevalence of 5.9 percent. Where water was not piped inside the house, persons having access to water in their yard had a diarrhea incidence one-third less than individuals obtaining water away from their premises (see also Schliessmann 1959).

In the San Joaquin Valley of California (USA), Watt and others (1953) reported that the *Shigella* excretion prevalences among children under 10 years old were higher in migrant worker camps (6.1 percent) than in poor but permanent housing on urban fringe areas (3.9 percent). A follow-up study on the camps (Hollister and others 1955) recorded the following *Shigella* excretion prevalences among children under 10 years old: in cabins with inside water, shower, and toilet, 1.6 percent; in cabins with inside water but with communal shower and toilet, 3.0 percent; in cabins with no internal facilities, 5.8 percent. Studies in the Lower Rio Grande Valley in Texas (USA) showed that fly control with DDT reduced the rates of both diarrheal disease and *Shigella* isolation (Watt and Lindsay 1948).

Gordon, Behar and Scrimshaw (1964) reported a comparison of acute diarrheal disease rates between families having a latrine and those having no latrine in rural Guatemala. The authors concluded that "the data give no indication that privies as used in the villages had any influence on the diarrheas of children in the first two years of life, the important part of the problem." A summary of surveys of diarrhea among preschool children in Bangladesh, Egypt, Iran, Mauritius, Sri Lanka, Sudan, and Venezuela (van Zijl 1966) concluded that water supplies and excreta disposal facilities were important determinants of shigellosis (see also Wolff, van Zijl and Roy 1969).

Rajasekaran, Dutt and Pisharoti (1977) studied 1,041 children under 5 years old for 1 year in five villages in Tamil Nadu (India). Thirty-two percent of all diarrheal episodes were associated with *Shigella* excretion. Those who used an open well (98 percent of water samples contained >10 coliforms per 100 milliliters) had a significantly *lower* incidence of diarrhea and shigellosis than those who used a street tap (25 percent of water samples contained >10 coliforms per 100 milliliters). Those who used tap water in the house had a lower incidence than both well users and street tap users. Preliminary findings from Teknaf (Bangladesh) suggested that diarrhea and shigellosis incidences were inversely related to the daily per capita usage of tubewell water (Rahaman 1979).

These and other studies (see table 2-1) indicate that a plentiful water supply located close to or in the home (to allow good personal cleanliness) and an adequate latrine that is properly used are key elements in the control of shigellosis. Good personal and domestic hygiene and restricting the access of flies to human excreta are also important. Water quality is not of particular importance in communities where shigellosis is highly endemic.

It must be emphasized that those who most commonly experience shigellosis, those who most commonly excrete *Shigella*, and those for whom the consequences of infection are potentially the most serious are small children. Small children are not only the major sufferers but also the major source of the bacteria, which will contaminate the domestic environment and subsequently infect other children and adults. The personal hygiene of small children, and mother-child behavior patterns, are therefore of great importance in controlling shigellosis.

Occurrence and Survival in the Environment

Although the shigellae are among the most important pathogenic excreted bacteria, their presence and persistence in the environment have been studied far less than is the case for *E. coli* and the salmonellae.

In water

Shigellae will be found in low concentrations in most surface waters contaminated by human feces. They will therefore be present in many contaminated drinking water sources in developing countries, although their presence is almost never sought in routine water testing. Unlike the salmonellae, *E. coli*, and fecal streptococci, the shigellae are excreted only by man, and because much contamination of village water supplies derives from animals, the concentrations of shigellae will in general be much lower than the concentrations of the fecal indicator bacteria or the salmonellae.

Tap water will only contain shigellae if it is untreated and drawn from a contaminated source, or if the treatment plant has broken down—as in the Montrose (Scotland) outbreak (Green and others 1968).

Some studies on the survival of shigellae in water are listed in the appendixes of Feachem and others (1980). Survival depends upon factors such as the concentrations of other bacteria, nutrients, and oxygen and on the temperature. In clean waters, survival times are typically less than 14 days at warm temperatures (>20°C), whereas the bacteria may survive for a few weeks below 10°C. At warm temperatures, 99 percent reduction in *Shigella* numbers is likely to occur in less than 5 days. McFeters and others (1974) found that shigellae died more slowly in well water at 9–12°C than the fecal indicators, salmonellae, or *Vibrio cholerae* (the half-life of the shigellae was about 24 hours).

Survival is most prolonged in very clean waters (such as unchlorinated tap water) or in polluted water containing nutrients but having a minimum of other bacteria present. In these latter conditions, *Shigella* may grow. Talayeva (1960) recorded survival of *Shigella flexneri* at 19–24°C for up to 21 days in clean river water, up to 47 days in autoclaved river water, up to 9 days in well water, up to 44 days in autoclaved tap water, and for up to 6 days in polluted well water. McGarry and Stainforth (1978) reported experiments in China that showed the survival of *Sh. dysenteriae* for up to 93 days in sterilized water at 11–28°C. Shrewsbury and Barson (1957) made up sterile synthetic well water of the same general composition as that obtained from Hagar's Well in Mecca (Saudi Arabia) at the time of the 1883 cholera outbreak. Shigellae could survive for between 2.5 and 29 months in this sterile but fecally contaminated water at 21°C. Hendricks (1972) reported that *Sh. flexneri* multiplied in sterilized river water collected downstream from a sewage outfall. Growth occurred at 30°C but not at 20°C or 5°C, and no growth at any temperature was recorded in water collected upstream from the sewage outfall (see also Hendricks 1971).

Limited tests on *Shigella* in seawater (Nakamura and others 1964) suggest that survival times (15 to more than 70 days at 15°C) may be somewhat longer than those in freshwater—in contrast to the fecal indicator bacteria (chapter 13), which die more rapidly in seawater than in freshwater. Tests in sterile saline waters (salinities = 0.5, 2.5, and 3.5 percent) at various temperatures (4, 25, 37°C), however, showed that *Sh. dysenteria* survived for less than 6 days, even at 4°C (Jamieson, Madri and Claus 1976).

In feces and sewage

Between perhaps 0.2 percent and 4 percent of a community will be excreting *Shigella* depending on the levels of hygiene that prevail. Sewage may therefore contain between about 10 and 10^4 *Shigella* per liter.

Few data are available, but it is likely that survival in feces and sewage is curtailed by the activity of the large populations of other bacteria present. Survival is enhanced at low temperatures, by sterilizing the feces or sewage prior to introducing the shigellae, or by raising the pH. Hutchinson (1956) studied the survival time of *Sh. sonnei* in naturally infected feces. At room temperature, survival times varied between 2 and 26 days depending on the initial concentration of shigellae, which varied from 7×10^3 to 3.2×10^7 per gram. With an initial concentration of 1.5×10^6 per gram, none could be detected after 7 days storage at 37°C, whereas an 82 percent reduction occurred at 20°C. Kligler (1921) reported that *Sh. dysenteriae* survived for less than 6 days in septic tank effluent. Experiments in China (McGarry and Stainforth 1978) showed that *Sh. dysenteriae* survived for up to 17 days in biogas plant effluent (11–28°C) but for less than 30 hours in the biogas plant itself (14–24°C).

On surfaces

The transmission of shigellosis depends substantially on the contamination of clothes, hands, and household surfaces; the bacteria are transferred from these surfaces to the mouth. Hutchinson (1956) recorded that *Sh. sonnei* could survive for over 3 hours on the fingers and for up to 17 days on a wooden toilet seat. Survival was prolonged by low temperature, high humidity, and poor lighting. Spicer (1959) found that *Sh. sonnei* survived for 7–10 days on cotton threads at cool temperatures and high or low humidities. Nakamura (1962) studied *Sh. sonnei* survival at various temperatures on metal, wood, cotton, paper, and glass in a laboratory with relative humidities between 17 and

33 percent. Survival times were 10–57 days at −20°C, 4–40 days at 4°C, 2–28 days at 15°C, 0–13 days at 37°C and 0–2 days at 45°C. At 15°C, survival was longer on cotton, wood, and paper than on glass and metal. It is noteworthy that toilets and latrines are often relatively cool, humid, and poorly lit—conditions ideal for the optimal survival of shigellae on interior surfaces.

In food

The contamination of food with shigellae is probably an important route of transmission in many communities (see, for instance, Barrell and Rowland 1979). Taylor and Nakamura (1964) reported that *Sh. sonnei* and *Sh. flexneri* survived for considerable periods (80 days or more) in foods such as flour, eggs, oysters, clams, and milk. At warm temperatures (25°C) some growth was noted. Acidic foods, such as orange juice, were more hostile environments for shigellae; even so, the organisms remained detectable in these foods for up to 10 days.

On crops

The few studies on *Shigella* survival on crops irrigated with night soil or sewage are listed in the appendixes of Feachem and others (1980). Shigellae on crop surfaces will typically be exposed to warm temperatures, bright sunlight, and rapid drying. These factors are all hostile to shigellae, and reported survival times are nearly always less than 7 days (Geldreich and Bordner 1971; Rudolfs, Falk and Ragotzkie 1951). It is probable that, in arid hot climates, only a very small fraction of shigellae on crops would survive beyond 2 days. Babov, Nadvornyi and Keimakh (1967) reported that a variety of vegetables grown on sewage farms at Odessa (USSR) were contaminated by *Sh. flexneri*. Contamination was eliminated when irrigation was stopped 2 weeks before harvest, but harvested vegetables could be recontaminated by being laid on the soil.

In the air

As with other enteric bacteria and viruses (see chapters 9 and 13), shigellae may be spread in aerosol droplets produced by flush toilets, activated sludge plants, and spray irrigation systems. Hutchinson (1956) found that toilet seats were contaminated by droplets containing shigellae when a loose stool was flushed away but not when a solid stool was flushed. Newson (1972) found that flushing a fluid suspension of 10^{10} *Sh. sonnei* produced an aerosol of about thirty-nine bacteria per cubic meter of air and that shigellae dispersed in splashes from the toilet could survive for up to 4 days.

Hickey and Reist (1975) failed to isolate any airborne shigellae downwind from two activated sludge tanks in the USA, and they attribute this to the very low concentrations of shigellae in the sewage. Katzenelson, Buium and Shuval (1976) found that the incidence of shigellosis on 77 kibbutzim practicing wastewater spray irrigation was 10 cases per 1,000 persons per year, whereas the incidence on 130 kibbutzim practising no form of wastewater irrigation was 4.5 cases per 1,000 per year.

Inactivation by Sewage Treatment Processes

Few studies have been conducted on the inactivation of shigellae by sewage treatment plants—in part because they are difficult organisms to enumerate in sewage and in part because it is quite common to fail to find any in sewage, even where the community is known to be infected (for instance Brezenski, Russomanno and DeFalco 1965; Daniel and Lloyd 1980; Dixon and McCabe 1964; Olivieri, Kawata and Krusé 1978; Wang, Dunlop and De Boer 1956). The data that are available suggest that removal of shigellae is very similar to *E. coli* removal (chapter 13). Conventional treatment plants, without tertiary processes, will remove between 90 and 99 percent (Kabler 1959), whereas waste stabilization ponds can remove a far higher proportion. It is likely that the survival of shigellae in sewage and sewage effluents is considerably shorter than the survival of *E. coli*. Slijkhuis, Betzer and Kott (1976) reported that *Sh. flexneri* were not detectable after 2 days in a waste stabilization pond in Israel.

Inactivation by Night Soil and Sludge Treatment Processes

There are no data available on *Shigella* destruction in most sludge treatment processes. However, the conditions of sludge treatment will be highly antagonistic to shigellae, and high rates of destruction may be expected. It is probable that *Shigella* destruction will proceed considerably more rapidly than that of the fecal indicator bacteria (chapter 13).

Processes about which data are available are composting and heating. Studies on aerobic thermophilic composting of night soil and garbage in Beijing

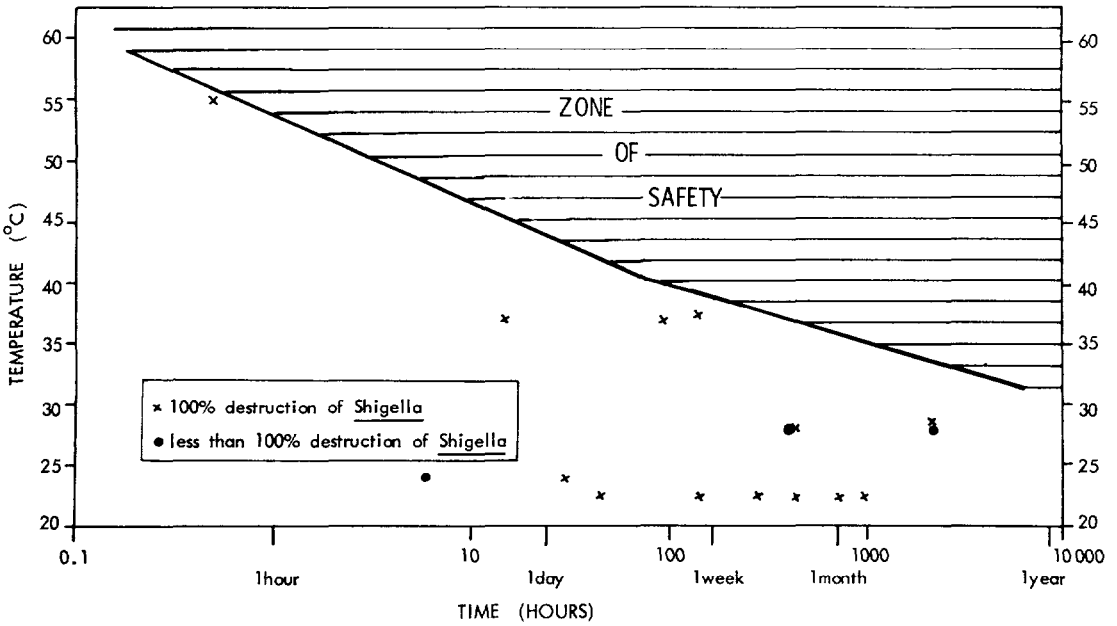

Figure 16-1. *The influence of time and temperature on* Shigella. The points plotted are the results of experiments done under widely differing conditions. The line drawn represents a conservative upper boundary for death

(China) showed that seeded *Sh. dysenteriae* were destroyed within 5 days in piles with a temperature of up to 50°C (Chinese Academy of Medical Sciences 1975). Other studies and reviews (for instance Bhaskaran and others 1957; Petrick 1954; Wiley 1962) confirm the elimination of shigellae from well-managed thermophilic composting systems but warn that shigellae, and other enteric bacteria, may survive on the edge of a pile where temperatures remain low (Reeves 1959). Various data on *Shigella* destruction by time-temperature effects have been compiled in figure 16-1, and it may be seen that 1 hour at 55°C, 1 day at 45°C, and 1 week at 40°C are lethal combinations.

Literature Cited

Babov, D. M., Nadvornyi, N. N. and Keimakh, A. S. (1967). The microflora of vegetables and other crops grown on sewage farms. *Hygiene and Sanitation*, **32,** 273–275.

Barrell, R. A. E. and Rowland, M. G. M. (1979). Infant foods as a potential source of diarrhoeal illness in rural West Africa. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **73,** 85–90.

Beck, M. D., Munoz, J. A. and Scrimshaw, N. S. (1957). Studies on diarrheal diseases in Central America. I. preliminary findings on cultural surveys of normal population groups in Guatemala. *American Journal of Tropical Medicine and Hygiene*, **6,** 62–71.

Bhaskaran, T. R., Ghosh Roy, B. K., Sampathkumaran, M. A., Radhakrishnan, I. and Mukherjee, D. B. (1957). Studies on the survival of pathogens in nightsoil compost. *Indian Journal of Agricultural Science*, **27,** 91–102.

Black, R. E., Craun, G. F. and Blake, P. A. (1978). Epidemiology of common-source outbreaks of shigellosis in the United States, 1961–1975. *American Journal of Epidemiology*, **108,** 47–52.

Bowen, G. S. (1980). An outbreak of shigellosis among staff members of a large urban hospital. In *Epidemic Intelligence Service 29th Annual Conference*, p. 46. Atlanta, Georgia: Centers for Disease Control.

Brezenski, F. T., Russomanno, R. and DeFalco, P. (1965). The occurrence of *Salmonella* and *Shigella* in post-chlorinated and non-chlorinated sewage effluents and receiving waters. *Health Laboratory Science*, **2,** 40–47.

Cahill, K. M., Davies, J. A. and Johnson, R. (1966). Report on an epidemic due to *Shigella dysenteriae* Type 1 in the Somali interior. *American Journal of Tropical Medicine and Hygiene*, **15,** 52–56.

Chinese Academy of Medical Sciences, Department of Environmental Health, Institute of Health, Beijing (1975). Sanitary effects of urban garbage and nightsoil composting. *Chinese Medical Journal*, **1,** 407–412.

Dale, D. C. and Mata, L. J. (1968). Studies of diarrheal disease in Central America. XI. Intestinal bacterial flora in malnourished children with shigellosis. *American Journal of Tropical Medicine and Hygiene*, **17,** 397–403.

Daniel, R. R. and Lloyd, B. J. (1980). Microbiological studies on two Oxfam sanitation units operating in Bengali refugee camps. *Water Research*, **14,** 1567–1571.

Dixon, F. R. and McCabe, L. J. (1964). Health aspects of wastewater treatment. *Journal of the Water Pollution Control Federation*, **36,** 984–989.

DuPont, H. L., Gangarosa, E. J., Reller, L. B., Woodward, W. E., Armstrong, R. W., Hammond, J., Glaser, K. and Morris, G. K. (1970). Shigellosis in custodial institutions. *American Journal of Epidemiology*, **92,** 172–179.

DuPont, H. L., Hornick, R. B., Snyder, M. J., Libonati, J. P., Formal, S. B. and Gangarosa, E. J. (1972). Immunity in shigellosis. II. Protection induced by oral live vaccine or primary infection. *Journal of Infectious Diseases*, **125,** 12–16.

Feachem, R. G. A., Bradley, D. J., Garelick, H. and Mara, D. D. (1980). *Health Aspects of Excreta and Sullage Management: A State-of-the-Art Review.* Appropriate Technology for Water Supply and Sanitation, vol. 3. Washington, DC: The World Bank, Transportation, Water and Telecommunications Department.

Gangarosa, E. J., Perera, D. R., Mata, L. J., Mendizábal-Morris, C., Guzman, G. and Reller, L. B. (1970). Epidemic Shiga bacillus dysentery in Central America. II. Epidemiologic studies in 1969. *Journal of Infectious Diseases*, **122,** 181–190.

Geldreich, E. E. (1972). Water-borne pathogens. In: *Water Pollution Microbiology.* Ed. Mitchell, R. pp. 207–241. New York: Wiley-Interscience.

Geldreich, E. E. and Bordner, R. H. (1971). Fecal contamination of fruits and vegetables during cultivation and processing for market: a review. *Journal of Milk and Food Technology*, **34,** 184–195.

Gordon, J. E., Behar, M. and Scrimshaw, N. S. (1964). Acute diarrhoeal disease in less developed countries. *Bulletin of the World Health Organization*, **31,** 1–28.

Green, D. M., Scott, S. S., Mowat, D. A. E., Shearer, E. J. M. and Macfarlane Thomson, J. (1968). Water-borne outbreak of viral gastroenteritis and Sonne dysentery. *Journal of Hygiene*, **66**, 383–392.

Hendricks, C. W. (1971). Enteric bacterial metabolism of stream sediment eluates. *Canadian Journal of Microbiology*, **17,** 551–556.

—— (1972). Enteric bacterial growth rates in river water. *Applied Microbiology*, **24,** 168–174

Hickey, J. L. S. and Reist, P. C. (1975). Health significance of airborne micro-organisms from wastewater treatment processes. 1. Summary of investigations. *Journal of the Water Pollution Control Federation*, **47,** 2741–2757.

Higgins, A. R. and Floyd, T. M. (1955). Studies in shigellosis. *American Journal of Tropical Medicine*, **4**, 263–300.

Hollister, A. C., Beck, M. D., Gittelsohn, A. M. and Hemphill, E. C. (1955). Influence of water availability on *Shigella* prevalence in children of farm labor families. *American Journal of Public Health*, **45,** 354–362.

Hutchinson, R. I. (1956). Some observations on the method of spread of Sonne dysentery. *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service*, **15,** 110–118.

Jamieson, W., Madri, P. and Claus, G. (1976). Survival of certain pathogenic microorganisms in sea water. *Hydrobiologia*, **50,** 117–121.

Kabler, P. (1959). Removal of pathogenic micro-organisms by sewage treatment processes. *Sewage and Industrial Wastes*, **12,** 1373–1382.

Katzenelson, E., Buium, I. and Shuval, H. I. (1976). Risk of communicable disease infection associated with wastewater irrigation in agricultural settlements. *Science*, **194,** 944–946.

Khan, M., Curlin, G. T. and Huq, I. (1979). Epidemiology of *Shigella dysenteriae*, type 1 infections, in Dacca urban area. *Tropical and Geographical Medicine*, **31,** 213–223.

Khan, M. and Mosley, W. H. (1968). The significance of *Shigella* as a cause of diarrhea in a low economic urban community in Dacca. *East Pakistan Medical Journal*, **12,** 45–51.

Khan, M. and Shahidullah, Md. (1980). *Epidemiologic Study of Dysentery Cases of Dacca Urban Area.* Scientific Report no. 33. Dacca: International Centre for Diarrhoeal Disease Research, Bangladesh.

Kligler, I. J. (1921). *Investigation on Soil Pollution and the Relation of the Various Types of Privies to the Spread of Intestinal Infections.* Monographs of the Rockefeller Institute for Medical Research, 15. New York.

Levine, M. M., DuPont, H. L., Formal, S. B., Hornick, R. B., Takeuchi, A., Gangarosa, E. J., Snyder, M. J. and Libonati, J. P. (1973). Pathogenesis of *Shigella dysenteriae* 1 (Shiga) dysentery. *Journal of Infectious Diseases*, **127,** 261–270.

Lewis, J. N., Loewenstein, M. S., Guthrie, L. C. and Sugi, M. (1972). *Shigella sonnei* outbreak on the Island of Maui. *American Journal of Epidemiology*, **96,** 50–58.

McCabe, L. J. and Haines, T. W. (1957). Diarrheal disease control by improved human excreta disposal. *Public Health Reports*, **72,** 921–928.

McFeters, G. A., Bissonnette, G. K., Jezeski, J. J., Thomson, C. A. and Stuart, D. G. (1974). Comparative survival of indicator bacteria and enteric pathogens in well water. *Applied Microbiology*, **27,** 823–829.

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

Nakamura, M. (1962). The survival of *Shigella sonnei* on cotton, glass, wood, paper and metal at various temperatures. *Journal of Hygiene*, **60,** 35–39.

Nakamura, M., Stone, R. L., Krubsack, J. E. and Pauls, F. P. (1964). Survival of *Shigella* in sea water. *Nature*, **203,** 213–214.

Newson, S. W. B. (1972) Microbiology of hospital toilets. *Lancet*, **2,** 700–703.

Olivieri, V. P., Kawata, K. and Krusé, C. W. (1978). Relationship between indicator organisms and selected pathogenic bacteria in urban waterways. *Progress in Water Technology*, **10,** 361–379.

Petrick, M. (1954). Utilization of night soil, sewage and sewage sludge in agriculture. *Bulletin of the World Health Organization*, **10,** 207–228.

Rahaman, M. M. (1979). A strategy for control of shigellosis (dysentery) in Teknaf—a rural Bangladesh village. *Progress in Water Technology*, **11,** 303–308.

Rahaman, M. M., Khan, M. M., Aziz, K. M. S., Islam, M. S. and Kibriya, A. K. M. G. (1975). An outbreak of dysentery caused by *Shigella dysenteriae* Type 1 on a coral island in the Bay of Bengal. *Journal of Infectious Diseases*, **132,** 15–19.

Rajasekaran, P., Dutt, P. R. and Pisharoti, K. A. (1977). Impact of water supply on the incidence of diarrhoea and shigellosis among children in rural communities in Madurai. *Indian Journal of Medical Research*, **66,** 189–199.

Reeves, J. B. (1959). Sanitary aspects of composted sewage sludge and sawdust. *Sewage and Industrial Wastes*, **31,** 557–564.

Reller, L. B., Gangarosa, E. J. and Brachman, P. S. (1969). Shigellosis in the United States, 1964–1968. *Journal of Infectious Diseases*, **120,** 393–396.

Richardson, N. J., Hayden-Smith, S., Brokkenheuser, V. and Koornhof, H. J. (1968). Salmonellae and shigellae in Bantu children consuming drinking water of improved quality. *South African Medical Journal*, **42,** 46–49.

Rosenberg, M. L., Hazlet, K. K., Schaefer, J., Wells, J. G. and Pruneda, R. C. (1976). Shigellosis from swimming. *Journal of the American Medical Association*, **236,** 1849–1852.

Rudolfs, W., Falk, L. L. and Ragotzkie, R. A. (1951). Contamination of vegetables grown in polluted soil. VI. Application of results. *Sewage and Industrial Wastes*, **23,** 992–1000.

Schliessmann, D. J. (1959). Diarrhoeal disease and the environment. *Bulletin of the World Health Organization*, **21,** 381–386.

Schliessmann, D. J., Atchley, F. O., Wilcomb, M. J. and Welch, S. F. (1958). *Relation of Environmental Factors to the Occurrence of Enteric Diseases in Areas of Eastern Kentucky*. Public Health Monograph no. 54. Washington, DC: US Government Printing Office.

Shaughnessy, H. J., Olsson, R. C., Bass, K., Friewer, F. and Levinson, S. O. (1946). Experimental human bacillary dysentery. *Journal of the American Medical Association*, **132,** 362–368.

Shrewsbury, J. F. D. and Barson, G. J. (1957). On the absolute viability of certain pathogenic bacteria in a synthetic well water. *Journal of Pathology and Bacteriology*, **74,** 215–220.

Slijkhuis, H., Betzer, N. and Kott, Y. (1976). Survival of *Shigella flexneri* in an experimental oxidation pond. *Israel Journal of Medical Science*, **12,** 704–705.

Spicer, C. C. (1959). The survival of *Shigella sonnei* on cotton threads. *Journal of Hygiene*, **57,** 210–215.

Stewart, W. H., McCabe, L. J., Hemphill, E. C. and DeCapito, T. (1955). Diarrheal disease control studies. IV. Relationship of certain environmental factors to the prevalence of *Shigella* infection. *American Journal of Tropical Medicine and Hygiene*, **4,** 718–724.

Sultanov, G. V. and Solodovnikov, Y. P. (1977). Significance of water factor in epidemiology of dysentery. *Zhurnal Mikrobiologii, Epidemiologii i Immunobiologii*, no. 6 (June), 99–101.

Talayeva, J. G. (1960). Survival of dysentery bacteria in water according to the results of a reaction with haptenes. *Journal of Hygiene, Epidemiology, Microbiology and Immunology*, **4,** 314–320.

Taylor, B. C. and Nakamura, M. (1964). Survival of shigellae in food. *Journal of Hygiene*, **62,** 303–311.

Thomson, S. (1955). The numbers of pathogenic bacilli in faeces in intestinal diseases. *Journal of Hygiene*, **53,** 217–224.

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.

Wang, W. L. L., Dunlop, S. G. and De Boer, R. G. (1956). The survival of *Shigella* in sewage. 1. An effect of sewage and faecal suspensions on *Shigella flexneri*. *Applied Microbiology*, **4,** 34–38.

Watt, J. and Lindsay, D. R. (1948). Diarrheal disease control studies. 1. Effect of fly control in a high morbidity area. *Public Health Reports*, **63,** 1319–1334.

Watt, J., Hollister, A. C., Beck, M. D. and Hemphill, E. C. (1953). Diarrheal diseases in Fresno County, California. *American Journal of Public Health*, **6,** 728–741.

Wiley, J. S. (1962). Pathogen survival in composting municipal wastes. *Journal of the Water Pollution Control Federation*, **34,** 80–90.

Wolff, H. L., van Zijl, W. J. and Roy, M. (1969). Houseflies, the availability of water, and diarrhoeal diseases. *Bulletin of the World Health Organization*, **41,** 952–959.