An Outbreak of Shigella Gastroenteritis

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IN September 1954, an explosive outbreak of gastroenteritis occurred in a rural elementary school in central Ohio. Investigation by the division of communicable diseases of the Ohio Department of Health determined that the outbreak was related to food served in the school cafeteria. The etiological agent was found to be *Shigella sonnei*, type 1.

The American literature contains few well-documented reports of foodborne shigellosis. In 1950, Feig reviewed reports of outbreaks of diarrheal disease received by the Public Health Service from 1945 through 1947 (*1*). During this period there were 476 outbreaks characterized bacteriologically. Of these, only 14 were associated with *Shigella* organisms (only 3 with *S. sonnei*).

In surveys of normal population groups in the late 1930's and early 1940's, Watt and Hardy found the prevalence of shigellosis by culture to be 11 percent in New Mexico, 4 percent in Puerto Rico, 3 percent in Georgia, and 0.1 percent in New York City (*2*). In general, the infection rate was greatest in the 1- to 9-year age group. In a study of clinical cases of diarrheal diseases, Hardy and Watt found that *S. sonnei* accounted for 20 to 25 percent of the shigella infections in New Mexico and in Georgia and for 57 percent in New York City (*3*).

Dr. Keller and Dr. Robbins, at the time of this study, were officers of the Epidemic Intelligence Service, Communicable Disease Center, Public Health Service, on assignment to the division of communicable diseases, Ohio Department of Health. Dr. Keller is now assistant resident in medicine at the Bronx Veterans Administration Hospital, New York City, and Dr. Robbins was scheduled to become senior resident in pediatrics at the Bronx Municipal Hospital Center on July 1, 1956.

Investigation of the Outbreak

The first illness in the Ohio outbreak occurred at 3 a. m. on September 22, 1954, and the majority of the cases began during the afternoon and night of the same day. Through inquiry among local physicians and at nearby schools, it was determined that the illness was limited to the children and teachers of one elementary school.

The illness was characterized by fever, chills, headache, abdominal pain, nausea, vomiting, diarrhea, and prostration. In most cases, the temperature (oral) ranged from 100° to 105° F. Several cases began with convulsions. The illness usually lasted from 24 to 72 hours, and severe symptoms rarely persisted longer than 24 hours. None of the patients were hospitalized. About one-third of them were seen by physicians. The drugs most frequently used were neomycin, sulfadiazine, kaopectate, and paregoric.

The school was closed before noon on September 23 because of marked absenteeism, and it remained closed until September 27. The school population consisted of 268 children and a staff of 12. On September 23, visits were made to some of the homes in the community to locate sick children. Eighteen acutely ill children were discovered in this manner, and his-

tories and rectal swabs for culture were obtained from them. Rectal swabs from the two food handlers in the school cafeteria and samples of the foods served during the preceding 2 days were also obtained for culture. The only food on the menu that could not be obtained was the potato salad served on September 21.

The parents of the school children were questioned on September 24, and data were obtained concerning the occurrence of illness, the time of onset, the clinical manifestations, and the history of foods consumed in the school cafeteria on September 21 and 22.

On September 25, *S. sonnei*, type 1, was isolated from 12 of the 18 original rectal swabs. Antibiotic sensitivity tests showed the organisms to be sensitive to aureomycin, terramycin, and chloramphenicol, but resistant to sulfadiazine and dihydrostreptomycin.

When the school reopened on September 27, all the children were questioned as to the occurrence of illness, the symptoms experienced, and the foods consumed. Rectal swabs were obtained from all the students and the staff.

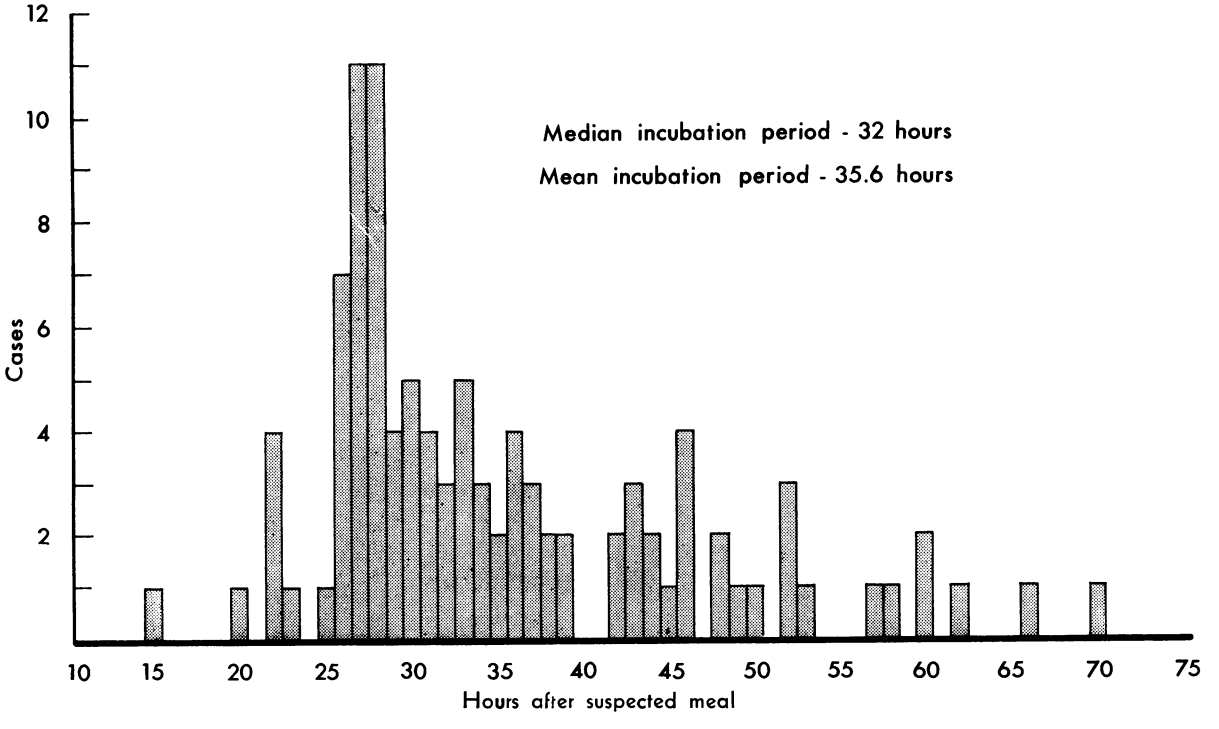
Six weeks later, rectal swabs were again obtained from the students and staff, and families were questioned concerning the occurrence of subsequent illness among other members of the household. No fecal specimens were obtained from the household contacts.

Laboratory Procedures

The fecal specimens were obtained by swabbing the walls of the anal canal with a sterile cotton swab. The soiled swab was placed in a tube containing a broth designed to act as a selective medium. Culture plates were streaked with the inoculated medium on the swab within 6 hours. The broth, which was prepared according to the formula of Hajna from the Bacto dehydrated product, is a medium in which gram-negative organisms are enriched and gram-positive organisms are inhibited (*4*). It has been shown by Croft and Miller to be particularly effective for the isolation of shigellae if the inoculated medium is kept no longer than 8 hours before streaking culture plates (*5*).

Colonies of non-lactose-fermenting bacteria from Endo and SS agar plates were transferred to triple sugar iron (TSI) agar slants. Those giving shigella reactions on TSI (alkaline slant,

Figure 1. Onset of clinical cases of gastroenteritis during the acute outbreak, September 22 through September 24, 1954.



acid butt, and negative H_2S) were tested with shigella group serums. After the causative organism was identified as *S. sonnei*, type 1, only the specific antiserum was used in the slide test for identifying suspected cultures.

The isolated organisms were tested for antibiotic sensitivity by the Difco disk method.

Table 1. Illness and culture data related to consumption of suspected meal

Consumption of suspected meal	Ill		Not ill		Total
	Positive culture	Negative culture	Positive culture	Negative culture	
Ate	92	56	13	46	207
Did not eat	0	3	2	29	34
Total	92	59	15	75	241

Table 2. Infection rate related to consumption of suspected meal

Consumption of suspected meal	Infected[1]	No evidence of infection	Total	Infection rate (percent)
Ate	161	46	207	77.8
Did not eat	5	29	34	14.7
Total	166	75	241	68.9

[1] Clinical illness or positive culture, or both.

The food samples were homogenized or diluted and streaked on culture plates. Staphylococcus 110 medium was employed for the isolation of staphylococci, and Endo agar, for the isolation of salmonellae and shigellae. No pathogenic organisms were found.

Bacteriological examination of water samples taken from the school gave negative results for coliform organisms.

Results

The time of onset of illness was learned for 104 of the 151 persons who were ill. The epidemic curve is given in figure 1. After the first case, the curve showed a rapid rise, reaching a peak in approximately 12 to 13 hours. The occurrence of cases then fell off gradually, the final case appearing 55 hours after the first. The epidemic curve indicates a single source, single exposure epidemic, and it appears that the incriminated meal was the lunch of September 21. This lunch would give an incubation period ranging from 15 to 70 hours, with a mean of 35.6 hours and a median of 32 hours. Feig reported a median of 42 hours in 6 outbreaks of shigellosis (*1*).

Information on the following was obtained for 241 of the 280 students and staff members: consumption of the suspected meal, occurrence of illness, and results of rectal culture. Illness was defined as the presence of two or more of the following: fever, abdominal pain, nausea, vomiting, and diarrhea.

Only the data for these 241 persons are given in tables 1 through 5. However, partial data were obtained for an additional 32 persons, 20 of whom showed evidence of infection either by illness or by positive fecal culture. No information was obtained for seven of the school population.

The number of persons ill and the number of positive fecal cultures among persons who ate the suspected meal and those who did not eat it are shown in table 1.

Table 2 compares the attack rates based on

Table 3. Clinical illness rate related to consumption of suspected meal

Consumption of suspected meal	Ill	Not ill	Total	Illness rate (percent)
Ate	148	59	207	71.5
Did not eat	3	31	34	8.8
Total	151	90	241	62.6

Table 4. Positive culture rate related to consumption of suspected meal

Consumption of suspected meal	Positive culture	Negative culture	Total	Positive culture rate (pecent)
Ate	105	102	207	50.7
Did not eat	2	32	34	5.9
Total	107	134	241	44.4

evidence of infection (clinical illness or positive culture, or both) for the same two categories. These rates offered statistically significant evidence that infection was related to consumption of the suspected meal.

Table 3 shows that for both those who ate the suspected meal and those who did not eat it the rate of clinical illness was nearly as high as the attack rate based on evidence of infection. These findings differ from the expected ratio of clinical cases to carriers.

From table 4, it can be seen that the positive culture rates were substantially lower than the infection rates. However, on the basis of one culture, the rate for those who ate the suspected meal represents a rather high percentage of isolation of *S. sonnei* from an exposed population.

Table 5. Positive culture rate related to clinical illness

Illness status	Positive culture	Negative culture	Total	Positive culture rate (percent)
Ill	92	59	151	61. 0
Not ill	15	75	90	16. 6
Total	107	134	241	44. 4

Table 5 shows that positive cultures were found in 61.0 percent of the persons who were ill, but in only 16.6 percent of those who were not ill.

Table 6, which gives data for 237 of the school children, reveals that there were no statistically significant differences in infection rate for the various ages.

Detailed descriptions of symptoms were obtained for 122 of the persons who were ill. The following symptoms were reported most frequently: diarrhea, for 103 persons (84 percent); fever, for 83 (68 percent); and vomiting, for 49 (40 percent).

Information concerning the specific foods eaten for lunch on September 21 and 22 was considered to be unreliable because of the age of the patients and the general imperfections of memory. Nearly all the students ate some of all of the foods served, as is the custom in school cafeterias.

Table 6. Infection rate according to age

Age, in years	Infected[1]	No evidence of infection	Total	Infection rate (percent)
5	1	1	2	50. 0
6	21	9	30	70. 0
7	22	11	33	66. 7
8	18	10	28	64. 3
9	8	13	21	38. 1
10	17	7	24	70. 8
11	28	6	34	82. 4
12	24	10	34	70. 6
13	15	4	19	78. 9
14	5	4	9	58. 3
15	1	1	2	
16	1	0	1	

[1] Clinical illness or positive culture, or both.

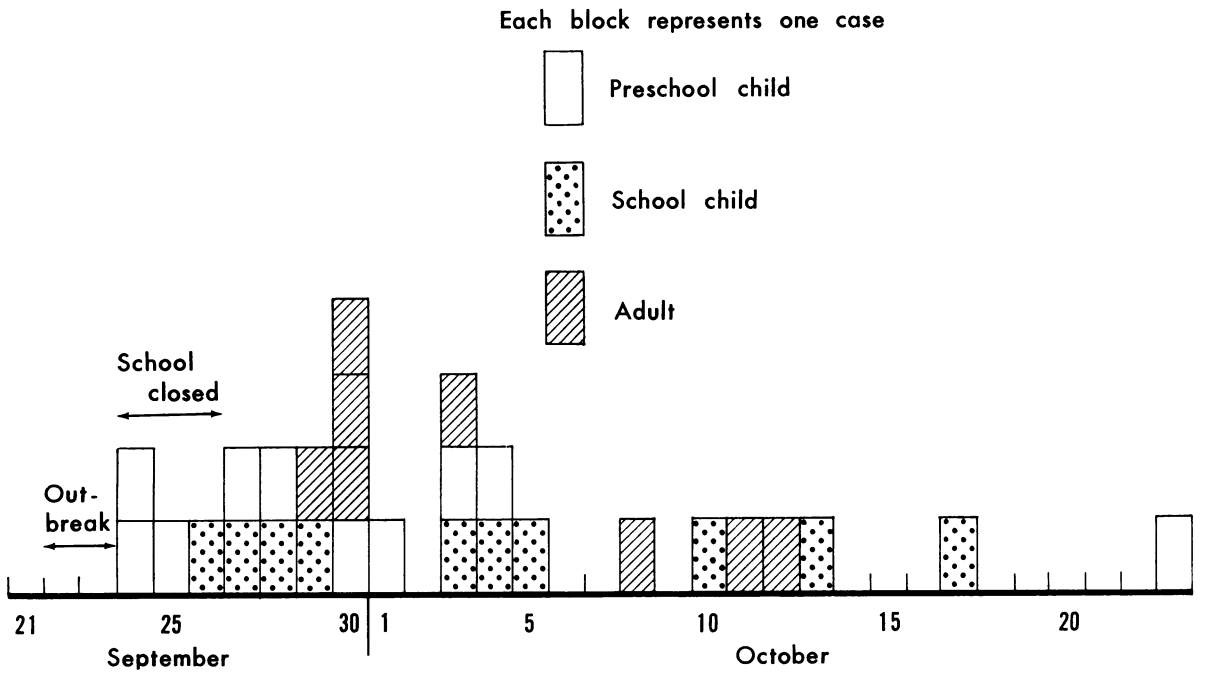
Six weeks after the onset of the first illness, rectal swabs were obtained from 234 of the students and staff members of the school. Only 5 cultures positive for *S. sonnei*, type 1, were found, and of these, only 3 were for persons who 6 weeks earlier had a positive culture. For all 5, 2 consecutive negative cultures were obtained during the following 2 weeks.

Information concerning subsequent cases of gastroenteritis in household contacts was obtained from 81 families 6 weeks after the outbreak. In these families there were 109 children infected during the initial outbreak and 293 household contacts. Twenty-eight of the household contacts were infected, a secondary attack rate of 9.5 percent. The dates of onset of illness and the distribution among preschool children, school children, and adults are shown in figure 2. Illness in household members with no exposure to the source of the infection was considered a secondary infection even though it occurred on the last day of the outbreak.

Discussion

The school lunch of September 21 was considered the common, single source of infection in this outbreak of shigellosis because of its relation to the time of onset of the cases. If the lunch of September 20 were the source, the first case would have had an incubation period of 39 hours and the median of the incubation periods would have been 56 hours. The lunch of September 23 is eliminated because at least seven

Figure 2. Occurrence of secondary cases in household contacts of persons infected during the acute outbreak.



cases had their onset before the lunch was served.

Neither of the two food handlers was ill, but both had cultures positive for *S. sonnei*, type 1. It is possible that they became infected in the same manner as did the rest of the school population. However, it is conceivable that one of them carried the organisms before the outbreak occurred and contaminated the foodstuffs served at the lunch on September 21.

The last contact the children had with one another in school during the outbreak was on the morning of September 23, approximately 46 hours after the lunch on September 21. There is a possibility that some of the late cases were secondarily infected from the early ones. However, it is reasonable to explain the epidemic curve on the basis of a single source and a single exposure.

The attack rate of 77.8 percent, based on the occurrence of illness or the finding of *S. sonnei*, type 1, or both, points to massive exposure of the population of the school. However, 39 percent of those who were ill had negative cultures, and 16.6 percent of those who had no clinical illness were shown to be asymptomatic carriers. It is reasonable to suppose that a considerable number of asymptomatic carriers were missed, since only one culture was obtained during the outbreak for each person.

Watt and Hardy found that infection without clinical disease was rare in infants, but that it progressively increased up to the age of 5 years (*2*). Thereafter, it occurred at a fairly uniform rate. For all age groups, the ratio of convalescent or passive carriers to each current case was 9.1. They also found that the ratios were fairly uniform for different types of shigellae. They concluded that manifest sources of infection are rare in comparison to hidden sources. In a study of shigella infection in a closed institutional population, Hardy, Shapiro, Chant, and Siegel found that the ratio of carriers to clinical cases was greater for *S. sonnei*, 24:1, than for *Shigella flexneri*, 7:1, (*6*).

In the present study, only 5 persons had positive cultures 6 weeks after the initial outbreak, and these became negative on at least 2 subsequent samples within the next 2 weeks. These findings are in keeping with the findings of Watt, Hardy, and DeCapito (*7*). Of 103 patients with proved shigellosis, 80 percent had positive cultures after the acute symptoms sub-

sided, but the duration of infection after recovery was only 32 days for *S. flexneri* and only 22 days for *S. sonnei*. These investigators felt that the chronic carrier was exceptional in bacillary dysentery and that the organism is more frequently perpetuated in the community by a constantly changing group of hosts.

Cruickshank and Swyer studied an outbreak of 32 cases of shigellosis in a residential school (*8*). Twenty-nine gave positive cultures initially, and the number of positive cultures decreased slowly until only one was found in the 10th week. However, 10 patients, 34.4 percent of the group, had 2 or more consecutive negative cultures followed by 1 or more positive cultures, and 5 patients had 3 or more consecutive negative specimens over a period of 3 weeks, only to yield positive cultures again. These findings indicate that the data obtained in the present study from one initial culture and one followup culture after a period of 6 weeks can give only a rough estimate of the extent of the original infection and the persistence of the infection in the group. However, the figures obtained by this method are in keeping with those found by other workers.

The Ohio outbreak appears to have been quite different from an outbreak of shigellosis in Oxford, England, reported by Davies (*9*). In the Oxford outbreak, the first case occurred in a school for infants, but the spread of infection seemed to be mainly in the homes. Of 74 primary cases, 47 occurred in the original school and 27 in 10 other schools. Among the 293 home contacts of these 74 cases, 234 of whom had at least one stool examination, 16.3 percent had clinical illness and 33.3 percent had positive cultures but no clinical illness. In the Ohio epidemic, the school was the immediate source of infection for 81 families. In these families, 109 children were infected during the initial outbreak, and only 28 cases of gastroenteritis were reported among their 293 household contacts. Since no fecal specimens were obtained from the household contacts, the subsequent cases can only be presumed to be related to the initial outbreak. However, it is probable that considerably more persons in these families became infected but did not have clinical illness.

The clinical manifestations observed in the Ohio outbreak were similar to those described by Blatt and Shaw (*10*) and Cruickshank and Swyer (*8*).

Zimmerman, Cooper, and Graber studied an epidemic of shigella infection in Korea in 1952 (*11*). They found sulfadiazine therapy unsatisfactory. Chloramphenicol, terramycin, and aureomycin were found to be highly effective, and streptomycin was intermediate. All 13 types of shigellae that they isolated were found to be highly sensitive to chloramphenicol, terramycin, and aureomycin, but not sensitive to sulfadiazine and dihydrostreptomycin. In recent years, few sulfadiazine-sensitive shigellae have been isolated by the Ohio Department of Health laboratories.

Thus, the expense of treating shigella infections poses a special problem. In massive outbreaks in rural areas, where income may be low, it is often not possible to treat all patients with the most effective antibiotics. However, in the outbreaks described in this paper, all those who were ill recovered without apparent complications within a period of a few days, despite the fact that very few were given antibiotic therapy.

Summary

An outbreak of gastroenteritis in a rural elementary school in central Ohio in September 1954 is described. From data collected concerning occurrence of illness, time of onset, consumption of a suspected meal, and results of fecal cultures, it appeared that the outbreak followed a mass exposure to *Shigella sonnei* at a school lunch.

The first illness occurred 15 hours after the suspected meal. After this, the number of cases increased rapidly, and the peak of the outbreak occurred 27 to 28 hours after the meal. The median of the incubation periods was 32 hours, and the range was from 15 to 70 hours.

For 241 of the 280 students and staff members of the school, the following findings were obtained: Of those who ate the suspected meal, 71.5 percent became ill; of those who did not eat this meal, only 8.8 percent. From one fecal specimen taken within 8 days of the beginning of the outbreak, *S. sonnei*, type 1, was isolated

from 50.7 percent of those who ate the suspected meal but from only 5.9 percent of those who did not eat it. Considering those persons who were ill or who had a positive fecal culture, the attack rate for all was 77.8 percent. Of the persons who were ill, 61 percent had positive fecal cultures; of those who were not ill, only 16.6 percent were positive.

Two food handlers gave positive fecal cultures, but they had no clinical illness. No specific food was implicated.

Six weeks after the occurrence of the first case, when 234 fecal specimens were taken, only 5 contained *S. sonnei*, type 1.

Among 293 household contacts of 109 infected children, in 81 families, only 28 (9.5 percent) became ill with gastroenteritis within 6 weeks after the outbreak. Of these, 20 were children under 15 years of age.

Other reports of shigella infection are discussed briefly and compared with the Ohio outbreak.

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Traineeships for Public Health Personnel

Authorized by the Health Amendments Act of 1956 (P. L. 911), the Public Health Service has appropriated $1 million to establish a program of traineeships for graduate or specialized public health training of professional public health personnel for the fiscal year ending June 30, 1957.

The funds will include necessary costs, such as living expenses, tuition, and travel, as determined by the Surgeon General. All professional public health personnel are eligible.

Information and application forms may be obtained from any of the regional offices of the Public Health Service, or from the Chief, Division of General Health Services, Bureau of State Services, Public Health Service, Washington 25, D. C.