

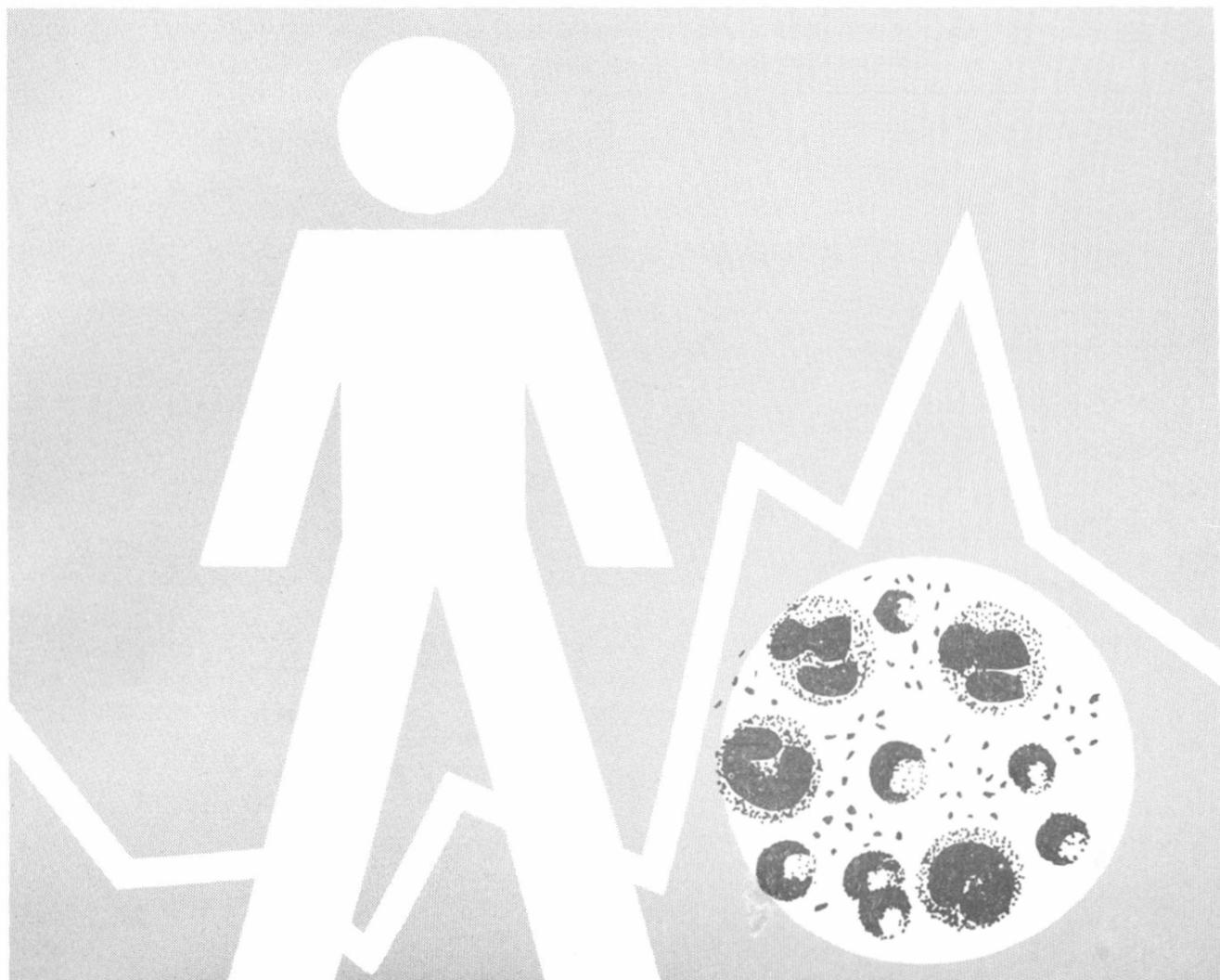
center for disease control

SHIGELLA

surveillance

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for the
Third and Fourth Quarters 1972

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I. SUMMARY

For the period July-December 1972, 7,486 shigella isolations from humans were reported. This represents an increase of 1,174 (18.6%) over the 6,312 isolations reported for the preceding 6 months and an increase of 282 (3.9%) over the 7,204 isolations reported for the corresponding months of 1971 (Tables I-A - I-B, pp 14-17)*

II. REPORTED ISOLATIONS

A. Human

1. General Incidence

For the 2nd half of 1972, 68.3% of reported isolations were from children under 10 years of age (Table II); this is consistent with previous 6-month periods. The highest attack rate was in the 1-4 age group, and the 2nd highest attack rate was in the less than 1 year age group.

2. Serotype Frequency

Fifty-three of the 54 reporting centers in the Shigella Surveillance Program reported isolations of shigella. Twenty-five different serotypes were reported. The 6 most frequently reported serotypes for the 6-month period were the following (Table 1).

Table 1

Rank	Serotype	Number Reported	Calculated Number**	Calculated Percent**	Rank 2nd Quarter
1	<u>S. sonnei</u>	5,907	5,926	79.2	1
2	<u>S. flexneri</u> 2a	284	572	7.6	2
3	<u>S. flexneri</u> 3a	125	264	3.5	4
4	<u>S. flexneri</u> 6	179	220	2.9	3
5	<u>S. flexneri</u> 2b	60	121	1.6	6
6	<u>S. flexneri</u> 4a	71	112	1.5	5
	Subtotal	6,626	7,215	96.3	
	Total (all serotypes)	7,486	7,487		

**From Table III

Table III is calculated from data compiled for the 2nd half of 1972. This table shows the frequency of reported isolations of the various serotypes; the isolations in each of the unspecified categories are distributed over their subgroups in the same proportions as the completely specified isolations of that group. The resulting distribution in the tables is called the "calculated number," and from this is derived a "calculated percent" for each serotype. These provide approximate indices of the relative frequency of reporting of the more common shigella serotypes in the United States. S. sonnei accounted for approximately 79.2% of all reported isolations. Table IV shows the distribution of shigella serotypes reported from mental institutions.

*No laboratory reports were received from California or the Virgin Islands.

3. Geographical and Seasonal Observations

There were more reported isolations of *S. sonnei* than *S. flexneri* in all but the following 6 states: Delaware (1:1), Montana (8:20), Nevada (3:5), South Dakota (6:45), Mississippi (20:24), and Arizona (128:192) (Figure 1). This is consistent with what has been observed in the past in that the reported incidence of *S. flexneri* is, in general, decreasing, while the reported incidence of *S. sonnei* is increasing. The seasonal distribution is depicted in Figure 2. Figure 3 shows the number of reported isolations per million population by state for July--December utilizing 1970 census figures. Approximately 40.6 isolations per million population were reported for the 2nd half of 1972 (California not included). Table V shows the general type of residence of those patients from whom shigella was isolated and reported.

Figure 1 PERCENTAGE *S. flexneri* AND *S. sonnei* OF TOTAL SHIGELLA ISOLATIONS REPORTED FROM INDICATED REGIONS UNITED STATES, JULY - DECEMBER, 1972

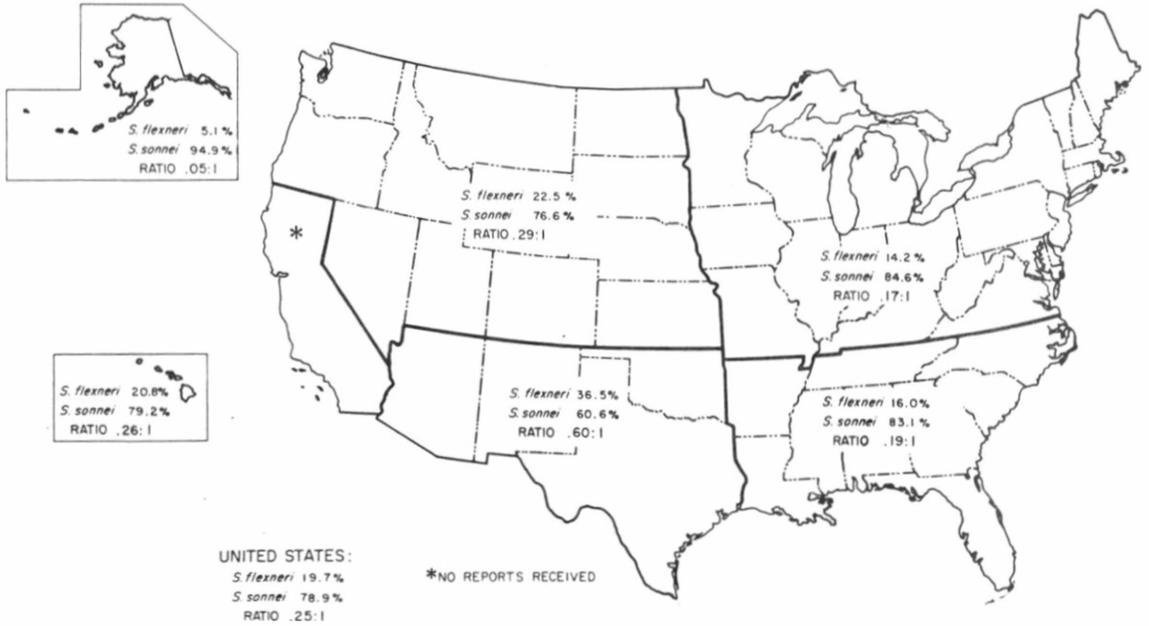
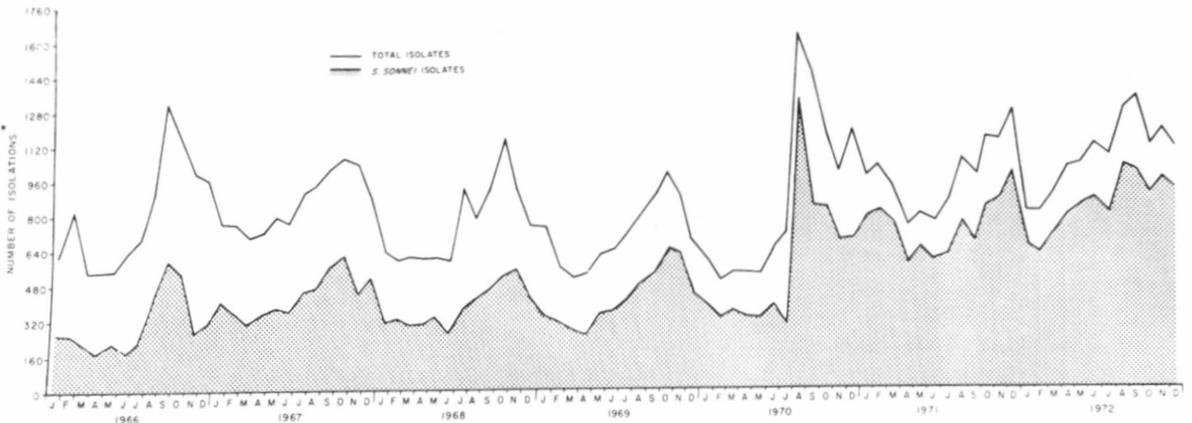
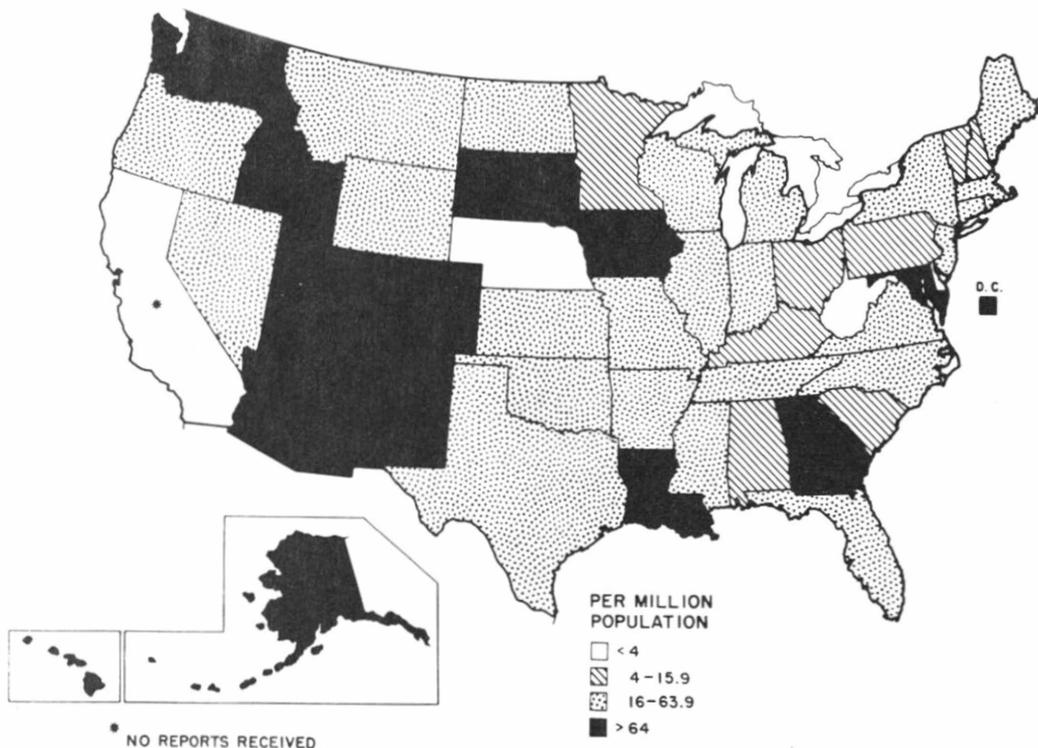


Figure 2 REPORTED ISOLATIONS OF SHIGELLA IN THE UNITED STATES



* ADJUSTED TO 4-WEEK MONTH

Figure 3 ATTACK RATES OF SHIGELLOSIS, BY STATE, JULY-DECEMBER 1972



B. Nonhuman

For the period July-December 1972 19 isolations from primates were reported.

Table 2

<u>Serotype</u>	<u>Number</u>	<u>Source</u>	<u>State</u>
<u>S. flexneri</u> (unspec)	1	spider monkey	Oregon
<u>S. flexneri</u> 1	1	spider monkey	Maryland
<u>S. flexneri</u> 2a	1	baboon	Hawaii
	1	primate	Texas
<u>S. flexneri</u> 3 (unspec)	1	monkey	Arizona
	1	gibbon	Hawaii
<u>S. flexneri</u> 3b	1	baboon	Louisiana
<u>S. flexneri</u> 4a	1	monkey	Hawaii
	2	monkey	Illinois
	1	rhesus monkey	Illinois
	1	rhesus monkey	Louisiana
<u>S. flexneri</u> 4b	2	monkey	Illinois
	2	stumptailed monkey	Illinois
	1	rhesus monkey	Illinois
<u>S. flexneri</u> 6	1	rhesus monkey	Maryland
<u>S. sonnei</u>	1	monkey	Illinois

III. CURRENT TOPICS

A. Waterborne Shigellosis in the United States

Although person-to-person transmission is the predominant form of spread of shigellosis, waterborne outbreaks have played a significant role in the overall epidemiology of the disease. Because contaminated water, particularly a public

water supply, may potentially infect thousands of individuals,¹ the public health implications of these sporadic outbreaks cannot be overemphasized.

Between 1938-1945, 327 waterborne outbreaks of intestinal illness were reported in the United States, accounting for 11,320 cases, 8,622 of which were due to shigellosis. In the 25-year period 1946-1970, there were 358 reported outbreaks of waterborne disease with 72,358 cases, 7,400 of which were attributed to shigellae.^{1,2} These data are summarized in Table 3.

Table 3

Waterborne Intestinal Disease in the United States, 1938-1972

Period	Outbreaks	Cases	Shigella	
			Outbreaks	Cases
1938-1945	327	111,320	35	8,622
1946-1960		25,984	24	5,734
1961-1970	130	46,374	9	1,666
1971	-	-	4	623
1972	-	-	2	233

Private water supplies such as wells are responsible for the majority of reported outbreaks; however, because municipal water supplies tend to serve large populations rather than single families, the majority of cases of waterborne illness are tracable to public water supplies.¹ Contamination of a single public water supply system accounted for over 16,000 cases of salmonellosis in 1 reported epidemic; cases of gastroenteritis numbering in the thousands are not unusual in outbreaks³ involving a public water system. A sample survey of 969 public water supply systems (5% of the U.S. total) undertaken in 1969 showed that 51% had no treatment or chlorination, emphasizing the continuing potential for these systems to be involved in disease transmission.⁸

The following reports, based upon recent outbreaks investigated in association with the Center for Disease Control, illustrate several principles relevant to the causes and control of waterborne shigellosis. One outbreak, which occurred several years ago, is included because of its unusual and important epidemiologic features.

New Jersey--New York⁴

In August 1971, 99 campers and staff members from a summer camp in Sullivan County, New York, visited a camp in Warren County, New Jersey, to play basketball. Although they had brought their own food and drink, many drank water at the New Jersey camp even after noticing its "swamp-like" odor. The following day, 64 people from the New York camp fell ill with gastrointestinal symptoms. Four of 9 stool specimens cultured grew S. sonnei. Food specific attack rates implicated the camp water as the vehicle of infection. Although little gastrointestinal illness occurred among the New Jersey campers coincident with illness in the New York camp, investigation disclosed a high incidence of gastrointestinal illness there throughout the summer. Two separate wells supplying the camp were both contaminated with coliforms; neither water supply had been adequately chlorinated.

Alaska⁵

Eighty-nine of 114 residents of a trailer court in Anchorage, Alaska, developed gastrointestinal illness between November 10 and December 5, 1971, for an attack rate of 78%. Their illness was characterized by nausea, vomiting, fever, abdominal pain, and diarrhea. There were no significant age or sex differences among patients. Stool specimens from 17 patients residing in 6 affected trailers yielded S. sonnei, and 9 of 14 persons with gastrointestinal illness who lived elsewhere but had visited the trailer court also had positive stool cultures for S. sonnei.

Residents had noted that on November 16 the water from all household taps was "dirty" and had a bad odor. The trailer court's water supply consisted of 2 wells, each 240 feet deep, enclosed in a cinder block well house. The well house floor was 3 feet below ground, and the well casing extended 1 foot above the well house floor. There was no chlorination apparatus in the system.

Investigation disclosed that a blockage in the area sewerage system had caused a back-up of sewage onto the well house floor. Because the well was unsealed, sewage entered the wells and subsequently the trailer court water system.

Despite repeated attempts to superchlorinate the wells, water samples continued to have unacceptable coliform levels. Since the wells could not be adequately decontaminated, they were closed. Normal use of water was restored only after connecting the trailer court water system to the borough water supply.

Mississippi⁶

One-hundred eighty-seven cases of gastrointestinal illness were recorded in July 1971 among participants at a month-long religious convocation in Bay St. Louis, Mississippi. Eight of 9 rectal cultures obtained from ill persons were positive for S. sonnei. Overall attack rate for the group was 62.3%.

Epidemiologic investigation incriminated the water system of the building housing the participants. Water was supplied by the city from an artesian well, and although a chlorination apparatus was part of the system, it was not being used. A booster pump, originally connected to the building's water supply system to boost city water pressure, was no longer necessary because of the adequacy of the city's water supply, but remained ready to activate if the water pressure fell.

The week prior to the epidemic, the water had been turned off to permit the repair of a nearby fire hydrant. Water pressure remained low for several days; the water system was closed down a second time later in the week to permit repair of a water meter at the site which had jammed with sand. After cleaning, the water supply was reestablished and functioned normally thereafter.

When the water was off, the auxiliary pump had turned itself on to maintain water pressure in the building, causing negative pressure in pipes between it and the city water supply.

Shortly after the epidemic began, the clay pipe sanitary sewerage line became clogged. When replaced, it was found broken in many places with tree roots growing through it. The main water line ran over the broken sewerage line and a break in this water line may have enabled sewage to enter the water supply at a time when water pressure was negative.

The investigation also disclosed a bathtub with a long shower hose attached to its faucet. This tub was used frequently to bathe youngsters from the infirmary, particularly those with diarrhea. When the tub was filled with water and the end of the shower hose submerged, contamination of the main water supply could have occurred if, when the city water supply was off, the auxiliary pump caused negative pressures in the water line and allowed water from the tub to enter the supply system through the shower hose. This was demonstrated by adding fluorescein dye to the bathtub water and observing its appearance at various faucets.

Another site of potential direct contamination was found in the laundry room where faucets extended below the top of the sinks which had no overflow drain.

Iowa⁷

In November 1972, an outbreak of waterborne S. sonnei infection occurred in a junior high school in Stockport, Iowa, with 208 cases among 314 students and staff members, for an attack rate of 66%. Rectal swabs from 108 students and 2 staff members were positive for S. sonnei. Ninety-nine apparent secondary cases of diarrhea occurred in household contacts of those attending the school. Gastrointestinal illness was significantly more frequent among those who regularly drank from the school water fountain than from those who did not. Players from a visiting high school basketball team who drank the water at the school were also affected. A sample of the tap water in the school showed high levels of coliform contamination and yielded S. sonnei.

The school obtained its water from a shallow well in the school yard. Fluorescein dye introduced into a drain from a shower in the gymnasium locker room appeared in the well water within 30 minutes. After the onset of the outbreak, the water system was chlorinated and thereafter switched to the municipal water supply.

Utah⁹

On July 19, 1956, an explosive outbreak of gastroenteritis occurred in Roosevelt, Utah, an isolated town of 1,600 in the eastern part of the state. Over 500 cases

were reported, with 16 hospitalizations. Eleven of 16 rectal swabs cultured from acutely ill patients, and 25 (32%) of 78 stool specimens from convalescents grew S. flexneri 6. Thirty-five (16%) of 218 specimens obtained in a culture survey subsequently were also positive for S. flexneri 6.

Epidemiologic investigation disclosed that approximately 2/3 of homes were supplied by the municipal water system, the remainder obtaining their water from private wells. The attack rate for families supplied with city water was 40% compared with only 12% for those who had independent water systems ($p < .001$). The city's water system was derived from 3 wells which were insufficient by themselves for the community's needs, and were supplemented by water from an irrigation canal. Water from both sources was supplied to consumers without any further treatment. Five of 7 samples of canal water showed heavy coliform contamination, further incriminating the water supply. Additional inquiries disclosed that a group of Indians had experienced an outbreak of gastrointestinal illness while encamped in a field bordering the canal. Evidence of indiscriminate defecation was present at the campsite, and a culture survey of Indians who had used the site revealed that 5 (6.3%) of 79 stool specimens obtained were positive for S. flexneri 6.

Several important aspects of waterborne shigellosis are illustrated by these outbreaks. Although the usual person-to-person spread of shigellosis accounts for substantial numbers of secondary cases in any outbreak, the truly spectacular attack rates in these representative reports are characteristic of common source infections (Table 4). The relatively explosive onsets described also point toward a common source of infection, rather than the more smouldering time course with spread by personal contact.

Table 4

<u>Case Report</u>	<u>Attack Rate</u>
1. New York Summer Camp	64%
2. Anchorage Trailer Court	78%
3. Mississippi Religious Gathering	62%
4. Stockport Jr. High School	66%
5. Roosevelt, Utah	~30%

When confronted with a high attack rate and explosive onset, one should suspect water as a common source because it is among the few items to which a large and disparate population may be commonly exposed.^{9,11}

A water source may become nonpotable in several ways. Direct contamination may occur if fecal material directly enters the water supply, either from the surface or at a subsurface level. The high coliform counts in the irrigation water in the Utah outbreak were most likely consequent to direct contamination of the water from the neighboring fields. Back-siphonage occurs when plumbing from a fixture, such as a toilet, bidet, bathtub, or sink, allows drainage to enter the water system, as occurred in the Mississippi outbreak. Cross connections are the consequence of direct linkages between pipes carrying sewage and those carrying potable water and most often occur when plumbing repairs are made in old structures for which accurate blueprints detailing pipe layouts are not available.

A 10-year review of waterborne diseases in the United States¹⁰ has underlined the many different points at which water contamination may occur (Table 5).

Contaminated well water is the most frequently encountered source of contamination, usually because it is untreated prior to use. As illustrated in these reports, if a well is improperly constructed or housed (particularly if it is unsealed or has no sanitary cap), flooding, seepage from septic tanks, or surface contamination of the well may result in massive introduction of waste materials into the water supply, often enough to cause a noticeable change in color, odor, or taste (outbreaks in New Jersey, New York, and Alaska).

Table 5

Waterborne Diseases, U.S.A., 1961-70

Cause	% Distribution of Outbreaks	% Distribution of Cases
Untreated surface water	7.7	1.9
Untreated ground water	47.3	46.6
Contamination of storage	1.6	0.3
Inadequate control of treatment	10.1	43.5
Contamination of distribution system	14.7	3.0
Miscellaneous	18.6	4.7
Total	100.0	100.0

Septic tanks of inadequate size may result in an insufficient holding time for solid wastes, which then pass through and block drain lines (Alaska). Old delapidated plumbing with breaks and leaks also allows for the escape of sewage from sewerage lines and possible entry into a faulty water supply.

Water pressure clearly plays an important role in contamination. A system with a submerged inlet, as occurred in the outbreak in Mississippi in which the hose on the tub spigot and the water faucet in the laundry room were both below the water line, may remain clean so long as incoming water pressure is sufficient to insure adequate flow in the proper direction. If water pressure becomes negative, however, sewage entry into the water supply system may occur. In the Mississippi outbreak, the water supply was turned off, and later a clogged meter caused reduction in flow, in both situations activating the auxiliary pump and creating negative pressures in the water system. Such booster pumps are illegal when connected directly to a municipal water system.

Occasionally cross-connections or other sources of contamination are obvious but more often they are cryptic. Consequently, the use of fluorescein and other dyes or tracers is of paramount importance. In the outbreaks in Mississippi and Iowa, dye introduced into disposal lines appeared in the water supply. In Iowa the outbreak was remarkable because shigella was recovered from a water sample. This is distinctly unusual even in cases in which coliform counts are exceedingly high, and again points to the importance of indirect techniques such as the use of recoverable dyes or chemical tracers in studying a suspect water supply.

In investigating an outbreak of shigellosis in which epidemiologic evidence points to water as the common source of exposure, the "apparent" adequacy of the plumbing should not deter the investigator from giving credence to his suspicions. Waterborne epidemics have involved otherwise model systems, and the multitudinous and often novel ways in which shigella and other infectious organisms are sometimes introduced makes any water system suspect if such suspicion is warranted by epidemiologic evidence. A sanitarian should be invited to participate in the investigation as soon as water is implicated, to collect water samples before superchlorination and to outline the appropriate studies before temporary purification measures are undertaken by concerned local officials, which may suppress contamination for a period of time and hinder diagnostic work, but which will rarely, if ever, remove the hazard.

References:

1. Craun GF, McCabe LJ: Review of the causes of waterborne disease outbreaks. Water Technology p 74, 1973
2. Ross AI, Gillespie EH: An outbreak of waterborne gastroenteritis and sonnei dysentery. Monthly Bull Great Britain Ministry Health and Public Health Lab Svc 11:36, 1952
3. Renteln HA, Hinman AR: A waterborne epidemic of gastroenteritis in Madera, California. Am J Epidemiol 86:16, 1967
4. Center for Disease Control: Morbidity and Mortality Weekly Rep 20(42):389, 1971
5. Center for Disease Control: Morbidity and Mortality Weekly Rep 21(6):49, 1972
6. Rice PA, Wells JG, Wood BT, et al: Outbreak of Shigella sonnei gastroenteritis in a summer camp, Bay St. Louis, Mississippi. Epid Aid memo No. EPI-72-3-2, 10 Feb 1972
7. Center for Disease Control: Morbidity and Mortality Weekly Rep 22(3):22, 1973
8. McCabe LJ, Seymour JM, Lee RD, et al: Survey of community water supply systems. J Am Water Works Assoc 62:670, 1970
9. Drachman RH, Payne FJ, Jenkins AA, et al: An outbreak of waterborne shigella gastroenteritis. Am J Hyg 72:321, 1960
10. Taylor A, Craun GF, Faich GA, et al: Outbreaks of waterborne diseases in the United States 1961-1970. J Infect Dis 125:329-331, 1972
11. Green CA, MacLeod MC: Explosive epidemic of Sonne dysentery. Brit Med J 2:259, 1943

IV. REPORTS FROM THE STATES

A. Shigellosis in 2 Neonates, Detroit, Michigan

Reported by Theodore Thompson, M.D., EIS Officer, Michigan Department of Public Health, members of the staff of the Oakland County Health Department, and members of the staffs of the Detroit hospitals in which these cases occurred.

Case 1

On July 15, 1972, a 1-day-old girl at a hospital in Detroit, Michigan, developed milk diarrhea. She was discharged on July 18, but was readmitted on July 21 because of fever and continuing diarrhea. There was no history of vomiting or blood in the stools. On further investigation, the mother was found to have had diarrhea at the time of delivery. Both mother and baby had S. sonnei isolated from their stools. Mother and child both recovered with symptomatic treatment only, and cultures from both 1 month later were negative.

Case 2

On July 15, a male infant at another hospital in Detroit, Michigan, developed bloody diarrhea and fever 1 day after birth. The infant was treated with ampicillin and fever abated rapidly although mild diarrhea persisted until ampicillin was discontinued 2 weeks later. Epidemiologic investigation disclosed that the child's mother, grandmother, and another sibling had had diarrhea beginning during the spring of 1972. The mother in particular had diarrhea beginning in the 6th month of pregnancy which continued for several weeks and subsided spontaneously. The mother experienced recrudescence of diarrhea around the time of delivery, and a culture obtained from the mother in the peri-natal period grew S. sonnei. The hospital at which the infant was born delivers an average of 150 babies monthly. No other cases of shigellosis were noted. Two cases of diarrhea in infants had occurred, one 3 weeks and another 1 week before the case in question, both infants were cultured and were negative for enteric pathogens.

Editorial Comment: The potential hazard of a neonate with shigellosis in a newborn nursery need not be stressed. The hospitals involved in these 2 cases isolated the infants as soon as they became symptomatic and doubtless prevented spread to other babies. In both cases the time between birth and illness was too short for infection to have likely occurred by secondary spread from someone in the nursery. Coupled with coincident infection in each of their mothers it seems probable that despite seemingly adequate pre-partum preparation and post-partum infant care, the neonates were infected at the time of delivery.

B. Shigella sonnei in 2 Summer Camps

Reported by Helen Sigurdson, R.N., Oneida County Health Department; Janet Agger, R.N., and Harvey Kelotz, Sanitarian, District 7, Rhinelander; H. Grant Skinner, M.D., M.P.H., State Epidemiologist, Wisconsin Division of Health and Social Services; Jane Nesbit, R.N., Pine Tree Crippled Children's Camp, Rome, Maine, Dean Fisher, M.D., Commissioner of Health and Welfare, State of Maine, and Timothy Townsend, M.D., EIS Officer, Maine Department of Health and Welfare.

Outbreak 1

On July 17, 1972, 3 children in a Wisconsin summer camp experienced diarrhea. Over the next several weeks, 60 of the 96 campers (63%) had diarrheal illnesses; 11 were hospitalized. S. sonnei was isolated from 9 of the hospitalized patients.

Epidemiologic investigation of the camp disclosed unsanitary conditions in kitchen and toilet areas. The swimming area had a coliform count of 3,760 per ml on August 1, 20 per 100 ml on August 9, and 60 per 100 ml on August 11. Dye introduced into the camp sewerage system, however, did not appear in the swimming area.

Outbreak 2

In July 1972, 37 of 180 campers and counselors (21%) at a summer camp for crippled children in Maine became ill with diarrhea, occasionally bloody. S. sonnei was isolated from stool specimens taken from 6 of the infected individuals.

Investigation revealed that the index case was a 17-year-old counselor who had onset of symptoms on the 3rd day of camp. Prior to the start of the camp season, he had worked at a nursing home in Maine where diarrhea was known to be endemic. The initial cases occurred in his cabin and all subsequent cases, except for 2, occurred in 4 cabins nearby. Approximately 2/3 of the campers were incontinent because of neurological disability and the counselors performed many hygienic duties for them, such as assisting with bathing and changing diapers. Examination of kitchen and toilet facilities, water supply, and sewerage system disclosed no sanitary inadequacies.

Editorial Comment: Summer camps represent the same kind of "closed community" circumstances as boarding schools and mental institutions which have long played a prominent role in the epidemiology of shigellosis. Because of frequent interpersonal contact, participation of the group in the same activities, and common food sources and preparation, both person-to-person and common source epidemics may occur. In the Wisconsin epidemic, the relatively high attack rate (63%) over a short period of time suggested that a common source outbreak occurred, and epidemiologic investigation incriminated the camp swimming area as a possible source of contamination, although the relationship of illness to frequency of swimming was not determined. The fact that shigella was not recovered from the water is not surprising as the actual isolation of this organism from an epidemiologically incriminated water supply is unusual. The person-to-person transmission in the Maine outbreak was probably spread by counselors who handled diapers and other frequently soiled articles in the course of caring for their disabled campers. It is of interest, however, that it was a counselor without physical disability who first introduced the illness into the community.

V. INTERNATIONAL NOTES

A. A Skin Test for Detecting Carriers of Shigellae

In the Soviet Union for several years an extract of shigellae has been used as a skin test to detect cases and carriers of shigellosis. The author of this series of papers from the Russian literature, E. N. Belan of the Pavlov Institute in Leningrad, used an intradermal test with a bacterial extract of shigellae ("dysenterin") in a variety of clinical situations to assess its usefulness. In 1968,¹ he showed that in a majority of patients with a positive reaction to dysenterin, shigellae could be recovered from their stools, despite the absence of symptoms in many cases. In children who went on to develop clinical illness, the skin test tended to be positive up to several days before the development of symptoms.

In a study of 1,015 children in 10 preschool "collectives"² (equivalent to our day-care centers) children were skin tested at 3-4 month intervals over a 14-month period. In patients with positive skin reactions, dysentery occurred 2-5 times more frequently than in patients with negative skin tests. Moreover, asymptomatic infections, as determined by culture studies, were present in half those with positive skin tests, whereas, asymptomatic carriage of the organism was unusual in children with negative skin tests. In another study of 571 people living in areas involved in 3 epidemics of shigellosis,³ 24.3% of those examined during the epidemics had positive skin tests. Of those who lived in affected households, 45% had positive skin tests, whereas 7-19% of those living in non-affected households had positive tests. As in the preschool study, bacteriologic examination showed that asymptomatic carriage of shigella occurred chiefly in persons with positive skin reactions. In 23% of those with positive skin tests, shigellae were isolated from the stool, compared with 3% of those with negative skin tests. Similar results were obtained in an examination of food handlers.⁴

Editorial Comment: The data suggest that skin testing may be useful in the identification of asymptomatic carriers of shigella. The dysenterin preparation is a combined sonnei-flexneri extract and as such could be used in outbreaks caused by either species. Presumably, only asymptomatic individuals would be tested, and of those, the positive reactors would subsequently undergo intensive bacteriologic examination to determine who were carriers of shigellae. A positive skin test reaction does not identify an individual case of shigellosis, but rather indicates which individuals are most likely to be carriers. The practical application of this technique in an epidemiologic investigation is limited, since the population investigated would have to be studied several times. First they would have to be tested, then the skin test would have to be interpreted (the skin test is read 18-24 hours after administration), then individuals would be cultured if warranted by the skin test, and finally treated as indicated by culture results. The advantage would be that a significant number of people who would otherwise be cultured could be eliminated by a negative skin test, but the difficulty of reaching a large number of people on repeated occasions during an outbreak might make it more practical to culture everyone. Philosophically, one wonders about injecting an antigenic material, the potential hazards of which are not fully known, when an innocuous and definitive test such as a rectal swab culture is a readily available alternative. Further refinements of skin test sensitivity to the point that a positive reaction positively identified an individual case, would markedly improve the potential usefulness of this tool.

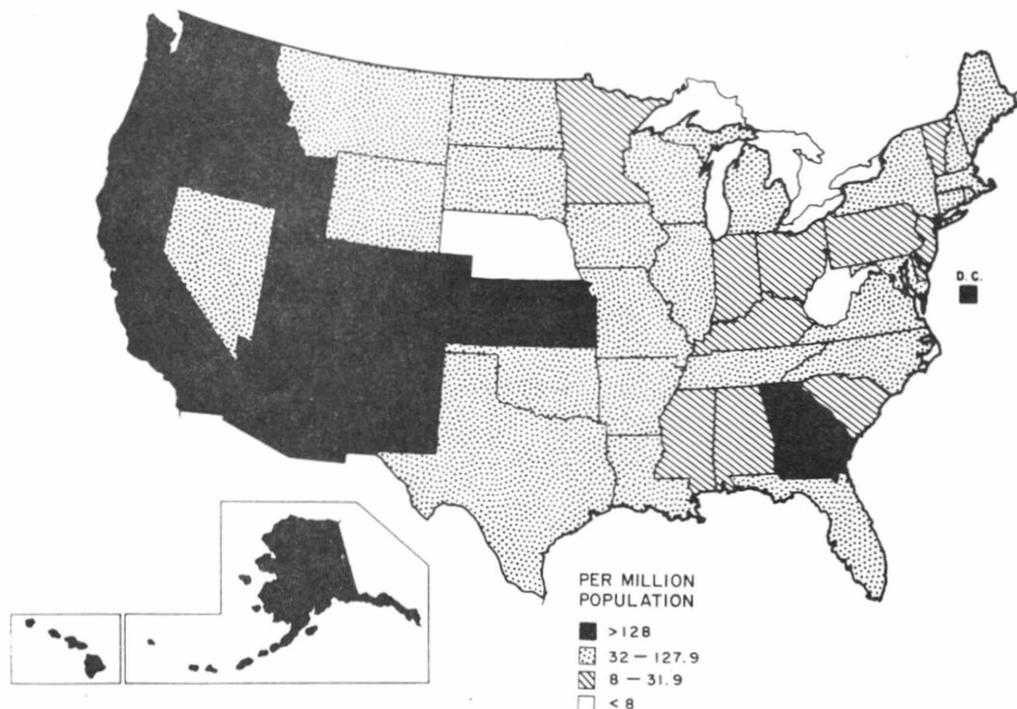
References:

1. Belan EN: Detection of patients with formes frustes of dysentery in children's collective bodies by an intradermal test with dysenterin. *J Microbiol Epidemiol Immunobiol (Moscow)* 6:76, 1968
2. Belan EN: Significance of skin allergic test for detection of children suffering from dysentery in organized collective bodies. *J Microbiol Epidemiol Immunobiol (Moscow)* 7:42, 1969
3. Belan EN: Significance of skin allergic test for detection of patients with formes frustes of dysentery during an outbreak of infection among the population. *J Microbiol Epidemiol Immunobiol (Moscow)* 10:47, 1969
4. Belan EN: Epidemiological significance of skin allergic test with dysenterin. *J Microbiol Epidemiol Immunobiol (Moscow)* 4:47, 1970

VI. SUMMARY FOR 1972*

In 1972, a total of 13,752 isolations of shigella was reported to CDC. This was an increase of 5.9% over the 12,988 isolations reported in 1971. Utilizing the population estimates for July 1, 1972, the overall United States attack rate was 66.0 reported isolations per million population in 1972, compared to 63.6 and 54.0 reported isolations in 1971 and 1970, respectively. Attack rates by state are depicted in Figure 4. California independently reported 4,359 shigella isolates in 1972.

Figure 4 ATTACK RATES OF SHIGELLOSIS BY STATE, 1972



The age and sex distribution of individuals from whom shigella was isolated in 1972 is presented in Table VI. Children 1-4 years of age were at greatest risk with an attack rate of 255.6 per million during 1972.

The seasonal distribution persisted; the greatest number of isolates have been reported each autumn (Figure 2).

The 6 most frequently reported serotypes during 1972 are shown in Table 6.

Table 6
Six Most Frequently Reported Shigella Serotypes During 1972

Rank	Serotype	Reported Number	Calculated Number	Calculated Percent	Rank in 1971
1	<u>S. sonnei</u>	10,842	10,935	79.5	1
2	<u>S. flexneri</u> 2a	483	936	6.8	2
3	<u>S. flexneri</u> 3a	293	547	4.0	3
4	<u>S. flexneri</u> 6	349	443	3.2	5
5	<u>S. flexneri</u> 4a	144	221	1.6	4
6	<u>S. flexneri</u> 2b	103	200	1.4	6

* This summary is based upon preliminary reports compiled quarterly through December 31, 1972. Additions or corrections will be published in a subsequent report.

Table VII shows the relative frequency of all shigella serotypes reported in 1972. The trend toward an increasing proportion of all isolates being S. sonnei continued as it has since the 4th quarter of 1966. In 1972, 78.8% of all reported shigella isolations were S. sonnei versus 76.2 in 1971 and 71.8 in 1970. Concomitantly, S. flexneri has progressively decreased in proportion of total isolations.

TABLE 1-A (Continued)
 SHIGELLA SEROTYPES ISOLATED FROM HUMANS
 THIRD QUARTER, 1972

SEROTYPE	SOUTHEAST														SOUTHWEST				OTHER				PREVIOUS QUARTER		TOTAL									
	ALA	ARK	FLA	GA	LA	MISS	NC	SC	TENN	SOUTHEAST TOTAL	ARIZ	NM	OKLA	TEX	SOUTHWEST TOTAL	SOUTH TOTAL	ALASKA	CALIF	HAWAII	VIRGIN ISLANDS	OTHER TOTAL	TOTAL	PERCENT OF TOTAL	TOTAL		PERCENT OF TOTAL								
	0	0	0	1	2	0	0	3	0	6	2	1	1	4	8	14	0	0	0	0	0	20	0.5	15		0.4	Total							
<i>S. flexneri</i> Unspec	12	1				11		1	25		8		1	9	34						0	162	4.1	145	4.1	1	18	0.5	13	0.4	1	Unspec		
		2		1					3		11			7	9	14	1				1	15	0.4	15	0.4	1A						1A		
											2			1	1	2					0	4	0.1	14	0.4	1B						1B		
	5	1	12	19					46		31			1	32	78					0	124	3.2	46	1.3	2	Unspec					2	Unspec	
		1								15	18	35		4	17	56	74				5	173	4.4	118	3.4	2A						2A		
										3	3	4		17	21	24	1				1	38	1.0	27	0.8	2B						2B		
	1	2	6	2						12	12	10		4	14	28					0	89	2.3	24	0.7	3	Unspec						3	Unspec
										7	14	10		1	15	15					0	62	1.6	66	1.9	3A						3A		
										0	0	14		1	0	3					0	15	0.4	3	0.1	3B						3B		
		1								3	3				0	3					0	4	0.1	6	0.2	3C						3C		
									1	1	9			9	10	1				1	15	0.4	6	0.2	4	Unspec						4	Unspec	
									2	6	15		1	12	28	34				0	56	1.4	34	1.0	4A							4A		
									0	0	1			1	2	2				0	6	0.2	13	0.4	4B							4B		
									0	1	1			1	2	2				0	4	0.1	6	0.2	5							5		
									0	1	1			1	2	2				0	4	0.1	6	0.2	5							5		
1		18							22	23	5		12	40	62					1	85	2.2	99	2.8	6							6		
7	19	39	23	19	11	10	1	25	154	105	80	5	75	265	419		3	0	7	0	10	870	22.2	637	18.1	Total						Total		
	2								2					0	2					0	6	0.2	2	0.1	<i>S. boydii</i>							<i>S. boydii</i>		
									0					2	2					0	6	0.2	12	0.3	2								Unspec	
0	2	0	0	0	0	0	0	0	2	4	0	0	3	7	9		0	0	0	0	0	6	0.2	2	0.1	Total						Total		
17	59	137	174	120	8	84	4	93	696	62	155	27	177	421	1,117		57		49		106	2,997	76.5	2,782	79.2	<i>S. sonnei</i>						<i>S. sonnei</i>		
																						14	0.4	61	1.7	Unknown						Unknown		
24	80	176	199	141	20	94	8	118	860	173	236	33	259	701	1,561		60	0	56	0	116	3,919		3,514		TOTAL						TOTAL		

TABLE I-B (Continued)
SHIGELLA SEROTYPES ISOLATED FROM HUMANS
FOURTH QUARTER, 1972

SOUTHEAST											SOUTHWEST					OTHER					PREVIOUS QUARTER		SEROTYPE		
ALA	ARK	FLA	GA	LA	MISS	NC	SC	TENN	SOUTHEAST TOTAL	ARIZ	NM	OKLA	TEX	SOUTHWEST TOTAL	SOUTH TOTAL	ALASKA	CALIF	HAWAII	VIRGIN ISLANDS	OTHER TOTAL	TOTAL	PERCENT OF TOTAL		TOTAL	PERCENT OF TOTAL
		1					1		1			1		0	1					0	2	0.1	1	0.0	<i>S. dysenteriae</i>
									1			1		1	2					0	2	0.1	9	0.2	Unspec
									0	1				1	1					0	2	0.1	4	0.1	1
									0	8				8	8					0	8	0.2	2	0.1	2
									0		1		1	2	2					0	4	0.1	2	0.1	3
									0											0					4
0	0	1	0	0	0	0	1	0	2	9	1	1	1	12	14	0	0	0	0	0	18	0.5	20	0.5	Total
1	5	1			13	3	1	1	25		1		2	27	1					1	108	3.0	162	4.1	<i>S. flexneri</i>
		1	4						5		7		7	12						0	19	0.5	18	0.5	Unspec
								1	1				4	4	5					0	10	0.3	15	0.4	1A
									0	5			8	13	13					0	16	0.4	4	0.1	1B
4		4	16			14			38		38	1	39	77						0	96	2.7	124	3.2	2 Unspec
				4				6	10	24		1	23	48	58			6		6	111	3.1	173	4.4	2A
				1				2	3	2			12	14	17					0	22	0.6	38	1.0	2B
		4	1	1				6	3	7			10	16	16			2		2	21	0.6	89	2.3	3 Unspec
				3				2	5	29			7	36	41					0	63	1.8	62	1.6	3A
								0	3				3	3						0	5	0.1	15	0.4	3B
								0	1				1	1						0	4	0.1	4	0.1	3C
								0			4		1	5	5					0	8	0.2	15	0.4	4 Unspec
		1						1	1		1	1	1	3	4			1		1	15	0.4	56	1.4	4A
								0					0	0						0	3	0.1	6	0.2	4B
			1					1	2		1	3	6	7						0	11	0.3	4	0.1	5
1		24	2	1		1		4	33	17	19		7	43	76					0	94	2.6	85	2.2	6
								0					0	0						0	1	0.0			Variant Y
6	5	35	24	10	13	18	1	16	128	87	76	4	67	234	362	1	0	9	0	10	607	17.0	870	22.2	Total
					1				1					0	1					0	5	0.1	6	0.2	<i>S. boydii</i>
									0	1				1	1					0	1	0.0			Unspec
									0					0	0					0	1	0.0	6	0.2	1
									0	5	2		7	7						0	10	0.3	2	0.1	2
									0				0	0						0	1	0.0			4
									0	2			2	2						0	2	0.1			5
									0	1			1	1						0	1	0.0	2	0.1	7
									0	1			1	1						0	1	0.0	2	0.1	10
									0	1			1	1						0	1	0.0	2	0.1	14
0	0	0	0	0	1	0	0	0	1	10	2	0	0	12	13	0	0	0	0	0	22	0.6	18	0.5	Total
21	25	135	237	148	12	75	9	106	768	66	120	83	138	407	1,175	18		12		30	2,910	81.6	2,997	76.5	<i>S. sonnei</i>
						2		2							2						10	0.3	14	0.4	Unknown
27	30	171	261	158	26	95	11	122	901	172	199	88	206	665	1,566	19	0	21	0	40	3,567		3,919		TOTAL

Table II

Age and Sex Distribution of Individuals Infected With
Shigella in the United States, 2nd Half, 1972

<u>Age (Years)</u>	<u>Male</u>	<u>Female</u>	<u>Unknown</u>	<u>Total</u>	<u>Percent</u>	<u>Cumulative Percent</u>	<u>Number of Reported Isolations/ Million Population*</u>
<1	158	137	4	299	5.9	5.9	85.8
1-4	1,036	989	4	2,029	40.3	46.2	148.4
5-9	563	548		1,111	22.1	68.3	55.7
10-19	291	276	2	569	11.3	79.6	14.3
20-29	198	373		571	11.3	90.9	19.1
30-39	98	117	2	217	4.3	95.2	9.6
40-49	42	57		99	2.0	97.2	4.1
50-59	23	39		62	1.2	98.4	7.2
60-69	22	20		42	.8	99.2	2.7
70-79	5	18	1	24	.5	99.7	2.6
80 +	3	6		9	.0	99.7	2.4
Subtotal	2,439	2,580	13	5,032			
Child (unspec)	21	10	1	32			
Adult (unspec)	13	22		35			
Unknown	1,157	1,183	47	2,387			
TOTAL	3,630	3,795	61	7,486			
Percent	48.9	51.1					

*Based on 1970 Census of Population, General Population Characteristics, United States Summary, Issued January 1972.

Table III

Relative Frequencies of Shigella Serotypes
Reported, 2nd Half, 1972

<u>Serotype</u>	<u>Reported</u>	<u>Calculated Number *</u>	<u>Calculated Percent*</u>	<u>Rank</u>
A. <u>S. dysenteriae</u>				
Unspecified	3			
1	11	12	.16	13
2	6	7	.09	16
3	10	11	.15	14
4	6	7	.09	16
9	2	2	.03	19
B. <u>S. flexneri</u>				
Unspecified	270			
1 unspecified	37			
1a	25	56	.75	7
1b	20	45	.60	8
2 unspecified	220			
2a	284	572	7.64	2
2b	60	121	1.62	5
3 unspecified	110			
3a	125	264	3.53	3
3b	20	42	.56	9
3c	8	17	.23	11
4 unspecified	23			
4a	71	112	1.50	6
4b	9	14	.19	12
5	15	18	.24	10
6	179	220	2.94	4
Variant Y	1	1	.01	20
C. <u>S. boydii</u>				
Unspecified	11			
1	1	1	.01	20
2	7	10	.13	15
4	12	17	.23	11
5	1	1	.01	20
7	2	3	.04	18
10	3	4	.05	17
14	3	4	.05	17
D. <u>S. sonnei</u>	5,907	5,926	79.15	1
Unknown	24			
TOTAL	7,486	7,487		

*Calculated number is derived by distributing the unspecified isolations in each group to their subgroup in the same proportion as the distribution of the specified isolations of that group.

Table IV

Shigella Serotypes from Mental Institutions
Number of Isolations by State, 2nd Half, 1972

State	dysenteriae 2	flexneri (unspecified)	flexneri 1 (unspecified)	flexneri 2 (unspecified)	flexneri 2a	flexneri 2b	flexneri 3 (unspecified)	flexneri 3a	flexneri 4a	flexneri 5	flexneri 6	sonnei	Total
Florida	0	0	0	0	0	0	1	0	1	0	35	7	44
Georgia	0	0	1	5	0	0	0	0	0	0	0	0	6
Idaho	0	0	0	0	0	0	0	0	0	0	0	6	6
Illinois	0	0	0	0	12	4	0	28	0	5	2	16	67
Iowa	0	0	0	0	0	0	0	0	0	0	0	3	3
Kansas	0	0	0	0	0	0	0	0	0	0	1	9	10
Louisiana	0	0	0	0	0	0	0	0	0	0	0	14	14
Maine	0	0	0	0	0	0	0	0	0	0	0	2	2
Maryland	0	0	0	0	0	0	0	0	0	0	0	1	1
Massachusetts	0	2	0	0	59	0	0	0	0	0	0	0	61
Michigan	0	1	0	2	0	0	9	0	0	0	0	14	26
Minnesota	0	0	0	0	2	0	0	0	0	0	0	0	2
New Jersey	0	0	0	0	0	0	47	0	0	0	0	1	48
New York	0	1	0	0	0	0	0	0	0	0	0	0	1
North Carolina	0	0	0	2	0	0	0	0	0	0	0	16	18
Oregon	0	0	0	0	0	0	0	0	0	0	0	17	17
Pennsylvania	0	0	0	0	0	0	0	0	0	0	0	15	15
South Carolina	3	1	0	0	0	0	0	0	0	0	0	0	4
Utah	0	0	0	9	0	0	0	0	0	0	0	4	13
Wisconsin	0	0	0	0	0	0	0	0	0	0	0	9	9
TOTAL	3	5	1	18	73	4	57	28	1	5	38	134	367

Table V

Sources of Reported Isolations of Shigella
By Residence at Time of Onset
2nd Half, 1972

Source	Jul	Aug	Sep	Oct	Nov	Dec	Total	Percent of Subtotal	Percent of Total
Mental institutions	47	93	96	27	62	31	367	10.3	
Indian reservations	18	24	20	23	14	0	103	2.9	
Other residencies	376	736	481	423	572	477	3095	86.8	
Subtotal	441	853	597	473	648	508	3565		47.6
Residencies unknown	540	761	634	673	740	485	3921		52.4
TOTAL	981	1614	1231	1146	1388	993	7486		

Table VI

Age and Sex Distribution of Persons Infected With
Shigella in the United States, 1972

<u>Age (Years)</u>	<u>Male</u>	<u>Female</u>	<u>Unknown</u>	<u>Total</u>	<u>Percent</u>	<u>Cumulative Percent</u>	<u>Number of Reported Isolations/ Million Population*</u>
<1	279	251	6	536	5.9	5.9	160.4
1-4	1834	1711	8	3553	39.1	45.0	255.6
5-9	1033	1050	2	2085	22.9	67.9	111.5
10-19	590	506	2	1098	12.1	80.0	26.9
20-29	318	670		988	10.9	90.9	30.1
30-39	172	240	2	414	4.6	95.5	17.8
40-49	81	103		184	2.0	97.5	7.8
50-59	39	65		104	1.1	98.6	4.8
60-69	31	35		66	.7	99.3	4.1
70-79	10	32	1	43	.5	99.8	4.5
80 +	3	13		16	.2	100.0	3.8
Subtotal	4390	4676	21	9087			
Child (unspec)	37	19	3	59			
Adult (unspec)	18	39	1	58			
Unknown	2247	2230	71	4548			
TOTAL	6692	6964	96	13752			
Percent	49.0	51.0					

*Based on 1970 Census of Population, General Population Characteristics, United States Summary, Issued January 1972

Table VII

Relative Frequencies of Shigella Serotypes Reported, 1972

<u>Serotype</u>	<u>Number Reported</u>	<u>Calculated Number</u>	<u>Calculated Percent</u>	<u>Rank</u>
A. <u>S. dysenteriae</u>				
Unspecified	5			
1	15	16	.12	15
2	19	21	.15	13
3	13	14	.10	16
4	8	9	.07	17
9	3	3	.02	20
B. <u>S. flexneri</u>				
Unspecified	543			
1 unspecified	60			
1a	55	115	.84	7
1b	37	78	.57	8
2 unspecified	308			
2a	483	936	6.80	2
2b	103	200	1.45	6
3 unspecified	158			
3a	293	547	3.98	3
3b	26	49	.36	9
3c	17	32	.23	11
4 unspecified	35			
4a	144	221	1.61	5
4b	24	37	.27	10
5	24	30	.22	12
6	349	443	3.22	4
Variant Y	3	4	.03	19
C. <u>S. boydii</u>				
Unspecified	15			
1	2	3	.02	20
2	23	30	.22	12
4	13	17	.12	14
5	2	3	.02	20
6	1	1	.01	21
7	3	4	.03	19
9	1	1	.01	21
10	3	4	.03	19
14	5	6	.04	18
D. <u>S. sonnei</u>	10,842	10,935	79.48	1
Unknown	117			
TOTAL	13,752	13,759		

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The State Epidemiologists are the key to all disease surveillance activities. They are responsible for collecting, interpreting, and transmitting data and epidemiologic information from their individual States; their contributions to this report are gratefully acknowledged. In addition, valuable contributions are made by State Laboratory Directors; we are indebted to them for their valuable support.

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