Epidemic Spread of *Shigella sonnei* Shigellosis and Evidence for Development of Immunity among Children Attending Day-Care Centers in a Communal Settlement (Kibbutz)

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An investigation of two Shigella sonnei shigellosis outbreaks that occurred in a communal settlement indicated that the transmission of the pathogen was restricted to day-care classes and secondary infection of family members was minimal. Development of serotype-specific immunity following S. sonnei infection was observed among infected children.

Two outbreaks of *Shigella sonnei* shigellosis that occurred among children in a communal settlement (kibbutz) were investigated. Ninety-one children aged 0 to 6 years lived in the community at the time of the investigation. All children were born in Israel, mostly in the settlement.

The children spend from 6:30 a.m. to 4:00 p.m. in small groups with assigned caretakers. The usual weaning age from diapers is 2 years. Staff members are involved both in diapering and in serving of food. No standard hand-washing protocol was in place for diapering infants. The ratio of caretakers to children is 1:6. The number of persons per room in families with children is 1.2 to 1.5. The sanitary conditions in the community are good. Only 4.4% of the fathers and none of the mothers had fewer than 12 years of schooling.

There was no use of other medical services without the approval of the medical team of the community during the investigation. Routinely, fresh stool samples were transported to the microbiology laboratory within 1 to 2 h after collection. Stools were examined by standard procedures for the detection of *Shigella* spp., *Salmonella* spp., and *Campylobacter* spp. (4, 5a). Drug resistance tests on the isolates were performed by the Kirby-Bauer disk diffusion method (1) (Difco, Detroit, Mich.).

A phage-typing scheme for S. sonnei was used to define the phage types involved in the present outbreak (8, 9). Briefly, this phage-typing scheme is based on the activity of seven phage preparations, grouped so that the first four phages identify 16 phage groups (A to P) and the other three identify 8 types (1 to 8) among each of the phage groups. Thus, in total 128 phage types can be defined. The scheme is based on typing rough forms of S. sonnei. However, some isolates may develop smooth forms. Previous experience has shown that a correlation exists between the phage types of rough and smooth forms of the same strain. e.g., The rare smooth forms of strains belonging to phage type A1 are always of phage type D7. Therefore, when a culture of phage type D7 is found in an outbreak caused by S. sonnei of phage type A1, the case may be considered as belonging to the outbreak. In this case the phage type is designated D7 (A1). Restriction fragment length polymorphism (RFLP) analysis of eight S. sonnei isolates from 20 cases was performed as previously described (13), after digestion of the chromosomal DNA with five restriction enzymes (*Eco*RI, *Bam*HI, *Sma*I, *Kpn*I, and *Pvu*II) (International Biotechnologies Inc., New Haven, Conn.). This RFLP analysis protocol emerged as a sensitive tool to differentiate among *S. sonnei* strains isolated in outbreaks of shigellosis (13).

A patient with *S. sonnei* shigellosis was defined as a person with diarrhea (at least three discharges per day and change in liquidity of fecal discharges) plus a stool culture positive for *S. sonnei*.

The first outbreak (five cases) occurred during 15 December 1991 to 19 January 1992. The second outbreak (22 cases) occurred between 12 April and 25 July 1992 (Fig. 1). The attack rates of *S. sonnei* infections in day-care classes (DCCs) is presented in Table 1. All cases appeared in children aged 17 months to 6 years, with the exception of two cases that occurred as a single case in the DCC: one in a 10-year-old sister of one of the children and one in a 16-year-old adolescent. Neither the parents nor the caretakers presented a clinical picture of dysentery.

The first cluster of *S. sonnei* shigellosis was entirely restricted to DCC D. The second outbreak affected more DCCs, including DCC D. None of the four children from DCC D who suffered from the disease in the first outbreak had recurrent shigellosis during the second outbreak. The five new cases of *S. sonnei* shigellosis in DCC D were among the eight children not infected during the first outbreak.

All isolates were resistant to ampicillin and trimethoprimsulfamethoxazole and susceptible to furadantin, polymyxin B, chloramphenicol, nalidixic acid, ciprofloxacin, and cefuroxime. All the isolates of *S. sonnei* except two were also susceptible to tetracycline. The tetracycline MIC for these two isolates was >32 μ g/ml.

Three isolates of *S. sonnei* were defined in the five cases that occurred in the first outbreak, and all were of phage type C4 (Fig. 1). From 17 cases of shigellosis of the second outbreak, 28 isolates of *S. sonnei* were obtained. Eighteen isolates from 11 cases were of phage type A1, and ten isolates from 6 cases were of phage type C1 (Fig. 1). As shown in Table 1, the phage types were clustered mostly among children from the same DCC. In two children, however, the phage type differed from the predominant phage type detected in the same DCC. In these children the infection could be ascribed to contact with a brother and a close friend who were sick before. The same phage type, C1, was detected in these two children and their contacts. Phage types of isolates that were obtained during a

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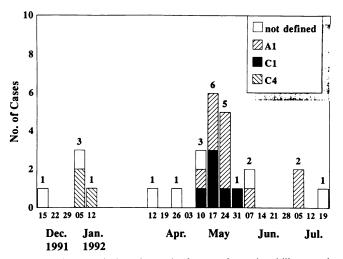


FIG. 1. S. sonnei isolates by week of onset of associated illness and phage types, December 1991 through July 1992. Numbers on the x axis indicate the date of the first day of the week.

relapse or during the phase of asymptomatic excretion of *S. sonnei* were identical to the phage types obtained before.

The strains analyzed by RFLP belonged to all the phage types (A1, C1, and C4) found in the outbreaks and comprised also a strain resistant to tetracycline. A single pattern of RFLP was obtained. This pattern was different from the RFLP patterns of isolates obtained from *S. sonnei* infections occurring in a neighboring settlement and in military units.

Microbiological testing of the community water supply did not reveal the presence of coliforms above the permitted number. *S. sonnei* was not isolated from a homogenate of flies collected from the playgrounds of the day-care center during May 1992.

The two outbreaks were probably due to the existence of a large reservoir of *S. sonnei* in the neighborhood communities and contact with the population in these communities by members of the studied community.

Documentation of two phage types in the second outbreak suggests that there were two sources of infection, that the original source of infection carried at least two *S. sonnei* strains, or that phage types A1 and C1 differ only slightly genetically and that such genetic variation occurred during this outbreak. The single RFLP pattern of *S. sonnei* isolates might be explained by the fact that this type of analysis examined variations between strains only in a conservative region of the DNA encoding rRNA. This region was identical among the

 TABLE 1. Attack rates and phage types of S. sonnei infection in DCCs in a communal settlement

Age (mo.)	DCC (no. of children)	Outbreak 1		Outbreak 2	
		No. (%) of ill children	Phage type (no. of strains typed)	No. (%) of ill children	Phage type(s) (no. of strains typed)
0-12	A (17)	0		0	
12-17	B (11)	0		0	
17 - 20	C (12)	0		5 (42)	C1 (4)
17-32	D (12)	4 (33)	C4 (2)	5 (42) ^a	A1(4);C1(1)
36–72	E (24)	0	. ,	3 (12)	A1 (1)
36–72	F (25)	0		8 (32)	A1(6);C1(1)

" None of the four children infected in outbreak 1 was infected in outbreak 2.

strains isolated in the settlement but differed from that in strains isolated outside the settlement.

The epidemic curve, the lack of cases among children in other DCCs, and the good quality of water and sanitary conditions in the community rule out the possibility of a common-source epidemic.

The transmission of S. sonnei was probably person to person, occurred because of poor hygiene practiced among children in DCCs (7, 11), and was facilitated by the low infectious dose of shigellas (3). Contamination of diaper-changing surfaces and the serving of food and changing of diapers by the same DCC staff might have contributed to the transmission of S. sonnei between the young children (6). The fact that children of the same family spent few hours together during the day, contrary to the long hours that they spent with their agemates, could account for the relatively low household transmission rate reported in this study compared with that in other studies (12). In spite of the fact that several studies have suggested that DCCs play an important role in community spread of shigellosis (10), it is assumed that the overall high level of sanitation prevented the transmission of Shigella infection outside the DCC.

It was suggested that clinical shigellosis confers protection against the homologous serotype (1, 2, 5). The duration of the protective effect is still not clear. The lack of recurrence of disease during the second outbreak among subjects who suffered from *S. sonnei* shigellosis in the first outbreak supports the assumption of the existence of serotype-specific natural immunity for at least 5 to 7 months following infection with *S. sonnei*.

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