A 1955 study demonstrates that the mere introduction of a highly virulent strain of poliomyelitis into a susceptible population is not enough to kindle a severe epidemic.

Poliomyelitis in Idaho After Use of Live Virus Vaccine

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FACTORS responsible for a poliomyelitis epidemic in the United States are not clear. Sporadic cases of poliomyelitis are reported every year and may be present at any season throughout the country. Epidemics, however, are geographically limited and are relatively infrequent. They have no apparent regularity but generally occur during the summer and early fall. It would appear that an epidemic should follow introduction of an especially virulent and invasive strain of poliovirus into a relatively susceptible population, but in an endemic area, it is not possible to establish the origin or introduction of such a strain.

An incident in Idaho during April 1955 gave an unparalleled opportunity to study a poliomyelitis outbreak. In that month two lots of commercially prepared vaccine were used to immunize 32,000 children in the school program of the State. After the first series of inoculations, poliomyelitis appeared in the vaccinated children, and the immunization program was terminated. Subsequently, viable poliovirus of all three types was shown to be present in the vaccine lots used (*1*). The introduction of viruses into a human population at a known time and the ease with which subsequent events could be followed created a favorable environment for study of the epidemic.

Vaccine containing live virus was given at a time when poliomyelitis did not constitute a problem in Idaho. The vaccination program was conducted at least 2 months prior to the season when an increase in incidence would have been expected on the basis of past experience. Thus, polioviruses were introduced into a relatively susceptible population at a season which experience had shown to be unfavorable for the natural occurrence of poliomyelitis epidemics.

Idaho's first recorded poliomyelitis epidemic was in 1947, when 371 cases were reported. Other epidemics occurred during 1949 (510 cases) and 1952 (253 cases). In a population of approximately 600,000 people, 1,884 cases were reported during the 8-year period from 1947 through 1954.

Our conclusions regarding conditions necessary for the occurrence of a poliomyelitis epi-

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demic are based primarily on data concerning the incidence of poliomyelitis in the recipients of two lots of vaccine used in Idaho, the infectivity of those individuals manifesting clinical disease, and the number of secondary cases that developed.

Methods

Attempts were made to obtain clinical and epidemiological data and selected specimens from all cases of poliomyelitis reported from the time of administration of the vaccine until January 1, 1956. Whenever possible, specimens were collected from family associates of patients with onset before October 1955. Blood specimens for serologic studies also were collected from 657 children in first and second grades 6 to 8 weeks after vaccination.

All specimens were examined by standard tissue-culture techniques in which epithelial cells from the kidneys of rhesus monkeys were used. Viruses were isolated in such cultures by inoculation of suspensions of certain tissues, sputum, or feces into bottles or tubes containing sheets of cells which had been incubated at 37° C. for 7 days. Agents producing typical cytopathogenic effects were identified by neutralization tests (*2*) with serums which had been produced at the Rocky Mountain Laboratory and used in a portion of the study conducted by the National Foundation for Infantile Paralysis during the evaluation studies in 1954. These examinations were made in the same laboratory that had conducted the evaluation study in Montana in 1954.

Samples of the two lots of vaccine used in Idaho were supplied by the Idaho Department of Public Health, district health department directors, practicing physicians, and, in the case of one lot, also by the manufacturer. Monkeys and special tissue-culture methods were used to detect virus in these vaccines (*1*).

The number of cases accepted for inclusion in the study and the criteria for their selection are given in table 1 and figures 1–7. All paralytic cases with clinical data compatible with a diagnosis of poliomyelitis have been included. In some cases, however, no specimens were submitted for examination. Nonparalytic cases were excluded unless virus was isolated from specimens collected from the patient or from a family contact.

Of 167 cases accepted as caused by type 1 poliovirus, 149 were paralytic and 18 were nonparalytic. Virus was isolated from 97 patients and from 12 family contacts. Eighteen persons from whom virus was isolated did not display evidence of paralysis, whereas 91 manifested some degree of paralysis. In 33 instances in which virus was not isolated, diagnosis was based on the presence of diagnostic titers of type 1 antibodies. In the majority of cases a rise in antibody titer was not demonstrated. These cases are included, however, since it is well known that the immunological response is already far advanced by the time

Table 1. Idaho poliomyelitis cases in 1955 probably due to type 1 virus

Relation to vaccine	Type of case	Virus isolation		Antibodies		No specimens received [2]	Total
		Patient	Family	Rise	No rise present [1]		
Poliomyelitis in vaccinated children.	Paralytic	7	4	3	2	1	17
	Nonparalytic	3	0	0	0	0	3
Poliomyelitis in contacts of vaccinated children.	Paralytic	37	6	0	5	4	52
	Nonparalytic	8	1	0	0	0	9
Poliomyelitis with no history of contact or onset after June 30.	Paralytic	36	1	0	23	20	80
	Nonparalytic	6	0	0	0	0	6
Total		97	12	3	30	25	167

[1] In all cases type 1 antibodies were present either alone or in conjunction with types 2 or 3 antibodies.
[2] These cases are included as probable type 1 poliomyelitis since there were so few isolations of type 2 or type 3 poliovirus from cases.

Figure 1. Poliomyelitis, type 1 and probable type 1, among children vaccinated on or after April 17, 1955, Idaho.

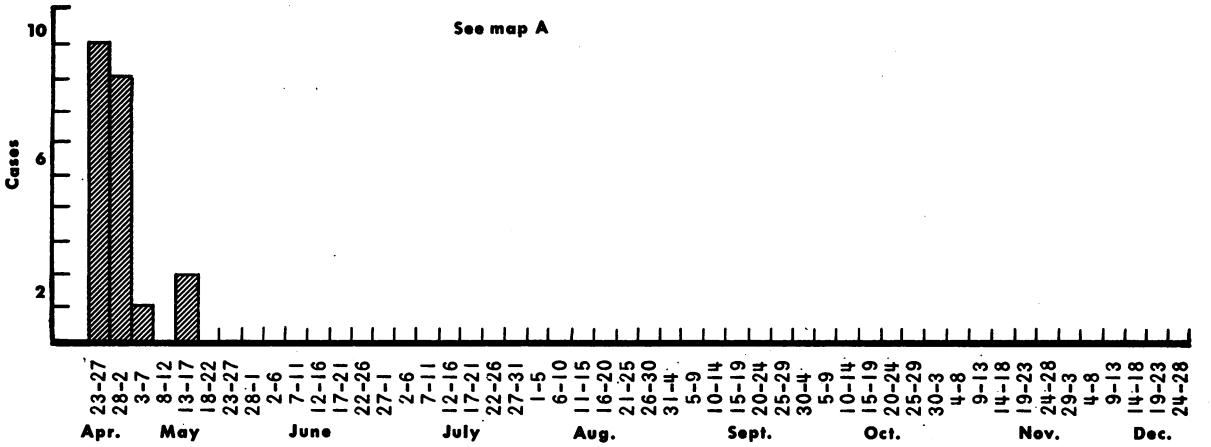
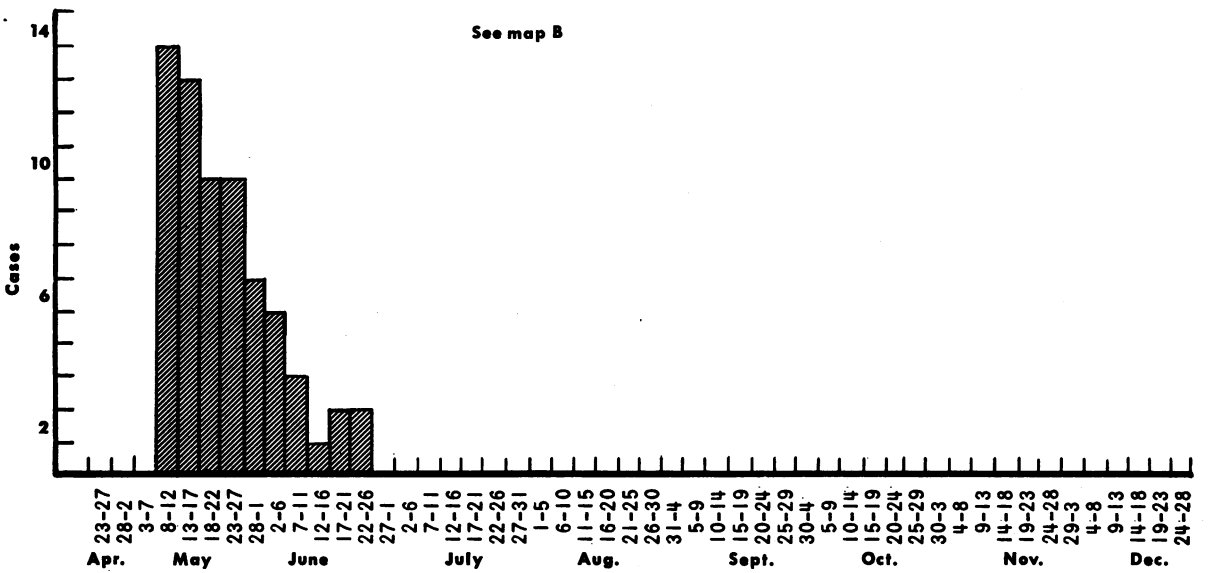
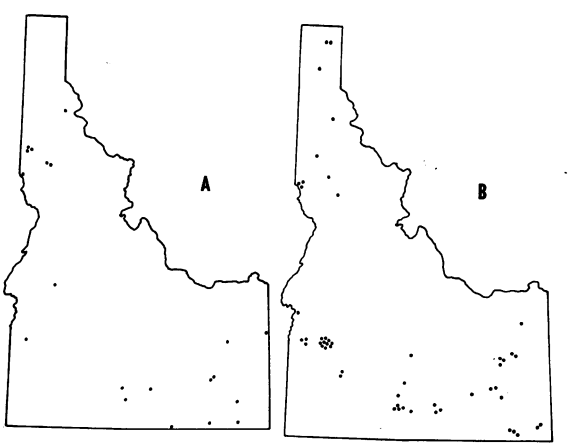


Figure 2. Poliomyelitis, type 1 and probable type 1, among contacts of children vaccinated on or after April 17, 1955, Idaho.





paralysis appears. Although no specimens were received from 25 paralytic patients, the majority of these cases were considered to be due to type 1 virus since so few type 2 and type 3 poliovirus were isolated from patients in Idaho during 1955.

Poliomyelitis occurred in 20 children who received vaccine, in 61 persons who were contacts of vaccinated children, and in 86 individuals who did not have a history of contact with vaccinated children or who had onset of illness during July or later. When onset was after July 1, 1955, the time lapse since vaccination

was so long that it appeared unreasonable to attribute the illness to contact with a vaccinated child.

Results

Epidemiological and clinical evidence showed a relation between the injection of poliomyelitis vaccines and the subsequent occurrence of poliomyelitis. Vaccination was begun April 17 and was practically completed within 5 days. Poliomyelitis was first reported in vaccinated children (fig. 1) in various scattered areas throughout the State (fig. 1, map A). These cases were followed by another group of cases (fig. 2, map B) limited to contacts of inoculated children. The geographic distribution of the two groups was similar. Although considerable effort was made to detect other types of poliovirus, only type 1 was isolated from these patients.

In 12 of 17 children with paralytic disease, the first evidence of paralysis was seen in the inoculated arm. In one child, the first signs, which developed on the 18th day after vaccination, were those of bulbar involvement and weakness of neck muscles. Of the 10 cases occurring within a week after administration of vaccine, 9 had onset of paralysis in the inoculated arm and 1 in a leg. In 5 of the 9, the disease progressed to bulbar poliomyelitis, and 1 child developed symptoms of severe encephalitis.

Initial involvement of an upper extremity with rapid progression to the opposite extremity and respiratory muscles was so unique clinically as to suggest that host response to poliovirus was influenced in some unusual manner. Equally striking was the lack of evidence of infection in members of patients' families when onset was less than 7 days after administration of vaccine.

Specimens were submitted from 31 family contacts (14 children) of the 7 children with onset on the fifth and sixth days after inoculation, but virus was not detected in any family contact nor was a rise in antibody titer demonstrated. Virus was first isolated from a family contact when the incubation period was 7 days. In two instances, virus was not recovered from family contacts when the incubation period was as long as 10 days. Usually, proliferation of poliovirus takes place in the upper and lower digestive tracts before the central nervous system becomes involved, and the person is eliminating virus before symptoms recognizable as poliomyelitis occur. Furthermore, members of the family usually are found to be carrying poliovirus in their intestinal tracts by the time the initial case of poliomyelitis is diagnosed. However, in the vaccinated children in Idaho, virus apparently did not reach the intestinal tract until after the appearance of signs pointing to disturbances of the central nervous system. This observation and the fact that poliovirus was not recovered from family contacts of seven patients indicated an unnatural spread of the virus.

Several observations suggested that live poliovirus was present in the two lots of vaccine used: the time relationship of cases to the administration of vaccine, the wide geographic distribution, the frequent occurrence of initial paralysis in the inoculated arm, and the absence of evidence of poliomyelitis infection in the families of patients in whom there was a short interval between inoculation of vaccine and onset. Therefore, attempts were made to determine the amount of such virus in these lots.

By using cortisone-treated monkeys, all three types of poliovirus were isolated from each lot of vaccine. The available amount of one lot was insufficient to permit repeated attempts to isolate virus, but sufficient vaccine of the second lot was available for such tests. With this lot, the maximum number of monkeys infected with types 1, 2, and 3 poliovirus, respectively, was 4 of 10, 3 of 10, and 2 of 7. The maximum number of inoculated monkeys paralyzed in any experiment was 4 of 7.

Treatment of poliovirus with 1:4,000 formaldehyde at 37° C. for several days must produce many complex changes in the viruses, and any attempt, with present knowledge, to determine the amount of virus in such a preparation must represent only a crude estimate. Detailed findings of experiments in monkeys with these vaccine lots will be given in another report.

Reactions to the Three Viruses

Approximately 32,000 children received vaccine in the school program during April 1955.

So far as can be determined, about the same amount of each of the two lots was used. A diagnosis of poliomyelitis was accepted in 20 of these children (an attack rate of 62 per 100,000); 17 of them suffered paralysis (a paralysis attack rate of 53 per 100,000), and 4 died (a rate of 12 per 100,000).

Although the paralytic attack rate was high for such a widely scattered population, it was much below that observed in some outbreaks of poliomyelitis spread by natural means among populations with a previous history of poliomyelitis. In Minnesota during the summer of 1952, the paralytic attack rate among children of comparable ages was 182 per 100,000, and the death rate was 17.2 per 100,000.

In addition to clinically recognizable poliomyelitis, minor febrile illnesses occurred among children who received the vaccine. However, since vaccination was carried cut at a time when respiratory illnesses were prevalent, it was difficult to relate minor illness to the use of vaccine. Approximately 20 percent of 649 vaccinated children complained of minor illnesses that occurred 1 to 2 weeks after vaccination. The illness consisted chiefly of headache, fever, general aching, and occasionally sore throat, nausea, or vomiting.

A relationship between these minor illnesses and the administration of vaccine was suggested when the antibody titers of the group with the minor illnesses were compared with those of vaccinated children who did not become ill. Since 6 percent of the children in each group did not possess antibodies to any type, they are excluded in this analysis. Children with minor febrile illnesses had a significantly greater proportion of type 2 antibody titers of 1:64 or less than the group without illness.

Type 2 antibody titers were distributed among 144 vaccinated children with minor illness and 463 children with no illness in the following manner:

	Minor illness (percent)	*No illness (percent)*
1:256 or greater	36	53
1:64 or less	64	47

In the group with type 2 antibody titers of 1:256 or greater, 37 percent had an antibody titer of 1:256 or greater for all 3 types; 85 percent for 2 types, and 15 percent for 1 type only. The corresponding figures for the group with a type 2 antibody titer of 1:64 or less were 0 percent, 25 percent, and 50 percent. Approximately 25 percent had a high type 1 antibody titer alone, 25 percent a high type 3 antibody titer alone, and 25 percent both type 1 and type 3 antibodies in high titer. These findings suggest that among individuals who had had no previous experience with type 2 virus, infection with type 1 or type 3 virus might be associated with the symptoms of a minor febrile illness.

That type 1 virus may have been more important than type 3 virus in causing minor illness is suggested by an examination of the group of serums with type 2 antibodies in a titer of 1:256 or greater. Eighty-one percent of the group with minor illness had type 1 antibodies in a titer of 1:256 or greater, whereas 65 percent of the group with no illness possessed such titers. This difference is statistically significant ($P=.022$) and suggests that type 1 infection even in the presence of high type 2 antibody titer may at times cause minor illness. A significant difference was not found in the proportion of high type 3 antibody titers between the two groups.

Many minor illnesses apparently were not caused by infection with a poliovirus since 18 children with minor illnesses either had no antibodies or had antibodies for 1 or 2 types of virus in a titer of 1:8.

Contacts of Vaccinated Children

In 61 instances of poliomyelitis, a history of contact with vaccinated children was obtained. Children under 15 years of age and women were chiefly affected (table 2). Multiple cases occurred in some families. In 56 instances a vaccinated child, usually with no recognized illness, appeared to be the source of infection. Poliomyelitis was diagnosed in only 2 of these vaccinated children and 4 others manifested symptoms of a minor febrile illness.

The number of days from the vaccination of a child suspected to be a carrier to the onset of illness in the 61 contact cases is given in table 3. This period was less than 45 days in 54 instances. When greater than 64 days, a history

Table 2. Age and sex distribution of cases of poliomyelitis among contacts of vaccinated children, Idaho, 1955

Age (years)	Paralytic		Nonparalytic	
	Male	Female	Male	Female
0–4	9	14	1	2
5–9	8	2	2	1
10–14	3	5	2	1
15–19	0	1	0	0
20–24	0	1	0	0
25–29	0	3	0	0
30–34	1	3	0	0
35–39	1	1	0	0
Total	22	30	5	4

NOTE: Three cases, in females aged 6 and 36 and a male aged 36, were fatal.

Table 3. Time of onset after vaccination of cases of poliomyelitis among contacts of vaccinated children, Idaho, 1955

Days after vaccination	All cases	Cumulative totals	Type 1 virus from suspected source	Cumulative totals
16–19	6	6	4	4
20–24	13	19	6	10
25–29	11	30	6	16
30–34	8	38	5	21
35–39	8	46	1	22
40–44	8	54	5	27
45–49	1	55	1	28
50–54	3	58	1	29
55–59	1	59	1	30
60–64	2	61	0	30

of association with vaccinated children was much less frequent, and a carrier other than a vaccinated child was considered to be the source. Periods as short as 16 days between administration of vaccine and onset of illness in an exposed person were observed, and in 6 instances the period was less than 20 days. Such short intervals between vaccination and onset in a contact suggest that several cycles of infection could take place within a 60-day period. It is therefore possible that some of the 61 so-called contact cases are not the result of exposure to the suspected vaccinated child but represent a cycle of infection further removed. It was impossible to designate a definite period during which contact cases could be attributed only to exposure to a vaccinated child.

Cases of poliomyelitis that occurred in Idaho later than 64 days after the vaccination program was terminated were arbitrarily classified as secondary cases. Type 1 poliovirus was isolated from 2 vaccinated children with whom poliomyelitis patients had had intimate contact, but the time of onset was 81 and 83 days, respectively, after vaccination. Whether these children were carrying virus from the time of vaccination or had become infected subsequently is uncertain.

If cases having a history of association with vaccinated children and onset within 64 days after the time of vaccination are accepted as contact cases, a carrier rate of 178 per 100,000 is obtained.

Other Associated Cases

Poliomyelitis occurred in 86 persons whose contact with a vaccinated child did not appear to be the source of infection but in whom type 1 poliovirus was shown to be the cause, or most probable cause, of illness. As shown in figures 3–5 and table 4, the disease occurred in some of these at the same time as it did in those having intimate contact with vaccinated children. Usually, however, onset was so long after the time of vaccination that poliomyelitis due to exposure to a vaccinated child did not appear probable.

As noted in figures 3–5, 19 cases occurred during each of the three 30-day periods from July 1 to September 28, but the number then fell to approximately 7 during the 30-day periods between September 29 and December 28. Probably these cases represent at least the third cycle of infection following the use of vaccine, since they began to appear immediately following the occurrence of known contact cases and occurred in areas where vaccinated children developed poliomyelitis.

Twelve poliomyelitis patients, 11 paralytic and 1 nonparalytic, were admitted to Idaho hospitals from adjoining Malheur County, Ore. Type 1 poliovirus was isolated from nine patients. Onset of the first case was August 24; of the last, October 28. An adjacent Idaho area, in which several cases of poliomyelitis occurred, is a trading center for the Oregon county. It appears possible that these patients

Figure 3. Poliomyelitis, type 1, among persons with onset after July 1, 1955, or with no known contact with children vaccinated on or after April 17, 1955, Idaho.

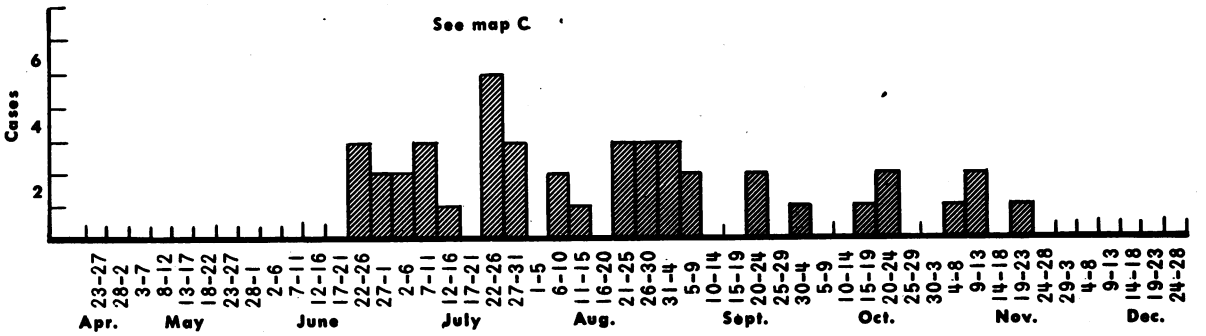


Figure 4. Paralytic poliomyelitis cases with type 1 antibodies and onset after July 1, 1955, or without contact with children vaccinated on or after April 17, 1955, Idaho.

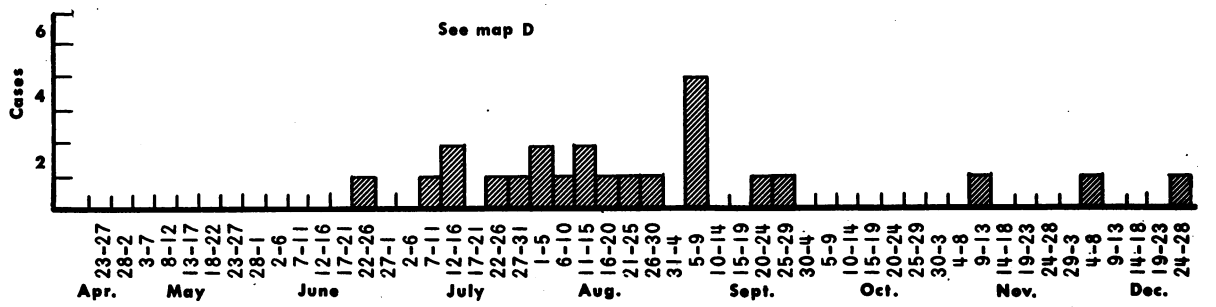
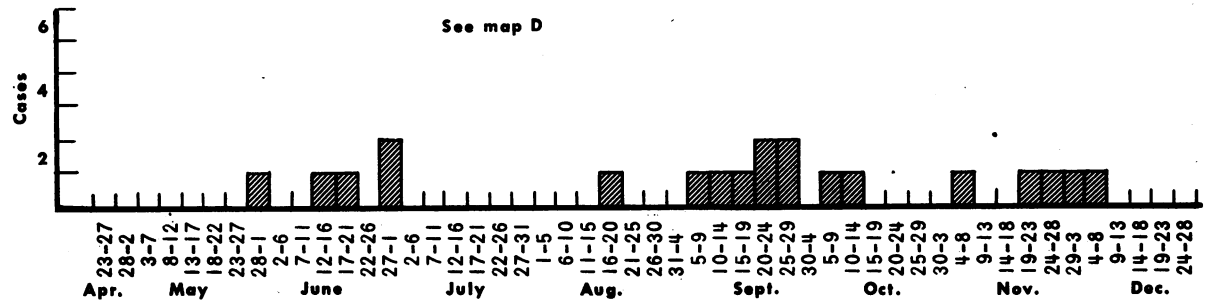
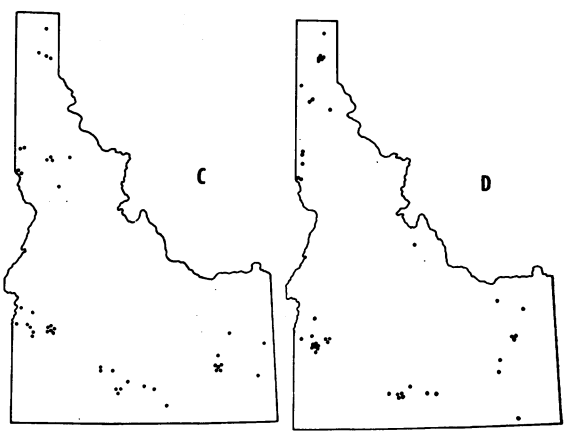


Figure 5. Paralytic poliomyelitis cases, for which no specimens were received, with onset after July 1, 1955, or having no known contact with children vaccinated on or after April 17, 1955, Idaho.



may have been exposed to poliovirus from Idaho cases.



Detection of Types 2 and 3

Type 2 poliovirus was not isolated from children receiving vaccines in Idaho or from their immediate contacts. However, type 2 virus was isolated from the family of a child whose paralytic disease began July 5 and from one patient with nonparalytic poliomyelitis with onset August 23. Serologic studies of 6 other patients with paralytic poliomyelitis showed significant antibody titers for type 2 virus. In

Table 4. Age and sex distribution of poliomyelitis cases unassociated with vaccinated children, Idaho, 1955

Age	Type 1 virus isolated				Type 1 antibodies, paralytic		No specimen, paralytic		Total
	Paralytic		Nonparalytic						
	Male	Female	Male	Female	Male	Female	Male	Female	
0–4	10	8	0	2	1	0	5	0	26
5–9	6	5	1	1	1	2	2	1	19
10–14	1	2	1	1	0	2	2	1	10
15–19	2	0	0	0	3	1	1	0	7
20–24	0	2	0	0	0	3	0	1	6
25–29	1	0	0	0	3	2	1	3	10
30–34	0	0	0	0	2	2	1	0	5
35–39	0	0	0	0	0	0	0	1	1
40–44	0	0	0	0	0	0	0	0	0
45–49	0	0	0	0	0	1	1	0	2
Total	20	17	2	4	10	13	13	7	86

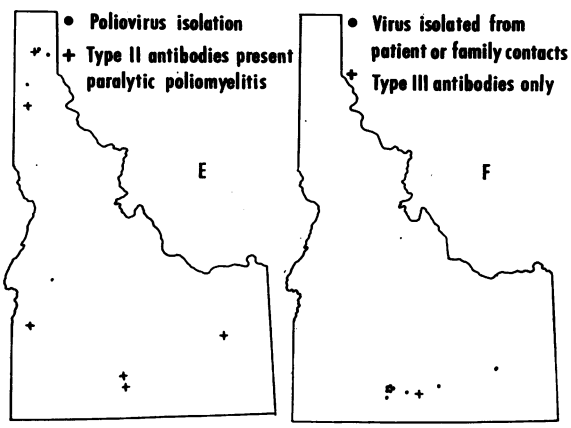
3 of these persons, the onset occurred during July (fifth, seventh, and eighth), 1 during August, 1 during September, and 1 during October. Three patients (2 male and 1 female) were in the age group 0 to 9, 1 patient (female) in the age group 10 to 19, and 4 patients (1 male and 3 females) in the age group 20 to 24. It was not possible to establish that the vaccine was the source of infection in these instances (fig. 6).

Although clinical poliomyelitis due to type 3 virus was not demonstrated in vaccinated children or in their intimate contacts, the possibility that minor illnesses resulted from injection of this virus cannot be excluded. A high neutralizing antibody titer for type 3 poliovirus and low or no neutralizing titers for the other two types of poliovirus were obtained from blood specimens of 21 children who gave a history of minor illness 6 to 8 weeks after vaccination.

Scattered cases of poliomyelitis caused by type 3 virus occurred in Idaho late in the summer. On nine occasions, diagnosis was established by isolation of virus either from patients or from members of the patient's family. In addition, only type 3 antibody was found in the serum of one paralytic patient from whom virus was not isolated. In this group of 10 patients, 4 had onset in July, 4 in August, 1 in September, and 1 in December (figs. 6 and 7). The earliest date of onset was July 9. Five patients (3 male and 2 female) were in the age group 0 to 9, 3 (1 male and 2 female) in the age group 10 to 19, and 2 males in the age group 25 to 34. The sex and age distribution, the time of onset, and the geographic distribution did not suggest a connection with use of the vaccine.

Antibody Levels

During the vaccine study conducted in late 1955 and early 1956, blood specimens were obtained from 480 nonvaccinated individuals, chiefly children, and the levels of antibodies against specific types of poliovirus were determined at the Rocky Mountain Laboratory (table 5). Since the antibody level for type 1 poliovirus was less than 1:8 in 53.5 percent and was 1:1,024 or greater in only 1.2 per-



cent of these children, a limited spread of type 1 virus during the summer and fall of 1955 is indicated.

The antibody titers of blood specimens from children who had received vaccine in 1955 and from those who had served as controls in vaccine studies in Idaho in 1954 were compared for each of the three types of poliovirus. Although titrations of the 1954 serums were performed in the poliomyelitis evaluation laboratory at the University of Oregon Medical College, Portland, the data are considered to be roughly comparable. More children in the 1955 group had titers of 1:1,024 or higher against each of the viruses, whereas a lower proportion had a serum titer of less than 1:8 (table 5). Since serums of approximately 30 percent of the children vaccinated in 1955 had no appreciable titer against type 1 and type 3 viruses, these children apparently were not infected by the amount of viruses present in the vaccines (table 6). Antibody responses to three doses of vac-

Figure 6. Poliomyelitis, type 2, after vaccinations beginning April 17, 1955, Idaho.

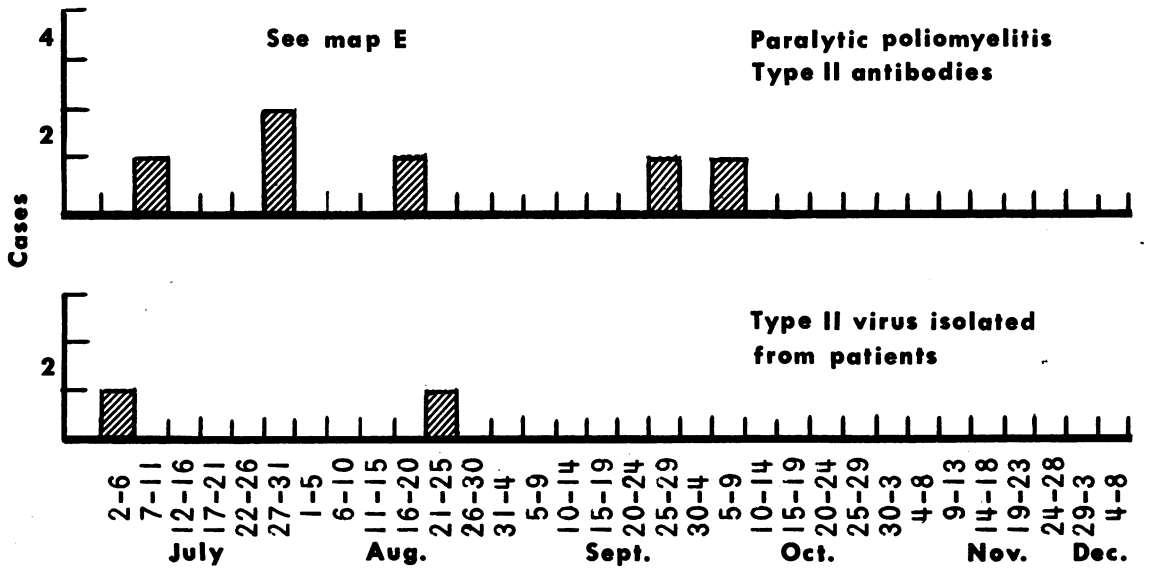


Figure 7. Poliomyelitis, type 3, after vaccinations beginning April 17, 1955, Idaho.

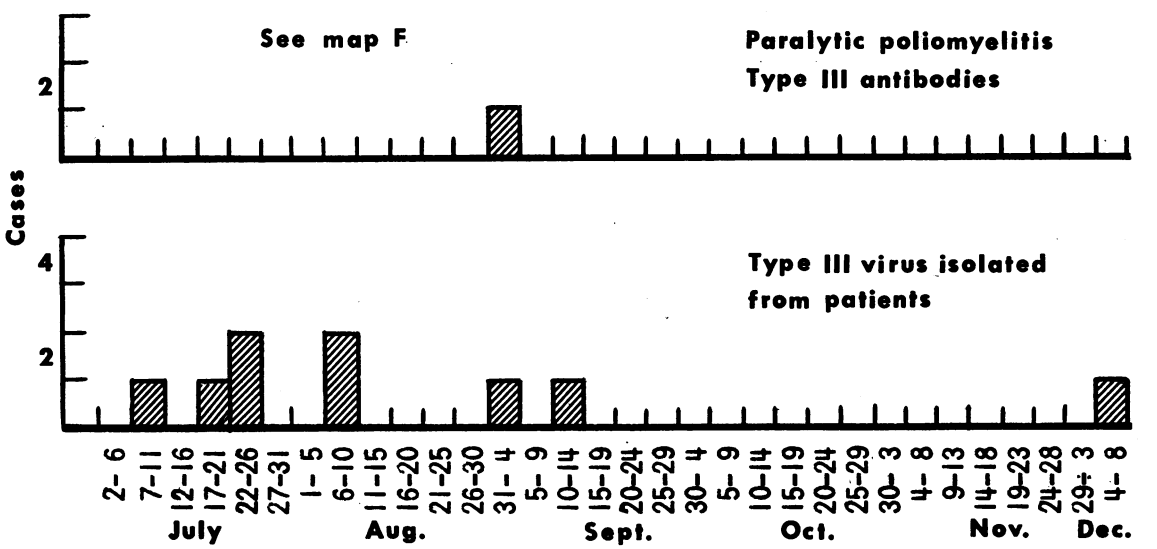


Table 5. Comparison of antibody titers of serums from children vaccinated in Idaho, 1955, with serums from nonvaccinated children

Category	Percentage of specimens with indicated reciprocals of titer							
	<8	8	16	32	64	256	Total 8 to 256	1,024 or>
Type 1 virus								
Vaccinated 1955 [1]	27	3	3	5	8	14	33	40
Nonvaccinated 1955 [2]	54	3	3	7	19	13	45	1
Nonvaccinated 1954 [3]	46	4	9	------	19	15	47	7
Type 2 virus								
Vaccinated 1955 [1]	13	10	8	9	14	15	56	31
Nonvaccinated 1955 [2]	65	2	2	3	8	12	27	8
Nonvaccinated 1954 [3]	59	2	6	------	13	12	33	8
Type 3 virus								
Vaccinated 1955 [1]	32	6	5	4	3	9	27	41
Nonvaccinated 1955 [2]	48	2	5	7	20	13	47	5
Nonvaccinated 1954 [3]	54	1	13	------	20	8	42	4

[1] 649 children vaccinated April 17–22, 1955; serums obtained June 2–14, 1955.
[2] Serums obtained from 480 children during late fall and winter 1955–56.
[3] Serums obtained from 281 children, November 1954, in connection with the 1954 NFIP poliomyelitis evaluation study.

cine with "good" antigenicities reported in the 1954 study (*3*) and of vaccine used in Idaho in 1955 are compared in table 6.

Antibody distribution for children vaccinated in 1954 is well illustrated by the results obtained through neutralization of type 1 poliovirus (table 6). The discrepancy between the proportion of children having intermediate titers in the group vaccinated in 1955 and the proportion in the group vaccinated in 1954 is what would be expected from administration of live virus. Individuals either developed infection and as a result produced antibodies in high titer or failed to become infected and consequently produced no antibodies.

Table 6. Antibody levels after 3 injections of "good" vaccine lots in the 1954 poliomyelitis evaluation study of NFIP compared with those developing after a single injection of vaccine containing live virus

	Total number	Percent with titers 8 or <	Percent with titers 16 to 256	Percent with titers 1,024 or >
Type 1				
1954	1, 250	14	51	35
1955	657	31	29	40
Type 2				
1954	1, 250	7	60	33
1955	657	23	46	31
Type 3				
1954	1, 250	10	58	32
1955	657	38	21	41

Discussion

It is rather surprising that the intramuscular inoculation of a virulent strain of type 1 poliovirus, which was capable of spreading from person to person, did not give rise to a more severe epidemic. The population had a large number of susceptible individuals, as indicated by antibody studies before and after administration of the vaccine and by the past history of poliomyelitis in the area. Vaccine was given in the spring when the weather was cold, but type 1 virus was still present in the population later in the summer. Yet an increase in the incidence of poliomyelitis did not occur. Consequently, one must conclude that a simple relationship of virulence of virus to

immune status of a population and to season of the year is inadequate to explain the epidemic occurrence of poliomyelitis.

Strangely, viable types 2 and 3 polioviruses present in the vaccine did not cause disease in vaccinated children or spread to their contacts. This could be due to a lack of virulence of these viruses for human beings when given intramuscularly or to interference between strains when administered simultaneously to susceptible individuals. The presence in vaccinated children of high antibody titers for types 2 and 3 polioviruses, however, suggests that these strains proliferated. The absence of central nervous system disease due to infection with type 2 and type 3 strains demonstrates the inability of these strains to invade the central nervous system following intramuscular inoculation. In monkeys, these strains, particularly type 3, showed little ability to produce viremia or to proliferate in peripheral tissues, characteristics which may be related to absence of disease in vaccinated children. The type 1 strain, which was definitely virulent for children, frequently was isolated from blood and peripheral tissues of monkeys. A type 1 strain without these characteristics appears desirable for use in a vaccine.

Summary

In Idaho, with a population of approximately 600,000, two lots of poliomyelitis vaccine were used during April 1955 to vaccinate 32,000 children in the first and second grades. All three types of poliovirus were isolated from these lots of vaccine. Poliomyelitis due to type 1 virus occurred in 20 vaccinated children and in 61 of their contacts. In addition, 86 persons developed poliomyelitis from possible contact with these two groups. Thus, 167 cases were associated with the use of these two lots of vaccine. Poliomyelitis due to type 2 or type 3 viruses was infrequent, and evidence relating infections with these types to the vaccine used was not obtained. The absence of a severe outbreak of poliomyelitis due to type 1 virus is noteworthy in view of the wide dissemination of a virulent strain of type 1 poliovirus throughout the State. Since the immunity of the population was rather low as indicated by the previous history of poliomyelitis and by the 1954 vaccine evaluation study, a more severe outbreak would have been expected. Factors in addition to virulence of a poliovirus strain and immune status of a population are yet to be discovered before the genesis of poliomyelitis outbreaks can be explained adequately.

REFERENCES

(*1*) Eklund, C. M., Bell, E. J., and Hadlow, W. J.: Detection of live virus in certain lots of poliomyelitis vaccine by inoculation of monkeys. Am. J. Hyg. 64: 85–91, July 1956.

(*2*) Salk, J. E., Youngner, J. S., and Ward, E. N.: Use of color change of phenol red as the indicator in titrating poliomyelitis virus or its antibody in a tissue-culture system. Am. J. Hyg. 60: 214–230, September 1954.

(*3*) Francis, T., Jr., and associates: An evaluation of the 1954 poliomyelitis vaccine trials. Summary report. Am. J. Pub. Health 45: 1–63, May (part 2) 1955.

U. S. Medical Supply Stocks Surveyed

A survey of the Nation's supply of medical items essential to survival following nuclear attack is being conducted by the Public Health Service. With the cooperation of the pharmaceutical industry, more than 700 wholesale drug houses, surgical supply firms, and chain drugstore warehouses will be covered in the survey, which is part of a program set up by the Office of Defense Mobilization. Also participating in this program are the Business and Defense Services Administration of the Department of Commerce, the Federal Civil Defense Administration, and the Department of Defense.