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The incidence of poliomyelitis virus in normal children aged 0–5 years

A report on a study by the Public Health Laboratory Service and Local Health Authorities*

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INTRODUCTION

Many reports have been published on the incidence of symptomless poliomyelitis carriers in the general community. These have given estimates of the ratio of carriers to clinical cases ranging from 50:1 to 1000:1. Some of the most thorough studies, such as those of Turner, Hollander, Buckley, Kokko & Winsor (1950), Melnick & Ledinko (1953), Melnick, Walton, Isaacson & Cardwell (1957), Fox, Gelfand, le Blanc & Conwell (1957) and Gelfand, le Blanc, Fox & Conwell (1957) were made in the United States and the results are not necessarily applicable in England and Wales. The present investigation was undertaken by the Public Health Laboratory Service in collaboration with Local Health Authorities to provide similar information for this country, and a base-line against which future studies undertaken after the full introduction of vaccination against the disease, could be judged. Also, in view of the wide variation in other reported results, any additional information on the normal carrier rate is of potential interest. A preliminary account of the first year's results has been published elsewhere (Report, 1958).

MATERIALS AND METHODS

The American investigations mentioned above were made on groups of individuals who were tested on successive occasions over a period of time for the presence of virus in the faeces or the appearance of antibody in the blood. The present investigation was carried out by sampling from the community at intervals, different subjects being taken on each occasion. Some areas taking part in the first year were unable to continue in the second, and other areas took part in the second year but not the first. Details of these areas for each year are given in Appendix A. An account of the method of sampling has been given in the report on the first year's results (Report, 1958).

The entry of various laboratories and Health Departments into the survey was

* A full list of the laboratories and local authorities taking part in the investigation is given in Appendix A (p. 155), and the report is based on the collation and analysis by the author of material obtained from these sources.

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irregular, and consequently there were fluctuations in the total numbers of specimens at the beginning and end of the survey. The limited staff available for collecting specimens made it impossible for the same number to be collected at each interval through the year. This was particularly the case at holiday periods such as Christmas and midsummer. The survey was confined to children aged less than 5 years. This limitation was imposed because the selection of a random sample of individuals in the general population presents considerable difficulty, and experience had shown that the proportion of subjects refusing to co-operate is high among adults. Mothers of young children on the other hand, being more accustomed to seeing health visitors and others from health departments, are usually willing to take part in surveys of this kind.

The method of selecting the children for participation in the survey was to take names at random from the birth registers and was carried out locally by Health Department staffs. Details of the method used varied somewhat from area to area according to local conditions. Usually sampling was begun in one definite week, 4 weeks or so before the start of the survey. From the births in this week names and addresses were taken at random to provide a sample of children in the age group 0–1. The next age group was provided by the corresponding week in the year before, the next in the year before that and so on. As many children were chosen in this way as could be conveniently visited in one week and usually a few extra names were included to replace refusals and unsuccessful visits. The next week's sample was chosen from the next set of week's births and so on.

A sample chosen in this way is not strictly random in relation to date of birth, since the dates of birth chosen are in constant relation to one another and to the season. Another minor objection is that sampling by birth entails a slight excess of males. On the other hand, the method is a practical one for use by those unfamiliar with random sampling procedures and it does cover the whole of one year's births.

The specimens were collected by either health visitors or public health inspectors. The actual method again varied from district to district, but as a rule the families were circularized first by letter and subsequently visited. The specimens were sent to the laboratory as quickly as possible, either by post or by direct delivery but almost always within 24 hr.

An undesirable feature caused by shortage of staff was that unsuccessful week-day visits could not as a rule be followed up in the evening or week-ends. In sampling human populations unsuccessful visits during the day are usually concentrated among a special group of households containing particularly those where both parents go out to work. Another source of difficulty was the transfer of the family to another address. This occurred more often with older than with younger children. Because of this the sample contained proportionately more younger than older children, as can be seen from Table 1. It was not practicable to overcome difficulties of this kind without a special team of full-time field workers, as the work involved would have been prohibitively great.

Areas taking part in survey

A full list of the areas taking part in the survey is given in Appendix A. The greater part of these were urban in character, because the collection of specimens in rural districts requires much more travelling than in towns. As the participation of an area in the survey was purely voluntary, and depended to a large extent on the staff available in the local Health Department, the areas were not a random sample of the country as a whole.

Table 1. *Age distribution of children submitting specimens*

(Figures in parentheses are percentages.)

	Age groups						
	0–1 yr.	1–2 yr.	2–3 yr.	3–4 yr.	4–5 yr.	Age unknown	All ages
1st year	2755 (25·6)	2419 (22·5)	2129 (19·8)	1993 (18·5)	1411 (13·1)	54 (0·5)	10761
2nd year	4166 (23·1)	4028 (22·3)	3565 (19·8)	3479 (19·3)	2691 (14·9)	107 (0·6)	18036
Both years	6921 (24·0)	6447 (22·4)	5694 (19·8)	5472 (19·0)	4102 (14·2)	161 (0·6)	28797

For most of the areas studied no up-to-date details were available of the population aged 0–5 years. In areas with no published figures rough estimates of these populations were made on the basis of the 1951 census figures and adjusted according to the most relevant administrative area for which current population estimates were published. Thus, for example, estimates are given in the Quarterly Returns of the General Register Office for administrative counties and the population in a district within a county was estimated from this according to its relative position in the 1951 census. Such estimates are very rough but their errors are small in comparison with the others which arise in studies of this kind.

Details of all confirmed cases of poliomyelitis in the study were provided by the local Medical Officers of Health.

Virological methods

Practice in this respect varied slightly from one laboratory to another, but most laboratories used HeLa cell cultures. The minimal method recommended by the Virus Reference Laboratory, Colindale, was given in the previous Report (1958). An examination (unpublished) of the records of virus isolations by the laboratories of the Public Health Laboratory Service had shown that the proportion of isolations from paralytic cases at comparable stages of the disease differed very little from laboratory to laboratory. It did, however, suggest that the frequency of isolation in single specimens from confirmed paralytic cases was only of the order of 50 %. For this reason it is probable that the reported isolations in the survey have underestimated the actual number of carriers by a factor of two and possibly even more if paralytic cases excrete more virus than the non-paralytic cases or carriers.

A possible source of error was cross-contamination of specimens in the laboratory. If specimens from the survey were examined during an epidemic period it would be inevitable that many positive specimens were also being examined from suspected clinical cases. Consequently the danger of contamination would be greatest at a time when it was likely to be misleading. This error would not have arisen in most laboratories, since the general practice was to store the specimens in the cold during busy periods and test for virus when the pressure on tissue culture resources was less. Two laboratories subsequently re-checked the survey specimens during the winter and discovered no earlier mistakes. It is felt that no serious errors due to contamination occurred and, as pointed out above, the methods used were more likely to have underestimated than to have overestimated the frequency of normal carriers.

As well as poliomyelitis virus, other cytopathogenic agents appearing in tissue culture were investigated, but practice in this respect varied from one laboratory to another, depending on the facilities available locally. Most laboratories also examined the specimens for the presence of *Salmonella* and *Shigella*.

RESULTS

The main results of the survey are summarized in Tables 2–4. Table 2 gives the distribution of cases and carriers in 4-weekly periods for the duration of the survey. The next two tables show the totals arranged by laboratories. The classification by laboratories is a matter of convenience, as most of them covered several local authority areas which it would be unwieldy to cite in full. The position of the laboratory can be taken to indicate roughly the geographical area concerned. Details of the areas actually sampled are given in Appendix A.

The relation between carrier rates and the notification rates can be judged from Table 2, where it will be seen that their variations run roughly parallel over the period. There is a slight tendency for the notifications per carrier to be higher at the periods of greatest prevalence which may be due either to over-notification or possibly to an increase in virulence. It can also be seen that there are rather more notifications per carrier in the second than in the first year, though the incidence of the disease was lower.

A notable feature of the second year is the complete absence of poliovirus Type 2 carriers, even though these were relatively common in the first.

Tables 3 and 4 show how the three types were distributed among the various areas and their relationship to the total notification rates. The number of isolations in any one area was usually small, as was the number of notified cases. The largest number of carrier isolations was made in Bath during the epidemic of 1957 and only amounted to 26. These small numbers imply a large sampling variability, of the order of 50–100%, in the individual carrier rates and the notification rates have a similar uncertainty. The combination of these rates to obtain estimates of the relationship between carrier rates and notifications presents a number of problems which will be dealt with in the discussion. These difficulties arise partly from the sampling errors and partly from the fact that the proportion of carriers at any

given moment can only be related to the incidence of new infections when the average duration of the carrier period is known.

Because of these complications the interpretation of Tables 3 and 4 is not straightforward, though it can be seen that there is a broad general relationship between the carrier rates and the notifications. The rather erratic nature of this is in keeping with the previous results of Melnick *et al.* (1957), Fox *et al.* (1957) and Gelfand *et al.* (1957).

Table 2. *Isolation rates of virus types and incidence of notified poliomyelitis in the survey areas by 4-weekly periods*

4-week period	No. of specimens examined	Polio virus positive				Isolation rate/1000				Cases all ages	Cases under 5	Case rate/1000 under 5
		1	2	3	All types	1	2	3	All types			
1957 14–17	504	1	1	—	2	1·98	1·98	—	3·97	5	3	0·0090
18–	686	4	—	—	4	5·83	—	—	5·83	13	6	0·0179
22–	765	8	—	—	8	10·46	—	—	10·46	32	7	0·0209
26–	899	8	2	3	13	8·90	2·22	3·34	14·46	68	23	0·0687
30–	846	21	3	3	27	24·82	3·55	3·55	31·91	128	28	0·0837
34–	853	12	6	3	21	14·07	7·03	3·52	24·62	119	30	0·0896
38–	847	9	6	6	21	10·62	7·08	7·08	24·79	47	16	0·0478
42–	848	16	—	5	21	18·87	—	5·90	24·76	13	5	0·0149
46–	883	7	2	6	15	7·93	2·26	6·79	16·99	44	19	0·0568
50–	661	8	3	4	15	12·10	4·54	6·05	22·69	21	6	0·0179
1958 2–	831	4	—	2	6	4·81	—	2·41	7·22	10	2	0·0060
6–	819	2	1	1	4	2·44	1·22	1·22	4·88	9	4	0·0119
10–	823	4	—	—	4	4·86	—	—	4·86	6	2	0·0060
14–	425	—	—	1	1	—	—	2·35	2·35	6	—	—
18–	824	—	—	—	—	—	—	—	—	3	1	0·0021
22–	1,440	—	—	3	3	—	—	2·08	2·08	7	3	0·0064
26–	1,481	4	—	—	4	2·70	—	—	2·70	28	9	0·0193
30–	1,477	7	—	—	7	4·74	—	—	4·74	39	17	0·0364
34–	1,418	5	—	4	9	3·53	—	2·82	6·35	31	10	0·0214
38–	1,522	5	—	1	6	3·29	—	0·66	3·94	46	23	0·0493
42–	1,443	6	—	—	6	4·16	—	—	4·16	41	16	0·0343
46–	1,482	2	—	—	2	1·35	—	—	1·35	24	4	0·0086
50–	948	5	—	—	5	5·27	—	—	5·27	9	2	0·0043
1959 1–	1,315	3	—	—	3	2·28	—	—	2·28	8	3	0·0064
5–	1,300	—	—	1	1	—	—	0·77	0·77	3	—	—
9–	1,301	—	—	1	1	—	—	0·77	0·77	3	2	0·0043
13–	1,104	—	—	—	—	—	—	—	—	3	1	0·0021
17–	662	—	—	—	—	—	—	—	—	5	2	0·0043
21–	289	—	—	—	—	—	—	—	—	3	2	0·0043
25–	54	—	—	—	—	—	—	—	—	—	—	—
29–32	9	—	—	—	—	—	—	—	—	—	—	—
Week unknown	38	—	—	—	—	—	—	—	—	—	—	—
Total	28,797	141	24	44	209	4·90	0·83	1·53	7·26	774	246	0·3070

The carrier rates for the three types of virus at various ages are given in Table 5. There is no very distinct trend with age in the rates for Type 1 virus, but the incidence of both Type 2 and Type 3 carriers increases with age up to 5 years. In view of possible bias in selection of children at the older ages this finding must be interpreted with caution, but it is reasonable to suppose that Type 1 virus, being commoner than either of the other two, is encountered earlier in life.

The distribution of carriers according to sex is shown in Table 6. The Type 1 carrier rate is significantly higher in males, but the other two types do not differ significantly in their incidence. There is no obvious explanation of this excess in males, which is most unlikely to be due to chance ($P \doteqdot 0{\cdot}00001$), and is in keeping with the fact that notified poliomyelitis is commoner in the male (Logan, 1952). Whether this sex difference is really absent in Type 2 and Type 3 infection is an interesting point that might be worth further investigation.

Table 3. *Isolation rates of three types of virus and total poliomyelitis notification rates in children under 5, in first year of survey*

Laboratory	Notification rate/1000 under 5 yr.	Isolations			No. of specimens	Isolation rate/1000 specimens			
		Type 1	Type 2	Type 3		Type 1	Type 2	Type 3	All types
Bath	0·759	11	15	—	563	19·54	26·64	—	46·18
Bedford	0·562	3	—	7	481	6·24	—	14·55	20·79
Birmingham	0·326	5	2	1	422	11·85	4·74	2·37	18·96
Cardiff	0·625	6	3	2	337	17·80	8·90	5·93	32·64
Carlisle	—	—	—	—	389	—	—	—	—
Chelmsford	0·229	2	—	—	662	3·02	—	—	3·02
Colindale	0·511	4	—	—	500	8·00	—	—	8·00
Dorchester	—	—	—	—	68	—	—	—	—
Exeter	2·037	3	—	—	425	7·06	—	—	7·06
Guildford	—	1	—	—	205	4·88	—	—	4·88
Leeds	0·311	1	—	4	240	4·17	—	16·67	20·83
Leicester	1·179	8	—	1	494	16·19	—	2·02	18·22
Luton	—	—	—	—	260	—	—	—	—
Maidstone	0·792	12	—	—	536	22·39	—	—	22·39
Manchester	—	9	2	1	625	14·40	3·20	1·60	19·20
Newcastle	0·185	10	—	3	515	19·42	—	5·83	25·24
Nottingham	0·440	5	—	—	501	9·98	—	—	9·98
Plymouth	0·355	1	—	—	255	3·92	—	—	3·92
Portsmouth	0·723	12	1	—	770	15·58	1·30	—	16·88
Salisbury	—	—	—	—	190	—	—	—	—
Sheffield	0·134	2	—	—	537	3·72	—	—	3·72
Stafford	0·312	—	—	1	356	—	—	2·81	2·81
Swansea	0·250	3	1	4	494	6·07	2·02	8·10	16·19
Taunton	—	1	—	—	260	3·85	—	—	3·85
Wakefield	0·682	4	—	10	504	7·94	—	19·84	27·78
Winchester	0·952	1	—	—	172	5·81	—	—	5·81
Total	0·451	104	24	34	10,761	9·66	2·23	3·16	15·05

Information was collected in some laboratories on the prevalence of viruses other than those causing poliomyelitis. This is summarized in Table 7, which gives the quarterly isolation rates for the period of the survey. The Coxsackie viruses were the most common and ran parallel in their seasonal incidence to poliomyelitis. By far the commonest type isolated was Coxsackie B virus though a few Type A strains were also reported. The isolation rate for these viruses was higher than that of poliomyelitis during the second year, when the incidence of poliomyelitis was low. Adenoviruses were frequently isolated, their prevalence being of the same order as that of poliomyelitis but showing a winter peak. The ECHO viruses were less common, and the rather small numbers that were isolated mainly occurred in autumn.

Because of variations in practice between laboratories and between the two

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years of the survey it is not possible to draw very firm conclusions about the incidence of these enteric viruses. However, their relative seasonal variations are probably correctly shown, though their frequency may be greatly underestimated, since the techniques of isolation used were not necessarily the most suitable.

Table 4. *Isolation rates of three types of virus and total poliomyelitis notification rates in children under 5, in second year of survey*

Laboratory	Notification rate/1000 under 5 yr.	Isolations			No. of specimens	Isolation rate/1000 specimens			
		Type 1	Type 2	Type 3		Type 1	Type 2	Type 3	All types
Bath	—	1	—	—	751	1·33	—	—	1·33
Bedford	1·538	4	—	—	358	11·17	—	—	11·17
Birmingham	0·045	—	—	1	362	—	—	2·76	2·76
Bradford	0·642	1	—	1	226	4·42	—	4·42	8·85
Brighton	0·194	2	—	—	1,881	1·06	—	—	1·06
Cardiff	—	4	—	—	339	11·80	—	—	11·80
Chelmsford	0·446	1	—	—	1,009	0·99	—	—	0·99
Colindale	—	2	—	1	2,808	0·71	—	0·36	1·07
Derby	0·112	—	—	—	1,330	—	—	—	—
Guildford	0·100	—	—	—	592	—	—	—	—
Hereford	—	—	—	—	510	—	—	—	—
Leeds	0·763	3	—	1	328	9·15	—	3·05	12·20
Leicester	—	—	—	—	719	—	—	—	—
Luton	—	—	—	—	259	—	—	—	—
Maidstone	0·122	—	—	1	530	—	—	1·89	1·89
Middlesbrough	0·420	—	—	—	134	—	—	—	—
Newcastle	0·093	2	—	—	466	4·29	—	—	4·29
Portsmouth	0·051	3	—	—	557	5·39	—	—	5·39
Salisbury	—	—	—	—	70	—	—	—	—
Sheffield	0·142	1	—	—	867	1·15	—	—	1·15
Stafford	0·057	—	—	3	1,421	—	—	2·11	2·11
Swansea	—	1	—	—	781	1·28	—	—	1·28
Wakefield	1·556	12	—	2	473	25·37	—	4·23	29·60
Worcester	—	—	—	—	1,265	—	—	—	—
Total	0·206	37	—	10	18,036	2·05	—	0·55	2·61

Table 5. *Frequency of virus types: rates by ages/1000 specimens for each year of survey*

	0–1 yr.	1–2 yr.	2–3 yr.	3–4 yr.	4–5 yr.	Total
Type 1						
1st year	7·62	9·92	12·68	7·53	12·05	9·66
2nd year	2·64	1·99	1·96	2·30	1·11	2·05
Both years	4·62	4·96	5·97	4·20	4·88	4·90
Type 2						
1st year	1·45	2·07	1·41	2·51	4·96	2·23
2nd year	—	—	—	—	—	—
Both years	0·58	0·78	0·53	0·91	1·71	0·83
Type 3						
1st year	2·18	2·48	3·76	4·01	4·25	3·16
2nd year	0·24	0·25	0·84	0·86	0·74	0·55
Both years	1·01	1·09	1·93	2·01	1·95	1·53
All types						
1st year	11·25	14·47	17·85	14·05	21·26	15·05
2nd year	2·88	2·23	2·81	3·16	1·86	2·61
Both years	6·21	6·82	8·43	7·13	8·53	7·26

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Most laboratories examined specimens for *Shigella*, *Salmonella* and pathogenic coliform organisms. The main results are given in Table 8, and some of those for the first year have been given detailed consideration elsewhere (Report, 1959). Taken over the two years the salmonellae and shigellae (the latter all being *Sh. sonnei* and the former mainly *S. typhimurium*) were about equally prevalent with

Table 6. *Isolation rates of the three types of virus classified according to sex of carrier*

	Polio isolation rate/1000 specimens			
	Type 1	Type 2	Type 3	All types
Male	5·66	0·23	1·91	7·80
Female	4·28	0·48	1·53	6·29
Sex unknown	4·20	4·50	—	8·71
Total	4·90	0·83	1·53	7·26

Table 7. *Quarterly isolation rates per* 1000 *specimens of viruses other than that of poliomyelitis*

		Rates/1000 specimens			
		Coxsackie	Adenovirus	ECHO	Unidentified
1957	April–June	3·9	10·3	—	7·2
	July–Sept.	11·7	7·3	17·9	1·9
	Oct.–Dec.	2·7	8·5	16·7	21·7
1958	Jan.–Mar.	7·2	12·0	8·8	9·2
	April–June	23·1	13·7	—	—
	July–Sept.	23·1	1·2	2·6	9·4
	Oct.–Dec.	3·2	1·7	6·2	4·8
1959	Jan.–Mar.	—	4·6	2·3	—
	April–June	2·9	9·2	1·7	—
	Total	9·8	6·5	3·6	8·2

Table 8. *Quarterly isolation rates per* 1000 *specimens examined of* Salmonella, Shigella *and pathogenic coliform organisms*

		Isolation rate/1000 specimens		
		Salmonella	*Shigella*	Coli
1957	April–June	1·5	4·4	12·3
	July–Sept.	3·1	3·1	20·0
	Oct.–Dec.	1·2	1·6	7·6
1958	Jan.–Mar.	1·3	4·6	13·6
	April–June	2·0	0·8	15·9
	July–Sept.	4·1	1·0	23·5
	Oct.–Dec.	2·7	1·7	15·3
1959	Jan.–Mar.	1·7	0·8	8·8
	April–June	1·4	3·4	18·2
	Total	2·3	2·1	15·4

frequencies of approximately 2 per 1000, but during the first year *Sh. sonnei* was predominant, and during the second the salmonellae. The pathogenic coliforms were more than five times as frequent.

DISCUSSION

The main information provided by the present survey is an estimate of the actual prevalence of virus in a widely separated set of areas during two different years. From this it is possible to deduce the relationship of the number of carriers to the number of notified cases and compare the results to those obtained in other surveys. The longitudinal type of survey which has been mainly used in other surveys is a more direct approach and gives immediate estimates of the incidence of new carriers in the community. Its main disadvantage is that it must usually be confined to a comparatively small number of persons or households who are willing to submit repeated samples of faeces. Any bias in the selection of these persons will be present throughout the survey. In a study of the type reported here the net is spread much more widely in the community and it is more likely that mothers will co-operate on one occasion than if they are asked to participate for a year or more. It is felt that the method used here is more suitable for delegation to local workers with little experience of survey work and random sampling, while the longitudinal method is one of choice and should be used when a special team is available for the field work.

As mentioned above, the method of sampling used does not give a direct estimate of the incidence of new carriers in the community, since the probability of picking a positive carrier depends partly on the carrier incidence and partly on the length of time for which a carrier excretes the virus. A disease with a high incidence and short carrier period is equivalent for sampling purposes to one with a low incidence and long carrier period, since the sample is estimating the total volume of virus being excreted in the community. If the sample is to give a true picture it is necessary that the community should be sampled with equal intensity throughout the period of the study, and that the average carrier period of the disease should not vary from one part of the period to another. Both these two conditions are believed to have been approximately fulfilled in the present study. The main disturbance of the first condition arises from the falling off in specimens collected during the summer and Christmas holiday periods. As regards the second condition there is no reason to believe that there is any variation in the carrier period throughout the year, though it must be admitted as a possibility in the absence of any positive evidence. If serious discrepancies with previous work had been observed this might have been worth special investigation, but in fact the assumption of a constant average carrier period seems justifiable.

No special study was made of the average carrier period of symptomless excreters during the survey. Estimates have been published by Gelfand *et al.* (1957) and Hatch, Hughes & Pilfold (1958), and an analysis has been made of routine isolations by the Public Health Laboratory Service from symptomless excreters, usually contacts of known cases. Gelfand *et al.* (1957) give an average

carrier period in their survey of 51 days or about 7 weeks. Hatch *et al.* (1958) give a period of 5 weeks in healthy contacts, dating the beginning of excretion at the time of onset in the primary case; this is close to the value of about 30 days similarly obtained from Public Health Laboratory Service records. However, the assumption that the contact carriers all originated at onset of disease in the index case is not strictly justifiable, and as Gelfand *et al.* (1957) obtained their estimate by a relatively direct process it is likely that their estimate of about 7 weeks for the average duration of the carrier period is more reliable and this figure will be used in converting the carrier rates to incidence rates.

The relation between the carrier rates and the notification rates in the various areas was calculated from the figures in Tables 3 and 4. As Type 2 and Type 3 of the virus were usually present in small numbers along with Type 1 in the same area, it is only possible to give a good estimate of the relationship between notification and carrier isolation rates for Type 1 virus in the second year. For this type the increase in notifications per unit increase in carrier isolation rate was about $0{\cdot}058 \pm 0{\cdot}010$. In the first year the corresponding ratio was $0{\cdot}027 \pm 0{\cdot}013$ which scarcely differs significantly from zero. However, the errors of estimation are large, and the observed figures could quite well have arisen if the ratio of carriers to cases was equal in the two years. The statistical problems involved in making the estimates are discussed in Appendix B.

The underlying relation between the incidence of new infections and notifications can only be made when the average duration of the carrier period is known. If this average is known to be T, the relationship between the notification rate (n) and the isolation rate (r) is

$$n = (h/T)r,$$

where h is the proportion of newly infected who become notified cases of poliomyelitis over the period of observation, and is the basic information required. The value of h/T given above is about 0·05, and the period of observation 1 year, so that taking $T = 7$ weeks, i.e. 7/52 of a year, h is approximately 7/1000, and there are about 140 carriers for every Type 1 virus infection that becomes a notified case. This is in agreement with the values of 100–175 found by Melnick & Ledinko (1953) for children aged 0–2 years, and is within the range of those found by Melnick *et al.* (1957), Fox *et al.* (1957) and Gelfand *et al.* (1957).

The survey provides little evidence for seasonal or geographical fluctuations in virulence, but it does demonstrate the presence of virus in the population throughout the year. It also shows that the carriers of the virus and the notified cases run more or less parallel in their incidence so that the seasonal variation is primarily a question of spread of virus rather than an increased liability of infected subjects to develop clinical symptoms.

A comparison of the two years of the survey also suggests that the lower incidence of poliomyelitis in the second year was due to diminished transmission of the virus and not to a loss of virulence, since the number of notified cases per carrier was, if anything, higher in the second year when the incidence of poliomyelitis was low. It seems very likely that the cold, wet weather conditions in the summer of 1958 may have reduced the spread of the virus.

The carrier rates during the spring, though small, leave little doubt that there exists a sufficient reservoir of carriers to maintain the virus in the population until seasonal conditions once again favour its spread.

The incidence of Type 2 and Type 3 virus was considerably less than that of Type 1, Type 2 being completely absent in the second year. If the two years of the study are typical they suggest that Type 2 and Type 3 are of low incidence as well as low virulence. A rough idea of the virulence of these two types relative to Type 1 may be obtained by comparing their proportions among the isolations in the survey with those among clinical cases in similar areas examined by the laboratories taking part. The proportions of the three types isolated from poliomyelitis patients in the two years are given in Table 9. A measure of the relative virulence of any two types can then be obtained by calculating the ratio

$$(p_1/p_2)(r_2/r_1),$$

where p_1 and p_2 are the proportions of the two types in isolations from clinical cases and r_1 and r_2 the corresponding proportions in the normal carriers. This calculation suggests that Type 2 and Type 3 are of about equal virulence each being about 1/10 as virulent as Type 1. It is difficult, however, to make definite statements, since these estimates have a large sampling error and are affected by differences in notification practice. In addition, the population in which the virus types causing clinical cases are known does not coincide exactly with that in which the carrier rates are known.

Table 9. *Percentages of the three types of poliomyelitis virus isolated from clinical cases in approximately the same geographical areas as the survey*

	Type 1	Type 2	Type 3
1st year	95·18	1·79	3·03
2nd year	98·50	—	1·50
Total	95·94	1·38	2·68

Taken as a whole the results of the present survey are in good accord with those carried out elsewhere, and it is reasonable to conclude that the method of sampling used was satisfactory. It is difficult to see how these findings and those of the other surveys quoted can be reconciled with a narrow-stream theory of infection. The areas studied form a fairly typical cross-section of urban England and Wales, and in them it was not uncommon to find that 10–20 % of the population under 5 had been excreting the virus at some time in the course of a year. The only sense in which a narrow stream of infection can be interpreted is that perhaps sometimes exceptionally virulent strains arise which cause a high local incidence of paralytic disease, but there is at present little evidence for this view.

SUMMARY

A survey has been made of a sample of children aged 0–5 years in a number of areas in England and Wales for the presence of symptomless excreters of poliomyelitis virus.

The investigation covered a 2-year period from the spring of 1957 to the summer of 1959.

Altogether 28,797 specimens were examined of which 140 were positive for Type 1 virus, 24 for Type 2, and 44 for Type 3, the corresponding rates being 4·85, 0·83, and 1·53 per 1000 specimens respectively.

It is estimated that for every notified case of poliomyelitis there were about 140 symptomless excreters of Type 1 virus. No accurate estimates of this ratio were possible for the other two types, but it was certainly much higher.

The incidence of positive specimens was 15 per 1000 in the first and 2·6 per 1000 in the second year of the survey, but there were considerable local variations.

Type 2 virus was not isolated during the second year. Most specimens were examined for the presence of salmonellae and shigellae which both had a frequency of about 2 per 1000. The salmonellae were mainly *S. typhimurium*, and all the shigellae were *Sh. sonnei*.

Some information was obtained on the prevalence of other viruses. Coxsackie B virus was the most commonly isolated and in the second year was commoner than poliomyelitis virus. Its seasonal incidence was roughly the same as that of poliomyelitis virus.

Adenoviruses were isolated in about 6 per 1000 specimens and were most common in the winter. ECHO viruses were about half as common as adenoviruses and the maximum incidence was in the autumn.

The limitations of the sampling method used and the problems of analysis of the results are discussed.

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APPENDIX A

Public Health Laboratory Service Laboratories and Local Authorities taking part in Survey

Laboratory	Local authority	Period covered
Bath	Bath C.B.	1st and 2nd years
	Calne and Chippenham R.D.	1st year
Bedford	Ampthill U.D. Bedford M.B. Kempston U.D. Bedford R.D.	1st and 2nd years
Birmingham	Smethwick C.B. West Bromwich C.B. Sutton Coldfield M.B. Dudley C.B.	1st and 2nd years
Bradford	Bradford C.B.	2nd year
Brighton	Brighton C.B.	2nd year
Cardiff	Barry M.B.	1st and 2nd years
Carlisle	Carlisle C.B.	1st year
Chelmsford	Southend C.B.	2nd year
	Braintree U.D. Brentwood U.D. Chelmsford M.B. Witham U.D. Braintree R.D. Chelmsford R.D. Maldon R.D.	1st year
Colindale	Acton M.B. Ealing M.B. Edmonton M.B. Enfield M.B. Feltham U.D. Friern Barnet U.D.	2nd year
	Harrow M.B.	1st year
	Hayes and Harlington U.D. Heston and Isleworth M.B. Ruislip–Northwood U.D. Southgate M.B. Staines U.D. Sunbury-on-Thames U.D. Twickenham M.B. Uxbridge M.B. Wembley M.B. Wood Green M.B. Willesden Yiewsley and West Drayton U.D.	2nd year

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Laboratory	Local authority	Period covered
Derby	Derby C.B.	2nd year
	Alfreton U.D.	
	Belper U.D.	
	Heanor U.D.	
	Ilkeston M.B.	
	Ripley U.D.	
	Swadlincote U.D.	
	Blackwell R.D.	
	Repton R.D.	
	South East Derbyshire R.D. (formerly Shardlow R.D.)	
Dorchester	Dorchester M.B.	1st year
Exeter	Exeter C.B.	1st year
Guildford	Esher U.D.	2nd year
	Malden and Coombe M.B.	
	Walton and Weybridge U.D.	
	Woking U.D.	1st year
Hereford	Hereford M.B.	2nd year
Leeds	Leeds C.B.	1st and 2nd years
Leicester	Leicester C.B.	1st year
	Coalville U.D.	2nd year
	Loughborough M.B.	2nd year
	Wigston U.D.	1st year
Luton	Luton M.B.	1st and 2nd years
Maidstone	Ashford U.D.	1st year
	Beckenham M.B.	
	Bexley M.B.	
	Broadstairs and St Peter's U.D.	1st and 2nd years
	Bromley M.B.	1st year
	Chatham M.B.	
	Chislehurst and Sidcup U.D.	
	Crayford U.D.	
	Dartford M.B.	
	Deal M.B.	1st and 2nd years
	Dover M.B.	1st and 2nd years
	Erith M.B.	1st year
	Folkestone M.B.	1st and 2nd years
	Gillingham M.B.	1st year
	Gravesend M.B.	1st year
	Hythe M.B.	2nd year
	Maidstone M.B.	1st year
	Margate M.B.	1st and 2nd years
	Northfleet U.D.	1st year
	Orpington U.D.	1st year
	Penge U.D.	1st year
	Ramsgate M.B.	1st and 2nd years
	Rochester M.B.	1st year
	Royal Tunbridge Wells M.B.	1st year
	Sandwich M.B.	2nd year

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Laboratory	Local authority	Period covered
Maidstone (*cont.*)	Sevenoaks U.D.	1st year
	Swanscombe U.D.	
	Tonbridge U.D.	
	Whitstable U.D.	
	Bridge-Blean R.D.	
	Cranbrook R.D.	
	Dartford R.D.	
	Dover R.D.	
	Eastry R.D.	
	Elham R.D.	
	Hollingbourn R.D.	
	Maidstone R.D.	
	Malling R.D.	
	Sevenoaks R.D.	
	Strood R.D.	
	Swale R.D.	
	Tonbridge R.D.	
	West Ashford R.D.	
Manchester	Chadderton U.D.	1st year
	Heywood M.B.	
	Prestwich M.B.	
	Rawtenstall M.B.	
Middlesbrough	Middlesbrough C.B.	2nd year
Newcastle	Newcastle C.B.	1st and 2nd years
Nottingham	Nottingham C.B.	1st year
Plymouth	Plymouth C.B.	1st year
Portsmouth	Portsmouth C.B.	1st and 2nd years
	Fareham U.D.	2nd year
Salisbury	Salisbury M.B.	1st and 2nd years
Sheffield	Sheffield C.B.	1st and 2nd years
	Worksop M.B.	1st year
Stafford	Shrewsbury M.B.	2nd year
	Stoke-on-Trent C.B.	
	Walsall C.B.	
	Stafford M.B.	1st year
Swansea	Swansea C.B.	1st and 2nd years
Taunton	Wellington U.D.	1st year
Wakefield	Wakefield C.B.	1st and 2nd years
Winchester	Winchester M.B.	1st year
Worcester	Worcester C.B.	2nd year
	Bewdley M.B.	
	Bromsgrove U.D.	
	Evesham M.B.	
	Halesowen M.B.	
	Kidderminster M.B.	
	Malvern U.D.	
	Redditch U.D.	
	Stourbridge M.B.	
	Stourport-on-Severn U.D.	
	Bromsgrove R.D.	
	Droitwich R.D.	
	Evesham R.D.	
	Kidderminster R.D.	
	Pershore R.D.	
	Upton-on-Severn R.D.	

APPENDIX B

The underlying relationship between the proportion of carriers as detected by an instantaneous survey, and the incidence of new infections is given by the expression

$$I = P/T,$$

where P is the proportion of positives in the sample and T is the average carrier period in units of the interval over which sampling was carried out.

If now a proportion F of new infections become clinical cases, the notification rate for the period will be $F \times I$, if F is small compared with unity, so the notification rate R is given by

$$R = FP/T.$$

This formula is sufficiently accurate provided that sampling is at random through the year and that the average carrier period is constant. The slope of the line relating R to P is F/T, from which F may be calculated if T is known.

If more than one type of virus is prevalent and the proportions of the types are known in the sample, but not among the notifications, the relationship between the total notifications of all types and the observed fractions in the sample will be of the form

$$N = aP_1 + bP_2 + cP_3,$$

where a, b, c are constants proportional to the respective F's and P_1, P_2, P_3 are the proportions of the three types. An analysis by the usual multiple regression methods should, therefore, provide estimates of a, b and c from which values of the F's could be deduced. This approach is only approximate since the observations are not of equal weight and all the variables involved are subject to error. The first of these deficiencies will introduce random errors of unknown bias and the second causes the slope of the line to be systematically underestimated; however, the method was used because it is the most convenient and objective way of combining the results. It gives if anything an underestimate of the relevant constants, and the standard errors being based on the deviations from the fitted lines are of the right order of magnitude. Using this technique the regression coefficients for Type 2 and Type 3 are found to be not significantly different from zero, and it is concluded that the observations cannot provide an adequate estimate.

As a check on this method an alternative analysis was applied to the data from areas in which only Type 1 virus was isolated. The theory of this analysis was as follows.

Let S_i = the number of specimens collected in the ith area;
r_i = the number of positives;
I_i = the incidence of all new infections;
T = mean carrier period;
c_i = the number of notified cases;
N_i = the population of ith area;
F = the proportion of infections that become notified cases.

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Then since the incidence of both notified cases and positive specimens is small, their distribution approximately follows the Poisson distribution and the likelihood of the results for the ith area will be given by

$$e^L \propto \exp[-S_i I_i T] \frac{(S_i I_i T)^{r_i}}{r_i!} \exp[-F I_i N_i] \frac{(F I_i N_i)^{c_i}}{c_i!},$$

and the log likelihood for all the available data is

$$L = \text{const} - T\Sigma S_i I_i - F\Sigma I_i N_i + \Sigma r_i \log I_i + \log F\Sigma c_i + \Sigma c_i \log I_i.$$

So, differentiating with respect to F and the I_i and equating to zero, we have the estimates

$$F = \frac{\Sigma c_i}{\Sigma I_i N_i},$$

$$I_i = \frac{r_i + c_i}{FN_i + TS_i}.$$

These can easily be solved by guessing a set of I_i, calculating F, and then obtaining improved estimates of the I_i's. The convergence is rapid.

The errors of the estimates are obtained as usual, by differentiating once again with respect to the constants. Only the error of F is of interest and this turns out to be

$$\text{var}(F) = \left[\frac{\Sigma I_i N_i}{F} - \Sigma\left(\frac{N_i^2 I_i}{TS_i + FN_i}\right)\right]^{-1}.$$

Using this method the values of F for the two years of the survey were found to be about 0·007 in both the first and second years, the differences between them being quite insignificant. There is thus no evidence of any variation in virulence of Type 1 virus alone, and the value of F is very close to that obtained by regression.

The estimate of F is a weighted mean of the estimates in the individual areas, and is at least as reliable as that found by regression since it is more rationally weighted. Neither method can be regarded as giving more than an estimate of the right order of magnitude, which is all that the data are capable of providing.

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