POLIOMYELITIS IN AUCKLAND, 1947–1949.
AN EPIDEMIOLOGICAL STUDY*

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(With 12 Figures in the Text)

FOREWORD

In conjunction with its predecessor published last year, this report records an attempt—perhaps the first of its kind—to trace the course of an epidemic of poliomyelitis in all its manifestations, subliminal and overt. The Auckland district was ideal for the purpose. It has now been recognized that very large-scale studies involve so many doubtful factors of time and local circumstance that their epidemiological value is small. There is therefore a definite place for intensive field investigations of limited scope. The present inquiry is concerned with a population of about 350,000, in which there were nearly 350 positive cases, and perhaps 100,000 cases never definitely diagnosed. In its concentration on these last lies the chief interest of this inquiry.

A. W. S. THOMPSON

AUCKLAND,
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CONTENTS

I. INTRODUCTION

The Auckland poliomyelitis epidemic of 1947–9 commenced in November 1947. By the end of April 1949, it was approaching what is usually regarded as the saturation point for any extensive outbreak, 345 cases having occurred in a population (whole district) of just over 350,000, or almost 1 case per 1000.

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This paper is a sequel to a report* prepared about a year ago dealing with an investigation of the background of the early stages of the epidemic. It is a study of the further course of the epidemic in the light of the findings of that investigation.

II. RESULTS OF PREVIOUS INQUIRY

These may be briefly summarized:

(*a*) The epidemic had been preceded, and was accompanied, by large numbers of cases of minor illness, characterized by fever, headache, sore throat, vomiting and diarrhoea, and sometimes pains in the abdomen and neck. Evidence was produced to show that these were in reality minor forms of the more serious disease.

(*b*) The ratio of these 'suspect illnesses' to positive cases appeared to be higher than has generally been estimated elsewhere. The over-all ratio was about 300:1, but there was considerable variation between one age/sex group and another.

(*c*) The disease had already established itself widely, in the form of these 'suspect illnesses', before the appearance of positive cases revealed its presence. The facts would have been consistent with an increase in the virulence of the causative organism during the months of October and November 1947.

(*d*) One result of the investigation was to focus attention on the schoolboy aged 10–15 years, and to a lesser extent on the girl aged 5–10 years. The older schoolboy appeared to be the person most frequently responsible for introducing the infection into households in which positive cases later occurred. Before, or concurrent with, the onset of the positive case in any family, 50 % of the two age groups mentioned above had a 'suspect' illness, which appeared to afford them personal protection later.

(*e*) Study of the intervals occurring between successive illnesses (suspect or positive) in the same household pointed to a comparatively poor capacity of the organism to pass from person to person in the home. This, and other evidence, suggested that faecal organisms, rather than droplet infection, played the major part in propagating the disease. The suggestion was made that dust-borne infection might be an important means of spread, as is believed to be the case with threadworms.

III. COURSE OF THE EPIDEMIC

The Central Auckland Health District comprises an area of almost 1900 square miles and a population of about 350,000. In the centre of the district the city of Auckland has a compact population approaching 275,000. The Waitemata Harbour cuts off a portion known locally as the North Shore, and to the south of the city certain independent urban populations (Otahuhu, Papatoetoe, Papakura) are functionally very closely connected with its life. The remainder of the district is semi-rural, although there are numerous small urban aggregations scattered here and there.

The course of the epidemic is shown on the graph (Fig. 1), in which distinction has been drawn between the three main subdivisions of the district as described

* 'A Contribution to the Epidemiology of Poliomyelitis in New Zealand'. This *Journal*, 1948.

above. The initial peak occurred in December 1947, and a similar flare-up but with only half as many cases followed in January 1949. Between times, in the middle of 1948, there was a moderate build-up of cases affecting all areas. The similarity of the curves for the city area and for the semi-rural districts outside is interesting. Most of the early notifications were from Auckland itself, but Papakura, 19 miles to the south, and the remote country district of Hunua, 30 miles to the south-east, produced cases at the very beginning.
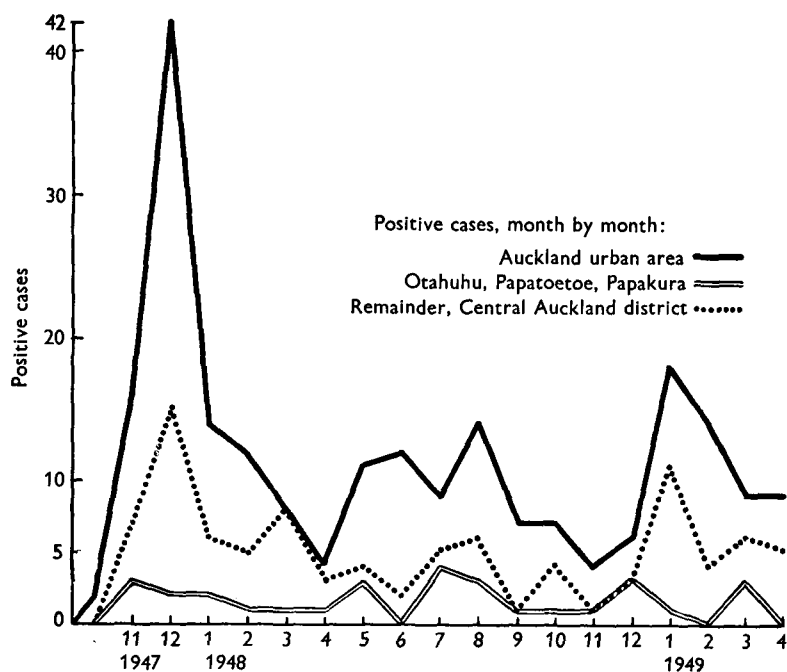

Fig. 1. Course of the epidemic, Central Auckland Health District (see Appendix, Table 6).

IV. INCIDENCE IN DIFFERENT AREAS

The graph gives a misleading impression of the relative intensity with which these areas were affected. By the end of April 1949, a total of 345 cases had occurred, equal to 9·8 per 10,000 population. They were distributed as follows:

(a) Auckland urban area: 218 cases, or 8·0 per 10,000.

(b) Otahuhu, Papatoetoe, Papakura: 30 cases, or 21·5 per 10,000.

(c) Remainder (semi-rural): 97 cases, or 15·4 per 10,000.

It will be seen that the incidence was almost twice as great in the semi-rural areas as in Auckland itself, but that the urban areas lying between city and country were affected more heavily still.

The next illustration (Fig. 2) shows this in greater detail. It covers the period to the end of 1948 only. Cases are shown by age groups.* The incidence in Papa-

* Figures for age and sex composition of these populations are not available. Cases in each age group have therefore been related to the total population in each area. In other words, the graphs have been constructed as if the populations concerned were equally distributed between the eight age/sex groups. The principal effect is to exaggerate the importance of cases amongst 'over 15's'.

kura was remarkable, 5 cases per 1000. In form generally the figures for the different areas have little in common, but there is one consistent feature which is of considerable interest. Scrutiny will show that as the incidence increases from Auckland, through the semi-rural districts, through Otahuhu and Papatoetoe, to its climax in Papakura, so the length of the column for the 5–10-year-olds in either sex grows too. In other words, there appears to be a positive relationship between the incidence as a whole in any area and the incidence in this particular age group. Children of this age are peculiarly situated in regard to infection and immunity. No child under the age of 10 in 1948 can have passed through the 1937 epidemic; above that age the majority must have had some opportunity of acquiring
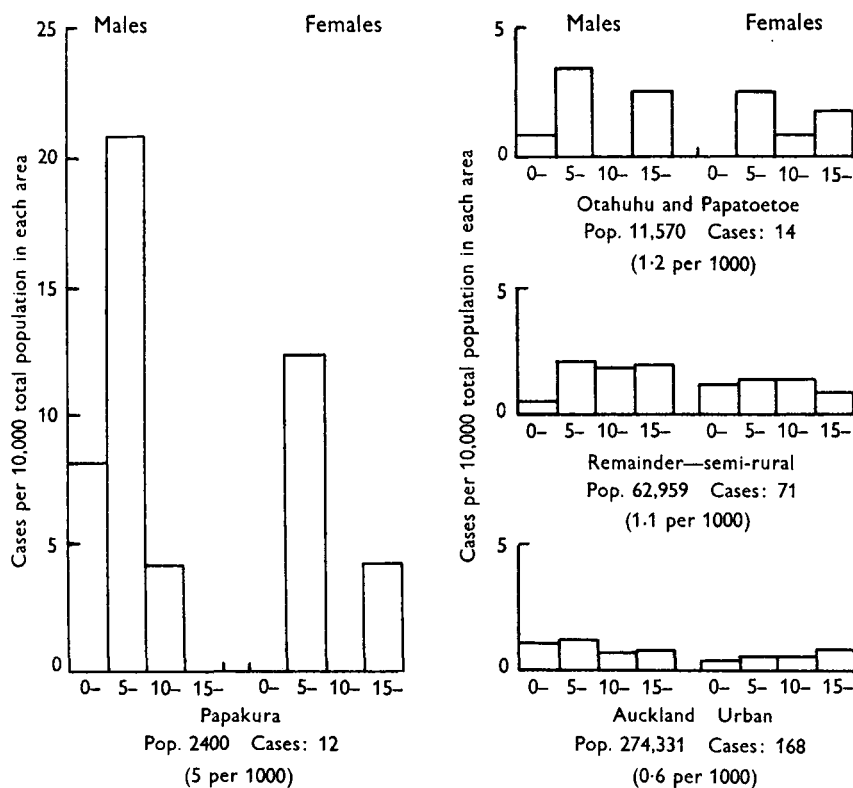
Fig. 2. Incidence in different parts of district: cases per 10,000 total population in each area to 31 December 1948 (see Appendix, Table 7).

immunity at that time or in previous outbreaks. The pre-school child, however, has relatively few opportunities of becoming infected at any time. If, therefore, we find that the 5–10-year-old group produces few cases during the epidemic, the presumption is that it must have gained immunity during the preceding inter-epidemic interval. Study of Fig. 2 therefore suggests that a lighter general incidence in some areas than others may be due to more effective circulation of the virus in these areas between epidemics, and that the converse is true of areas which suffer heavily.

This explanation seems plausible enough when we compare a densely populated

7-2

area like Auckland, where free circulation of infection is probable at all times, with country districts, where there must normally be little interchange of infecting agents. But what of the towns lying between city and country—Otahuhu, Papatoetoe, Papakura—where germ exchange must be considerable, and yet the toll of the epidemic has been highest of all?

The answer will be found, I think, in the history of these places. Between 1936 and 1945 their aggregate populations increased by 39 %, and they are still growing rapidly. It seems probable that the greater part of this growth is due to influx not from other urban districts but of people from country areas.

If these people with the low immunity of rural dwellers were subjected to the rapid circulation of a virus at epidemic virulence before they had had time to acclimatize themselves to town life, one would expect the incidence of positive cases amongst them to be high.

The suggestion, then, is that what happens to any population during a poliomyelitis epidemic depends largely on the degree of immunity acquired during the preceding 'silent' interval. This, in turn, I have assumed to be dependent on population density. When the circulation of a virus is a matter of only a small proportion of individuals being affected at a time, even minor differences of population density might influence its range. During an epidemic, however, the proportion of the population responsible for spreading the virus must increase enormously; this is what we should expect, and a similar process has actually been demonstrated during epidemics of cerebrospinal meningitis. In a prolonged epidemic like the present, therefore, it is hardly likely that any ordinary degree of density variation could influence the total number of persons ultimately affected by the virus; but the number reacting unfavourably (positive cases) should be smaller in dense areas than in more sparsely populated districts, at least up to a point. If, however, dispersal is carried to extremes, sooner or later a state must be reached where mere distance between families prevents effective circulation of the virus, even under epidemic conditions.

Let us put the theory to the test, beginning with the broad divisions of the district mentioned above:

	Persons per acre	Cases per 10,000 (to 30 April 1949)
(a) Auckland urban area	6·0	8·0
(b) Otahuhu	5·6	13·2
Papatoetoe	3·2	17·5
Papakura	1·2	58·3
(c) Remainder (semi-rural)	0·05	15·4

It will be seen that as the density falls, the incidence rapidly increases, until we come to the very sparsely populated rural districts, when the incidence drops again. The result is so neat that the reader may wonder why I have bothered to drag in the hypothesis that an influx of country dwellers has boosted the incidence in group (b). The reason is that it would clearly be absurd to pretend that density reductions of the order shown could alone account for such enormous differences in incidence. Other factors must obviously be at work.

V. RELATIONSHIP BETWEEN DENSITY AND INCIDENCE

Let us look more closely into this question. To get dependable results we should require figures for a large number of areas similar in all respects except in density. The nearest approach at our disposal consists of the Auckland urban area, less the North Shore, with Otahuhu (which is virtually part of Auckland) thrown in. This area is divided into eleven subdivisions (see Fig. 4) of varying density. Functionally the population concerned is fairly homogeneous, but unfortunately there are many differences, socially and developmentally, between one area and another, and it cannot be held that the requirements mentioned above have been completely fulfilled.
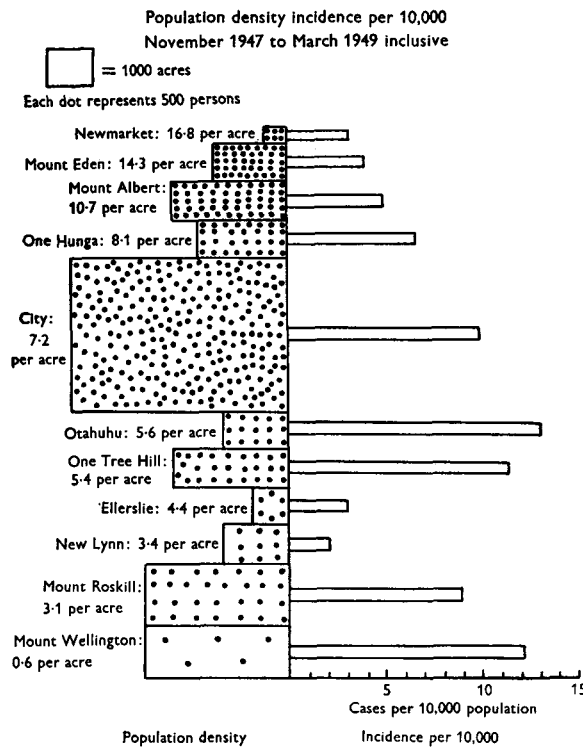
Fig. 3. Relationship between density and incidence: cases in Auckland urban area (less North Shore) and Otahuhu to 31 March 1949 (see Appendix, Table 8).

The next illustration (Fig. 3) shows, on the left, the area and population of each subdivision, and on the right the incidence of positive cases to the end of March 1949. An inverse relationship between density and incidence is remarkably well demonstrated down to and including One Tree Hill, especially if we accept that Otahuhu's incidence may have been boosted by influx from the country. Beyond that something appears to have gone wrong. That Ellerslie and New Lynn should have escaped so lightly seems very strange; though relatively sparsely populated, they show incidences such as we should have expected only from the heart of the city.

Ellerslie would not have puzzled a racing-man for so long as it puzzled me.

I have been told (by an Aucklander) that its racecourse is the most important in New Zealand. Several times each month the actual density of persons within Ellerslie's confines is intense. For this reason its proper place is probably at the head of the column, and its low incidence is in accord.

New Lynn is not so easily accounted for; in its case the story is perhaps not complete. (Two of the seven cases notified in April in the area now being discussed were from New Lynn, bringing its incidence to 6·3 per 10,000; this was the only area significantly affected during April.) It is a district, however, whose expansion (23 % between 1936 and 1945) has been largely due to an influx of urban dwellers from the city, and it differs sharply in this respect from the heavily affected towns on the southern side.

It is at least arguable, therefore, that these two apparent exceptions are not really exceptions at all, and that, in fact, their closer study lends support to the theory. If we ignore them for the moment, the other areas of low density (Mount Roskill and Mount Wellington) may perhaps be regarded as showing the early effects of increasing dispersals in eventually curtailing the incidence.

If the reader will look at Fig. 3 he can imagine Papatoetoe (3·2 per acre) just below New Lynn, with its incidence bar (17·5) projecting just below the edge of the diagram; and Papakura (1·2 per acre) second from the bottom, with its bar (58·3) projecting right off the page, more than three times as long. It is obvious that while they follow the same general tendency as the others, in their cases the effect is widely exaggerated, probably for the reasons mentioned above.

If Fig. 3 is held at arm's length, the eye gets some impression of the actual differences in density between the areas concerned. It is admittedly not easy to believe that variations of density of the order shown between the seven most closely settled areas could produce the effects attributed to them. But it would be even more difficult to believe them due to chance or to the operation of some factor unrelated to density.

VI. PREVIOUS EPIDEMICS AND 'SILENT' INTERVALS

It is difficult from the records now available to arrive at strictly comparable figures, but the following is an estimate of the incidence in the Central Auckland District during the three previous major epidemics:

1916	15·5 per 10,000
1925	7·8 per 10,000
1937	2·6 per 10,000

Between these years the disease was constantly active in New Zealand. In the Auckland Province during the 11 years intervening between the epidemics of 1925 and 1937 there were 90 cases, an average of 8 per annum. Following on the 1937 outbreak, however, there was a significant lull. Details are lacking for the first 3 years, but here is the record for the Auckland urban area and Otahuhu from 1941 on:

1941	Nil	1944	Nil
1942	1 case	1945	1 case
1943	Nil	1946	5 cases (plus 1 just outside)

The lull ended with a crash, the new epidemic commencing earlier in the season than ever before and producing more cases in a month than the last epidemic cast up in its full course. The incidence to date, 9·8 per 10,000, is sufficient to put it in a different class from the outbreaks of 1925 and 1937. If we consider the successively declining toll of the first three epidemics and the continuing activity between them, it becomes clear that the 1937 affair was merely the final movement of a composition which began in 1916, and that in 1947 we were in the presence of a new opus.
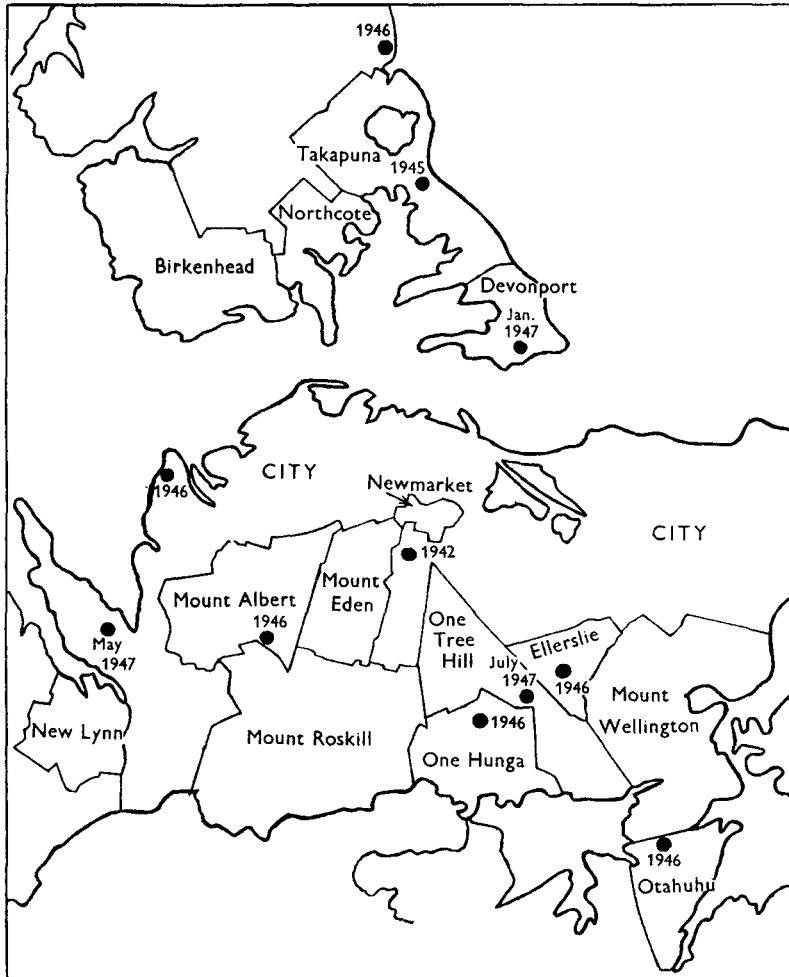
Fig. 4. Auckland urban area and Otahuhu: location of positive cases from 1942 to July 1947.

It would be interesting to know what was really happening during the lull, which can hardly have been a period of complete inactivity. Fig. 4 shows the location of the positive cases which came to light after 1940. They were pretty well dispersed. The two most densely, and the three most sparsely, populated of the districts shown in Fig. 3 produced no cases; the middle group had 1 each, except for Auckland city, which had 3. The 3 cases recorded in the early part of

1947, before the epidemic began, were also widely separated; similarly with the 6 occurring in 1946.

If our theory is correct, all this time a furtive but beneficent process was going on, in which an attenuated virus was passing from person to person and silently conferring immunity. This process, it would seem, was most effective in the more densely populated areas—in Newmarket, Mount Eden and Mount Albert, for example. This could only be the case, however, so long as the number of spreaders, and perhaps the dispersive powers of the virus, remained low. Once they increased beyond a certain point, the differences between areas must be reduced to insignificance.
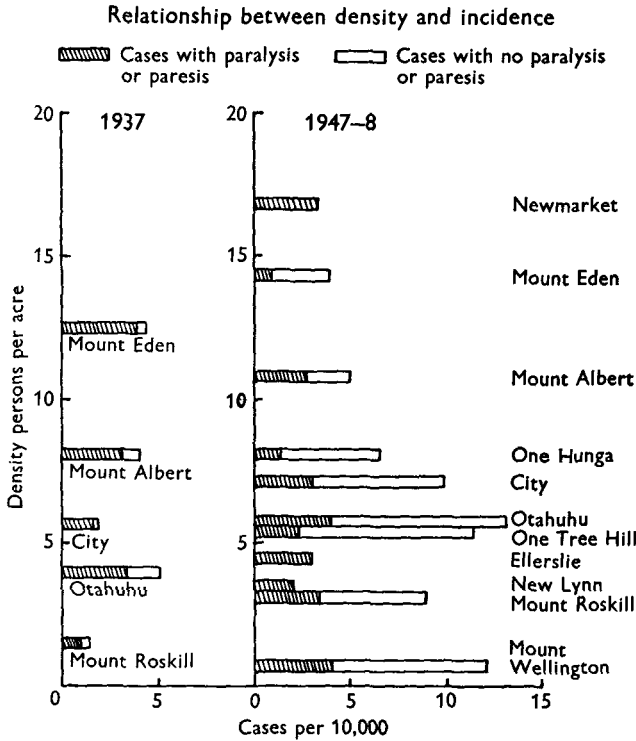
Fig. 5. Density and incidence in two epidemics (see Appendix, Table 8). (N.B. 1947–9 epidemic: positive cases to 31 March 1949; cases with paralysis or paresis to 31 December 1949.)

The results, if the virus increased in virulence as well as in dispersiveness, must now be very different. Those who had previously been visited might respond with a minor illness, or not at all. In areas where there had previously been little circulation, however, the path of the new invasion would be strewn with positive cases. These alone find their way into hospital and appear in the statistical returns; of the minor reactions we normally hear nothing.

Precise records being available for the 1937 epidemic (but not, unfortunately, for previous ones) it has been possible to plot incidence against density for the 43 cases which occurred in the areas dealt with in Fig. 3. In Fig. 5 the result is compared with the position in the present epidemic. It will be seen that in the

four Auckland areas affected, incidence in 1937 was related directly to density, the opposite of our experience now. This diagram also distinguishes cases with paralysis or paresis from those without either, in case an altered standard of diagnosis should have obscured the issue. This does not help very much, however, the effect in the case of the present epidemic being complete confusion.

There are three possible explanations of this anomaly:

(*a*) The apparent inverse relationship shown in the latest epidemic may be false. Such a relationship has been noted elsewhere,* however, and the results are too consistent to be lightly dismissed. If anything, the 1937 diagram is less likely to be reliable in view of the small number of cases involved.

(*b*) The very small proportion of non-paralytic cases in the 1937 record suggests that the picture may be incomplete. Present standards of hospitalization might have told a different story.

(*c*) It has been suggested above that the 1937 outbreak was not in reality an epidemic complete in itself, but merely a last flare-up in a process which began much earlier. The tail end of an epidemic might well be irregular. As I have already pointed out, when the spreading process is rapid the effect of variations of density must be slight or nil. If most of those who escape in the closing phases of an epidemic are persons who have gained immunity during the earlier stages, one would expect to find most signs of activity in the regions least affected when the process was at its height. Imagine a shower of sparks continually falling on heaps of materials, some of which are much less inflammable than others; at the end only the heaps which burn least readily will be alight.

The last seems the most likely explanation.

VII. SPREAD IN THE AUCKLAND URBAN AREA

In my previous paper spot maps were presented showing that in the early stages of the epidemic there was nothing to indicate spread from any particular focus. The next four diagrams (Figs 6–9) show the further course of the epidemic in the urban area†. Cases in residential institutions have not been included.

A certain ebb and flow over the area is apparent, but no tendency to centrifugal spread. The pattern of the last fifty cases is not unlike the first. The story is one of victims struck down at random almost simultaneously, and a battle fought to a finish without shifting ground. When poliomyelitis comes, it comes not single spies, but in battalions; not uniformed troops, but as a fifth-column, quietly infiltrating; then suddenly, here and there, the guise of a minor illness is thrown off, and paralysis is in our midst.

Some of the most popular beaches in the Auckland district are situated along the coast-line shown near the top right-hand corners of these diagrams. Auckland's main sewer discharges at a point about a quarter of the way along from the right-hand border of the diagram, and beaches crowded with bathers in the summer

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* In London in 1947. Sir Allen Daley and B. Benjamin (1948), *The Medical Officer*, **80**, 171.

† Figure 6 corresponds to Fig. 1 (1) in the previous paper. Some minor corrections have been necessary.

months stretch for a couple of miles to the east, beginning not far from the outfall. The sea water at these beaches has been heavily polluted for years. It is interesting to note that although the contiguous areas are quite densely populated, and the local inhabitants probably use these beaches more than anyone else, the distribution of poliomyelitis cases in this neighbourhood is very sparse indeed. An unbiased observer might even say of these diagrams that it looked as if this area
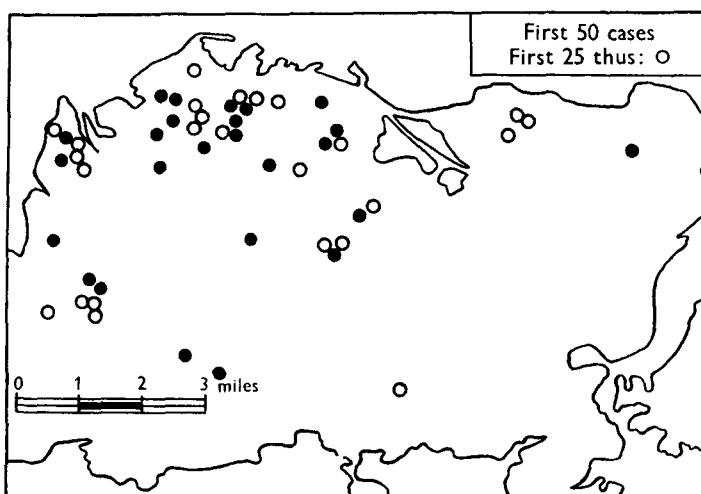
Fig. 6. Location of first fifty cases in the metropolitan area (date is of notification).
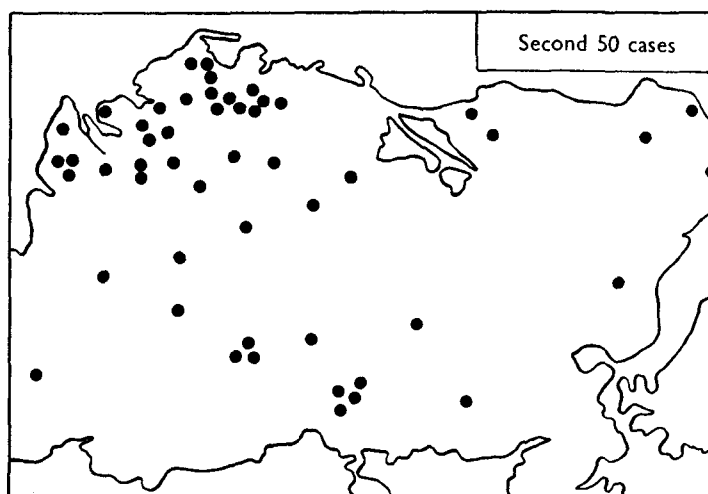
Fig. 7. Location of second fifty cases in the metropolitan area (date is of notification).

had been specially favoured in its avoidance by the virus. This is interesting in view of the fact that in the earlier stages of the epidemic certain interested parties made efforts to throw the entire blame for the outbreak on the state of the harbour, arguing (incorrectly) that poliomyelitis cases had been commonest near the beaches.
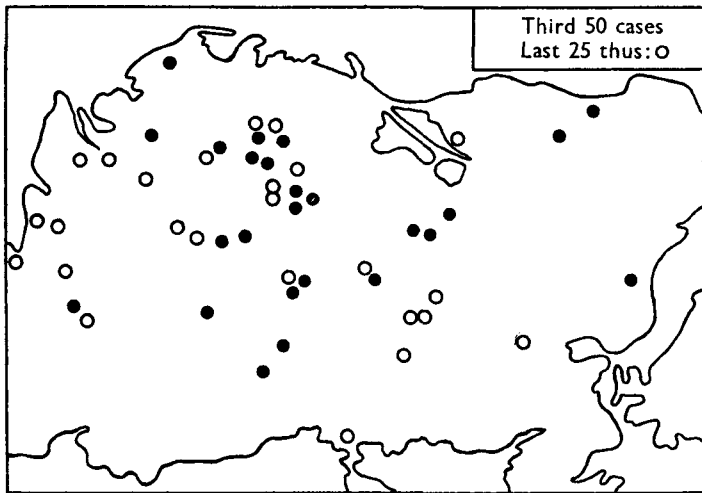
Fig. 8. Location of third fifty cases in the metropolitan area (date is of notification).
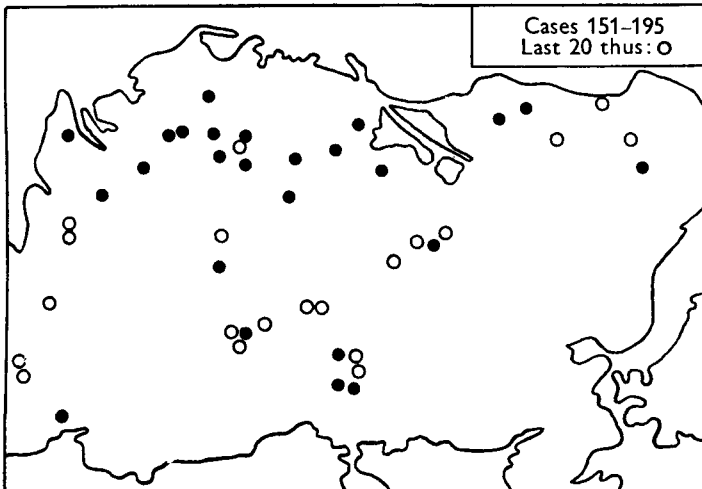
Fig. 9. Location of cases nos. 151–195 in the metropolitan area (notified 6 January 1949 and 30 May 1949 respectively).

VIII. SEVERITY OF DIFFERENT AGES

In the previous inquiry an estimate was obtained of the ratio of minor illnesses related to poliomyelitis ('suspect' illnesses) to positive cases. Separate estimates were made for different age/sex groups, as follows:

Ratio of suspect illnesses to positive cases

Sex	Age	Calculated ratio	Round figures
Males	0–	112 : 1	100 : 1
	5–	99 : 1	100 : 1
	10–	319 : 1	300 : 1
	15 and over	546 : 1	500 : 1
Females	0–	203 : 1	200 : 1
	5–	230 : 1	200 : 1
	10–	168 : 1	200 : 1
	15 and over	972 : 1	1000 : 1
Total		301 : 1	300 : 1

These results were based upon the incidence of 'suspect' illnesses recorded amongst families in various parts of the city from November 1947 to February 1948 inclusive (applied by calculation to the urban area as a whole), and the incidence of poliomyelitis over the same period. The latter included all positive cases admitted to hospital. The proportion of cases with paralysis or paresis was not taken into account and at that time was not known. As can be seen from Fig. 5, the relationship of cases with paresis to total notifications is, in any case, extremely irregular.

As no further investigations have been made into the occurrence of minor illnesses in the general population, the accuracy of these estimates cannot be checked directly. An indirect test of limited value is available, however.
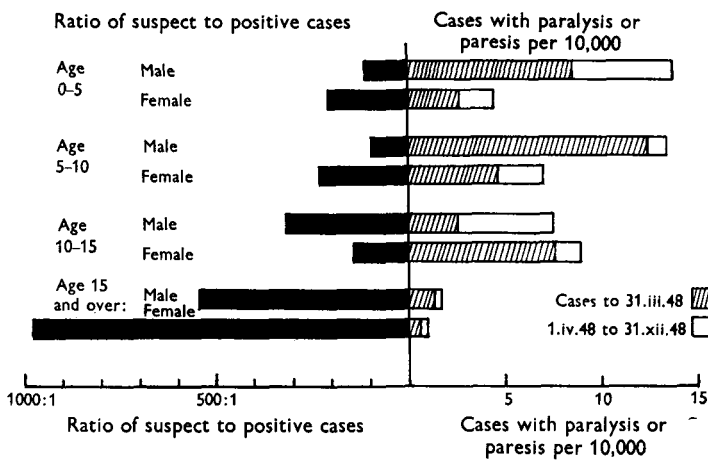
Fig. 10. Severity in various age groups: relationship between ratios of 'suspect' to positive cases, and incidence of paralysis or paresis (see Appendix, Table 10).

This consists of a comparison between the incidence of cases *with paralysis or paresis** in each age group with the ratio of suspect to positive cases as previously estimated. A high ratio of suspect to positive cases in one age group and a low ratio in another suggests that in the first age group the disease is less

* Fig. 5 shows that the proportion of positive cases with paralysis or paresis varies enormously, and irregularly, between different localities. The irregularity is too gross to be accounted for by differences in age/sex composition, although wide variations also occur between the age groups. Cases to the end of 1948 from Auckland itself included the following percentages with paralysis or paresis:

	Percentage of cases with paresis		Percentage of cases with paralysis	
	(%)	(%)	(%)	(%)
0–	50	23	7	23
5–	29	24	6	12
10–	11	31	21	19
15–	27	33	45	28

It might be thought that the presence or absence of paralysis (or paresis) would be a more reliable criterion for statistical purposes than a clinical diagnosis on general grounds, but such does not seem to be the case. Epidemiologically paralysis is a mere accident, unpredictable, and totally unreliable as a standard of comparison.

severe than in the second. We should therefore expect to find relatively fewer paresed cases in the former age group.

Fig. 10 shows this comparison graphically. On the right-hand side incidences of paresed cases are shown from the beginning of the epidemic to the end of March 1948 (end of the initial surge, and of the period covered by the previous inquiry), and from then until the end of the year. It will be seen that in each age group the higher the ratio of suspect to positive cases, the lower the incidence of paralysis or paresis. The diagram also shows that the severity of the disease is greater in males than in females, except in the age group 10–15 years, when the opposite is the case.

Some confirmation is therefore forthcoming in support of ratios of suspect to positive cases arrived at in the previous inquiry. In that it is based on figures from a much longer period, and on data not available when these estimates were made, it is of interest, despite its admittedly doubtful reliability.

IX. PERCENTAGE AFFECTED IN DIFFERENT AGE GROUPS

By applying these ratios to the cumulative totals of positive cases in each age group, it is a simple matter to calculate the total number of persons who must have had the disease in one form or another since the beginning of the epidemic. If we also know the population in each age group in the area, we can discover what percentage of the total has been affected to date.

The accompanying diagrams (Figs. 11 and 12) have been constructed accordingly. They apply only to the Auckland urban area. At the bottom of each chart the cumulative totals of cases, male and female, are shown month by month. The graphs indicate the presumptive progress of the epidemic in the age group as a whole. The reader will see at once, on glancing at Fig. 12, that this report carries the story to a most interesting and critical point. One age group, that of boys aged 10–15 years, had just touched 100 % by the end of April 1949.

The steady progress to saturation point of the male group aged 10–15 years contrasts sharply with the behaviour of the disease in girls of the same age. In their case a tendency to settle down about the 40 % mark was evident as early as May 1948.

In the pre-school groups there were more positive cases in males than in females (34 males, 21 females), but it is probable that the percentages affected by poliomyelitis in some form or another was identical. By the end of April 1949, about one-third had been attacked.

Amongst 5–10-year-olds progress was remarkably steady throughout, especially in girls. The graph for female percentages begins at a lower level and ascends more steeply than that for males. In this age group I do not think that the apparent differences between them are illusory.

The diagram for ages 15 and over has been omitted, being of little interest. By the end of April 1949, thirty males cases and nineteen female cases had occurred in these age groups. By then the calculated percentages affected amounted to 16 and 17 % respectively. Throughout most of the epidemic the female percentage was higher than the male, but they ran together at the beginning of 1949. It is seldom

appreciated that this age group comprises nearly 80 % of the total population. They contributed only 22 % of the positive cases in the Auckland urban area.

To summarize, Table 1 shows the calculated percentages of each age group which had been affected by poliomyelitis, whether 'suspect' or positive, by the end of April, 1949.
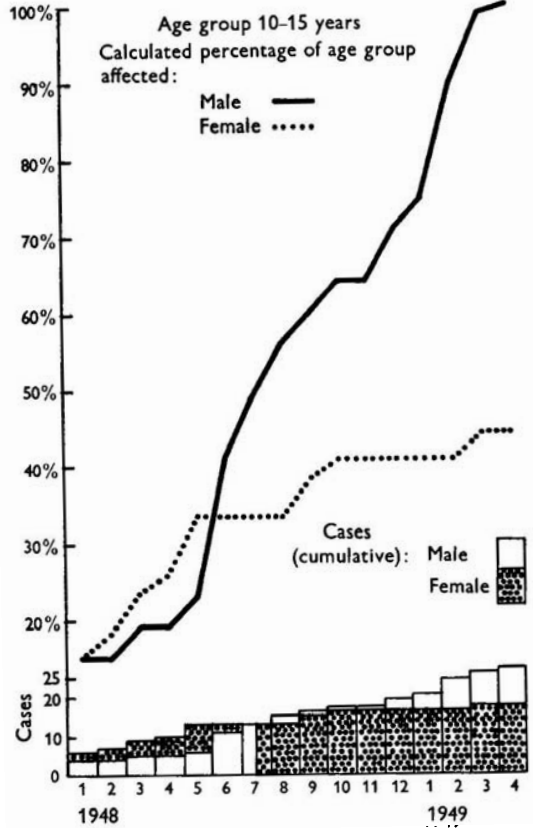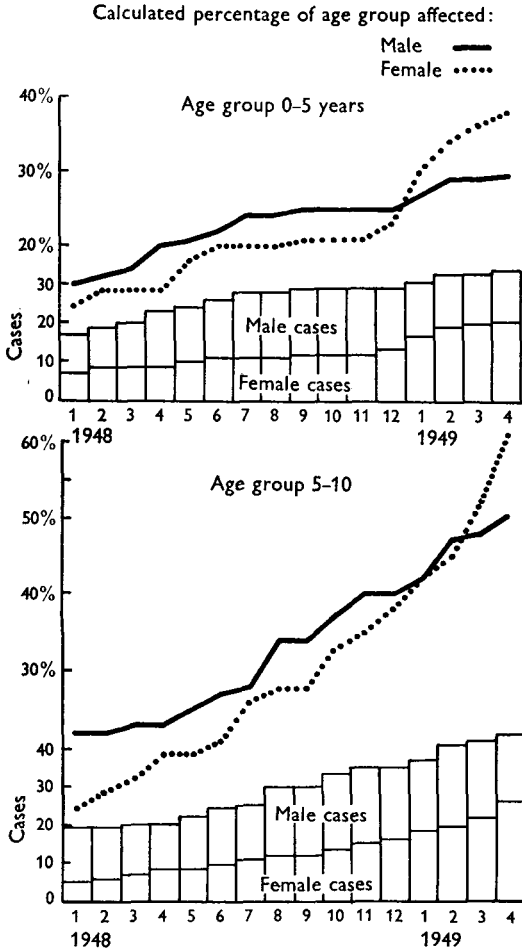
Fig. 11. Auckland urban area: progress of the epidemic in the younger age groups (see Appendix, Table 9).

Fig. 12. Auckland urban area: progress of the epidemic at ages 10–15 years (see Appendix, Table 9).

Table 1. *Calculated percentages of age groups affected by poliomyelitis, 'suspect' or positive, by the end of April* 1949

	Age 0–5 (%)	Age 5–10 (%)	Age 10–15 (%)	Age 15 and over (%)
Male	29	50	100	16
Female	38	61	44	17

X. COMPOSITION OF FAMILIES OF POSITIVE CASES

Multiple cases were admitted to hospital from five families in the Auckland urban area and from six families living elsewhere in the district. In each of these instances there were two cases, except for one family in a rural area which produced three. Several other instances came to notice in which undoubted clinical cases occurring amongst the family contacts of positive cases were not admitted to hospital and do not therefore appear in the published figures.

In the earlier investigation forty households in which positive cases had occurred were observed over a period of about three months. During this time a second positive case occurred in four different houses and 43 % of the remaining contacts had an illness which (for reasons explained in detail in the report) might be regarded as poliomyelitis in a mild form. Six seen during the attack were definitely diagnosable on clinical grounds, but were not admitted to hospital.

It appears, therefore, that multiple family illnesses related to poliomyelitis must be far commoner than hospital returns alone would lead us to believe.

Visiting the homes of positive cases in the early stages of the epidemic left one with two impressions about them: (*a*) that better-class homes appeared to predominate; (*b*) that these households included schoolchildren more frequently than the average family.

The first, about the class of family affected, was undoubtedly correct. Part V of this paper, showing an inverse relationship between density and incidence, confirms it.

The second appears to be wrong. An analysis was made of the composition of families in which positive cases had occurred, and of families including children of the same age group in the general population. (The latter were the families visited in the house-to-house inquiry during the previous investigation.) No significant difference could be demonstrated. Amongst pre-school children affected by poliomyelitis the percentage of only children was rather low (9·3 %, as against 16·8 % in the general population) and 69·8 % were in families which included schoolchildren, as against 64·3 % in the general population. In neither case was the difference statistically significant. The families of poliomyelitis cases aged 5–10 years appeared to be quite typical of those including children of this age in the population generally. It was not even possible to show that boys aged 10–15 years were any more likely to figure in the background of positive cases than in the average family including children.

This failure to demonstrate any peculiarity in the composition of the families of positive cases was rather surprising. It is another indication of the extraordinarily widespread nature of the subliminal epidemic, whose ramifications we are, of course, unable to follow directly. In the early stages of the outbreak the pre-school child's comparative isolation may, perhaps, afford some protection; but as time goes on it seems as though he is just as likely to pick up infection from his parents, or from the children outside the home, as to have it brought to him from school by an older brother or sister. It is interesting to recall that the earliest evidence of the epidemic found during the previous investigation was an

outbreak of suggestive illnesses in *adults*, especially adults in families without children. This finding was so unexpected that it was felt to be of doubtful significance, and reasons were mentioned why too much reliance should not be placed on it. I am inclined to a rather different view now.

XI. INCIDENCE IN SCHOOLS

In September 1948, all schools in the Auckland area were visited by Health Inspectors, who called attention to sanitary defects, and prepared reports giving details of numbers on the roll and of the closet and ablution facilities provided. These schools can therefore be classified according to the proportion of water-closets or hand-basins available. In addition, lists can be given of schools which, judging by the Inspectors' remarks, made a particularly good impression generally, and those in which sanitary defects were specially noted.

It is obvious, however, that one school may have a smaller number of hand-basins, for example, than another school of equal size, and still be less hazardous from the sanitary point of view. A moment's thought will show that ratios of sanitary accommodation to pupils are more reliable as a general index of over-crowding than of anything else. If there is any truth in the theory put forward in my previous report that the virus of poliomyelitis may be dust spread, especially through contaminated particles in the dust of school closets, then the school with the smallest proportion of closets per hundred pupils should provide the heaviest concentration of virus, other things being equal. But 'other things' never are equal; the school with the best accommodation is sometimes the worst maintained. Similarly, the number of hand-basins in a school is no indication of the amount of hand-washing done by the pupils.

Keeping these reservations in mind, let us glance at the incidence of poliomyelitis in different types of schools, classified according to the sanitary reports. Confining attention to the Auckland urban area less the North Shore, detailed reports are available for 72 schools, totalling 29,742 pupils. The following analysis is concerned with 77 positive cases in these schools, an incidence of 2·6 per 1,000.

Of the 72 schools reported upon, cases only occurred in 41, totalling 20,992 pupils, an incidence in the affected group of 3·7 per 1000. In Tables 2–5 the incidences in the affected schools, and in the school populations as a whole, are shown separately.

The populations concerned, and the numbers of positive cases, are so small that very wide variations would have to be recorded before the results from an analysis such as the above could be accepted as significant. A statistician would not, in fact, be impressed by any of the differences in incidence shown in these tables, except possibly by some of those in Table 5. It is nevertheless interesting to note that the incidences recorded are rather higher, in every case except one, in the group of schools with the best accommodation than in those which were worst. The only valid conclusion from this analysis is, however, that it provides no evidence that differences in standards of sanitary accommodation (and, by inference, overcrowding) have any influence on the incidence of poliomyelitis in schools.

Table 2. *Sanitation: general impression*

Inspector's remarks	Schools		Roll numbers			Incidence per 1000	
	Affected	Total	Affected schools	Total	Cases	Affected schools	Total
Good	4	6	2,244	2,755	7	3·1	2·5
No comment	29	52	14,089	20,335	57	4·0	2·8
Bad	8	14	4,659	6,652	13	2·6	2·0

Table 3. *Hand-basins*

Scale	Schools*		Roll numbers			Incidence per 1000	
	Affected	Total	Affected schools	Total	Cases	Affected schools	Total
1: 30 or more	9	19	4,666	7,261	17	3·6	2·3
1 : 31 to 1 : 49	9	17	4,830	7,492	22	4·5	2·9
1 : 50 or less	12	18	6,704	8,563	18	2·7	2·1

* Details lacking for 18 schools, including 11 affected schools.

Table 4. *Water-closets: boys*

Scale	Schools		Roll numbers			Incidence per 1000	
	Affected	Total	Affected schools	Total	Cases	Affected schools	Total
1 : 30 or more	13	35	2,570	6,210	18	7·0	2·9
1 : 31 to 1 : 40	9	17	2,977	4,629	17	5·7	3·7
1 : 41 or less	7	5	2,453	4,093	15	6·3	3·7

Table 5. *Water-closets: girls*

Scale	Schools		Roll numbers			Incidence per 1000	
	Affected	Total	Affected schools	Total	Cases	Affected schools	Total
1 : 15 or more	6	18	1,555	2,878	9	5·8	3·1
1 : 16 to 1 : 20	6	21	1,557	4,140	7	4·5	1·7
1 : 21 to 1 : 30	3	17	1,094	4,624	3	2·7	0·7
1 : 31 or less	7	11	2,504	3,168	8	3·2	2·5

XII. DISCUSSION

This report has traced the course of the epidemic to a crucial point. The incidence as a whole has touched, or almost touched, the 1 per 1000 mark. If my calculations are correct, one age group—that of boys aged 10–15—has just reached saturation. What happens now?

The answer will be known by the time the report is published, but early in May the writer was rash enough to state publicly that 'he would be surprised if, a month from now, the epidemic in the Auckland district was not virtually at an end'.*

My previous study focused attention on the older schoolboy. It was he who was most liable to introduce infection into the home. He tended to react to it himself at an early stage, getting the disease over quickly in its milder form. By the time

* This prediction proved correct. Notifications (whole district) in the succeeding five months were: 4, 4, 6, 3, 3; hardly an epidemic prevalence for a population of 350,000.

the first positive case came to light in the household, half the boys of this age had already had a 'suspect illness' and were immune from further trouble. To a lesser extent the schoolgirl aged 5–10 years old was similarly implicated.

Some may think that the conclusion that practically every boy of this age in Auckland has either been in hospital with poliomyelitis or has had a 'suspect' illness' during the present epidemic is absurd. Most of them must have been in Auckland during the last epidemic. Surely many must have gained immunity then?

The answer is: Very probably; if not then, certainly at some time during the intervening years. Many of these 'suspect illnesses' must in reality be minor reactions in persons who have had a previous attack—which, of course, will usually have been a minor one too. These suspect illnesses are usually very mild, although recognizable when the observer is on the look-out for them and knows there is poliomyelitis about. Details will be found in my previous paper.

Consider the case of another virus disease, vaccinia. Nowadays for purposes of international certification no such thing as a negative reaction is recognized. A person who has never been vaccinated before normally reacts violently; the result is called a 'typical primary vaccinia'. Revaccination of one who has been successfully vaccinated before usually results in no more than redness and swelling along the scratch, the so-called 'reaction of immunity'. Revaccination after a very long interval sometimes produces an accelerated reaction, similar to primary vaccinia, but seldom so upsetting.

It seems to me that in poliomyelitis we meet with equivalents of all these types of reaction, but with a different emphasis. In vaccinia the virus is one whose virulence, though attenuated, is not allowed to fall below a certain level. In poliomyelitis it would appear that the normal condition of the virus, except during an epidemic, is a state of low virulence. Most people when attacked react in a manner akin to the 'redness and swelling' of the reaction of immunity in vaccinia.* Occasionally even during inter-epidemic times someone throws a pukka reaction and comes to notice as a 'sporadic' case; various factors might account for this— abnormally low resistance, heavy dosage with the virus, some intercurrent condition which aids the virus, or its own temporarily exalted virulence. When an epidemic occurs, three types of reaction are seen in varying proportions. Those who have never been infected before react to the virulent organism by developing the disease in classical form—a 'typical primary reaction'. Those with limited immunity from a minor attack a long time before find themselves in hospital as 'positive cases' with little or no paralysis or paresis (comparable with the accelerated reaction). Those with good immunity have a minor illness of limited duration, like the well-protected person who on being revaccinated develops a 'reaction of immunity'.

To take the analogy a little further, everybody knows that primary vaccination

* I am aware, of course, that the latter is now regarded as being merely a sensitivity reaction to the lymph which can have no real equivalent in poliomyelitis. Nevertheless, a true reaction to the virus probably occurs also, even though it may not be the reaction which we see.

in later life is much more severe in its effects than vaccination in childhood. I think the same thing happens in poliomyelitis. For some reason the tissues of the adult react more violently to first contact with some viruses than do the tissues of a child. The average age at death of the fifteen fatal cases in the Auckland district was 22 years; two-thirds were aged 20 or older, although less than one-fifth of all cases came from this age group.

The cycle of poliomyelitis in the community may be regarded as a process of active immunization with a living but attenuated virus, interrupted every 10 years or so by one of nature's experiments in which widespread and almost universal inoculation is carried out with a virulent organism. During the 'experimental phase' the most closely settled areas came off best, because they have been the most effectively immunized. Distribution of the organisms is, apparently, mainly excremental, and I have suggested that dust spread may be important. It seems as if too great an anxiety to protect a child during the latent periods between epidemics from the ordinary contacts of everyday life may do more harm than good.

If my observations on the incidence in schools are published, some misguided person will no doubt suggest that it would be a good thing to close up half the sanitary blocks in schools and remove all the hand-basins. That would be putting the clock back with a vengeance; although it might, indeed, have some effect on the poliomyelitis incidence: poliomyelitis is unknown amidst the squalor of primitive Asiatic communities, although there is evidence that the organism itself is not lacking. Poliomyelitis seems to be one of the penalties we must pay for bettering our sanitary condition generally; it is a phase we must pass through. In the course of time humanity will adapt itself. It is certainly a lesser evil than the diseases we have abolished by improving our sanitary environment.

There is probably a considerable difference, in any case, between the effects of crowding and poor sanitation during an epidemic and in the intervals. Attention to personal hygiene may not give absolute protection during an outbreak, when spread appears to pass all barriers. But it is the only measure by which the individual can hope to reduce the dose of virus which he himself is likely to get. It may for aught we know make all the difference between a minor attack and life-long paralysis. It is probable that the usual and now familiar precautions taken during an epidemic have no effect whatsoever in reducing the total number of persons ultimately affected; but they may reduce the average dose, and by slowing up circulation of the virus keep its virulence at a lower level. We know that in many organisms rapid passage exalts virulence.

If, as I anticipate, this epidemic dies down when the incidence has not far surpassed the 1 per 1000 level, the future can be predicted with some confidence. It must be remembered that in the first big epidemic of 1916 the incidence in the Auckland area was half as great again, being over 15 per 10,000. At that time, however, the population must have been largely unsalted to begin with. I think it is safe to assume that the new cycle will be similar to the last, in which case we may expect a certain number of sporadic cases every year (perhaps an average of six yearly in the Auckland district) and another epidemic about half as severe as the present one around about 1957.

8-2

There is every reason to believe, however, that this prediction need never come true.

The solution to poliomyelitis will not be found through field studies such as the present, but in the laboratory. Nature has shown herself capable of producing an efficient active immunity. One day, perhaps very soon, the bacteriologists will go one better. Then at last poliomyelitis will be conquered, just as to-day we are conquering diphtheria.

SUMMARY

(a) This paper is a study of a poliomyelitis epidemic in the Central Auckland Health District which commenced in November 1947, and by the end of April 1949 had produced 345 cases in a population of about 350,000.

(b) Incidence per 10,000 reached 8·0 in the city, 15·4 in the country districts, and 21·5 in certain urban areas lying between city and country.

(c) In general, incidence was inversely related to population density. Abnormally high incidences in some urban areas was attributed to influx of rural dwellers in recent years. Unusually low incidence in one city area may have been related to the periodic effect of race meetings in increasing the local density.

(d) The three previous Auckland epidemics showed a successively declining incidence, and were linked by periods of grumbling activity. The present epidemic was preceded by a lull, and its toll has approached that of the 1916 epidemic. It is probable that a new cycle has begun which will imitate the first, but on a lower scale.

(e) Spot maps showed that the disease was already widespread before the first positive cases revealed its presence. Some ebb and flow, but no actual movement from one area to another, occurred during its course. An interesting feature was the comparative rarity of cases in the neighbourhood of the city's sewage-polluted bathing beaches.

(f) An inverse correlation was noted between the ratios of 'suspect' to positive cases established in a previous inquiry, and the incidence of cases with paralysis or paresis in various age/sex groups. High ratios indicated lower severity.

(g) It was estimated that by the end of April 1949, all boys aged 10–15 years in Auckland had been affected by poliomyelitis, either 'suspect' or positive. Less than half the girls of this age had been affected. Estimates are given for the other age groups.

(h) Families of cases showed no significant difference in composition from the average in the area.

(i) No correlation, whether inverse or direct, was found between sanitary conditions in schools and the incidence of positive cases.

(j) The cycle of poliomyelitis in the community is discussed in the light of these findings.

APPENDIX

Table 6. *Course of the epidemic* (see Fig. 1)

	1947			1948												1949				Total
	Before Nov.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	
Auckland urban area	2	16	42	14	12	8	4	11	12	9	14	7	7	4	6	18	14	9	9	218
Otahuhu, Papatoetoe, Papakura	—	3	2	2	1	1	1	3	0	4	3	1	1	1	3	1	0	3	0	30
Remainder of district	—	7	15	6	5	8	3	4	2	5	6	1	5	1	3	11	4	6	5	97
Total	2	26	59	22	18	17	8	18	14	18	23	9	13	6	12	30	18	18	14	345

Table 7. *Incidence in different parts of district* (see Fig. 2)

	Population 1 April 1947	Cases to 31 December 1948								Cases per 10,000 total population							
		Males				Females				Males				Females			
		0–	5–	10–	15–	0–	5–	10–	15–	0–	5–	10–	15–	0–	5–	10–	15–
Auckland urban area	274,331	29	35	19	22	13	16	16	18	1·1	1·3	0·7	0·8	0·5	0·6	0·6	0·7
Otahuhu and Papatoetoe	11,570	1	4	0	3	0	3	1	2	0·9	3·5	0	2·6	0	2·6	0·9	1·8
Papakura	2,400	2	5	1	0	0	3	0	1	8·3	20·8	4·2	0	0	12·4	0	4·2
Remainder of district	62,959	4	14	11	12	7	9	9	5	0·6	2·2	1·8	1·9	1·1	1·4	1·4	0·8

Table 8. *Density and incidence in two epidemics (see Figs. 3 and 5)*

| | | | Epidemic of 1947–9 | | | | | Epidemic of 1937 | | | | |
| | | | Positive cases | | With paresis or paralysis | | | Positive cases | | With paresis or paralysis | | |
Authority	Population April 1947	Density per acre 1947	Cases to 31 March 1949	Incidence per 10,000	Cases to 31 December 1948	Incidence per 10,000	Density per acre 1936	Cases	Incidence per 10,000	Cases	Incidence	Population 1936
Newmarket	3,060	16·8	1	3·3	1	3·3	—	—	—	—	—	2,997
Mount Eden	21,100	14·3	8	3·8	2	0·9	12·5	8	4·3	7	3·8	18,515
Mount Albert	26,050	10·7	13	5·0	7	2·7	8·1	8	4·1	6	3·1	19,721
Onehunga	15,200	8·1	10	6·6	2	1·3	—	—	—	—	—	11,082
Auckland City	131,800	7·2	131	9·9	49	3·0	5·6	18	1·8	16	1·6	102,295
Otahuhu	7,570	5·6	10	13·2	3	4·0	3·9	3	5·1	2	3·4	5,252
One Tree Hill	13,050	5·4	15	11·5	3	2·3	—	—	—	—	—	8,027
Ellerslie	3,270	4·4	1	3·1	1	3·1	—	—	—	—	—	2,690
New Lynn	4,720	3·4	1	2·1	1	2·1	—	—	—	—	—	3,492
Mount Roskill	14,700	3·1	13	8·8	5	3·4	1·5	6	1·4	4	0·9	6,979
Mount Wellington	2,490	0·6	3	12·1	1	4·0	—	—	—	—	—	1,685

Table 9. *Progress of the epidemic, Auckland urban area* (see Figs. 11 and 12)

(a) Monthly cumulative totals of positive cases, by age groups

Sex	Age group	Population 1945 (nearest 100)	Ratio suspect to positive cases	1948 Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	1949 Jan.	Feb.	Mar.	Apr.
Males	0–	11,600	100 : 1	17	19	20	23	24	26	28	28	29	29	29	29	31	33	33	34
	5–	8,800	100 : 1	19	19	20	20	22	24	25	30	30	33	35	35	37	41	42	44
	10–	8,000	300 : 1	4	4	5	5	6	11	13	15	16	17	17	19	20	24	26	27
	15–	92,300	500 : 1	10	13	14	14	15	16	18	19	19	19	20	22	29	30	30	30
Females	0–	11,200	200 : 1	7	8	8	8	10	11	11	11	12	12	12	13	17	19	20	21
	5–	8,500	200 : 1	5	6	7	8	8	9	11	12	12	14	15	16	18	19	22	26
	10–	7,800	200 : 1	6	7	9	10	13	13	13	13	15	16	16	16	16	16	17	17
	15–	110,200	1000 : 1	6	10	11	11	12	16	12	16	18	18	18	18	18	18	19	19

N.B. Population figures are for 1945, and therefore lower than the actual populations at risk.

(b) Calculated percentage of age group affected by poliomyelitis, 'suspect' or positive

Sex	Age group	1948 Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	1949 Jan.	Feb.	Mar.	Apr.
Males	0–	15	16	17	20	21	22	24	24	25	25	25	25	27	29	29	29
	5–	22	22	23	23	25	27	28	34	34	37	40	40	42	47	48	50
	10–	15	15	19	19	23	41	49	56	60	64	64	71	75	90	98	100
	15–	5	7	8	8	8	9	10	10	10	10	11	12	16	16	16	16
Females	0–	12	14	14	14	18	20	20	20	21	21	21	23	30	34	36	38
	5–	12	14	16	19	19	21	28	28	28	33	35	38	42	45	52	61
	10–	15	18	23	26	33	33	33	33	38	41	41	41	41	41	44	44
	15–	5	9	10	10	11	11	11	15	16	16	16	16	16	16	17	17

Table 10. *Incidence of cases with paralysis or paresis* (see Fig. 10)

Sex	Age	Population 1945	Ratio suspect to positive cases	Paresed cases to 31 March 1948	Incidence per 10,000	Paresed cases to 31 December 1948	Incidence per 10,000
Males	0–	11,619	112 : 1	10	8·6	16	13·8
	5–	8,772	99 : 1	11	12·5	12	13·6
	10–	7,965	319 : 1	2	2·5	6	7·5
	15–	92,330	546 : 1	13	1·4	16	1·7
Females	0–	11,184	203 : 1	3	2·7	5	4·5
	5–	8,500	230 : 1	4	4·7	6	7·1
	10–	7,822	168 : 1	6	7·7	7	9·0
	15–	110,273	972 : 1	8	0·7	11	1·0