A CONTRIBUTION TO THE EPIDEMIOLOGY OF POLIOMYELITIS IN NEW ZEALAND*

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(With 10 Figures in the Text)

FOREWORD

The investigation described in this paper was undertaken in Auckland during the summer of 1947–8. The field work was carried out by the writer in conjunction with Dr G. A. de Lautour, Medical Officer of Health, and Miss D. F. Gatenby, Nurse Inspector; assistance was also given by Drs Houghton, Miller and Parr, of the staff of the Auckland Health District. My best thanks are due to all for their ready co-operation and hard work, and for their carefully kept records, which greatly facilitated the task of analysis.

In consideration for the reader, most of the tables have been relegated to the Appendix, and full use has been made of graphic methods of presentation of data.

A. W. S. Thompson

Auckland, 27 *May* 1948

Note. Unless otherwise stated, all cases of illness are referred to in this paper by date of *onset*, not of notification.

CONTENTS

I. POLIOMYELITIS EPIDEMIC, 1947–8

On a Saturday morning in October 1947, an Auckland school-girl, running from her home to a waiting car to commence a trip to the country, was noticed to stagger to one side as if giddy. She denied feeling ill, but when her temperature was taken it was 104° F.

This girl was the first victim of the epidemic of poliomyelitis with which this paper is concerned. The correct diagnosis in her case was not established until several weeks had passed. The earliest cases to be notified were reported towards the end of the third week in November. By the end of the year 87 cases, and by the end of March 142 cases, had occurred in the Central Auckland district.

The earliest cases were scattered widely. The first as mentioned above, lived in Auckland City (onset 25 October). The next was at Papakura, 19 miles to the south (10 November). As early as 19 November, when the outbreak in the city was just getting under way, a farmer in a remote valley near Hunua, some 30 miles to the south-east, experienced the commencing symptoms of an attack.

In Auckland itself there was nothing to indicate spread from any particular focus. This is well shown on the spot maps (see Fig. 1), on which the first 5, 10, 20 and 50 cases have been plotted. The first 10 cases affected seven different residential areas, and the first seven children attended four different schools. Even when two patients were drawn from the same school, they were usually found to have been in different standards and had had no traceable contact with each other.

Efforts to clarify the situation by detailed studies of the movements of cases and those with whom they had been in contact were not very helpful. Quite

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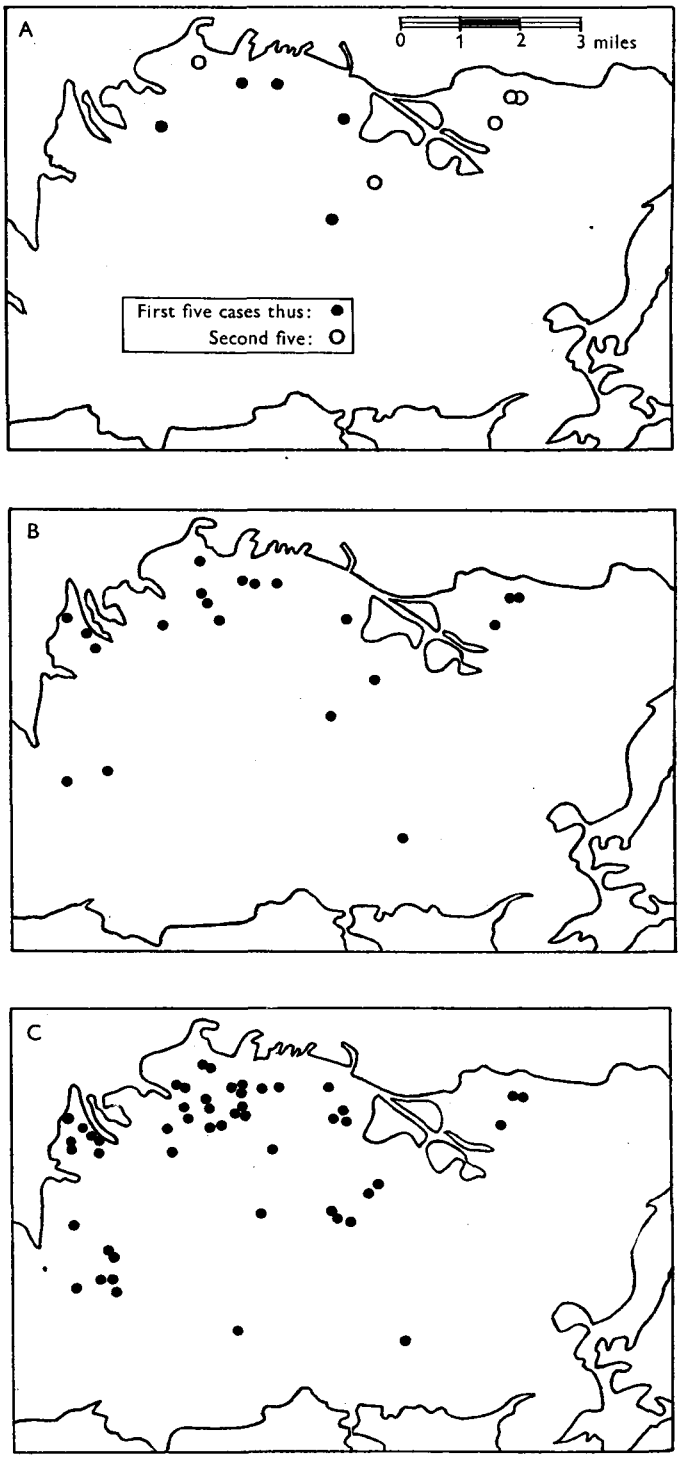

Fig. 1. Spot maps to show development of epidemic in Auckland: A, 10 cases, 20 November; B, 20 cases, 1 December; C, 50 cases, 17 December. N.B.—Dates refer to onset.

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often one was rewarded by tracing a relationship, sometimes between widely separated cases; but it was disconcerting to return to the office after discovering an ingenious link between 'cases', only to find that in the meantime one of them had been declared negative; and this happened more than once. It was soon obvious that in the neighbourhood of minor sickness about of a nature possibly related to poliomyelitis, but it varied enormously in severity, and occasionally an important link in the chain appeared to consist of somebody who had not been ill at all. It was easy to construct schemata, but difficult to defend them in detail.

An example may be of interest.

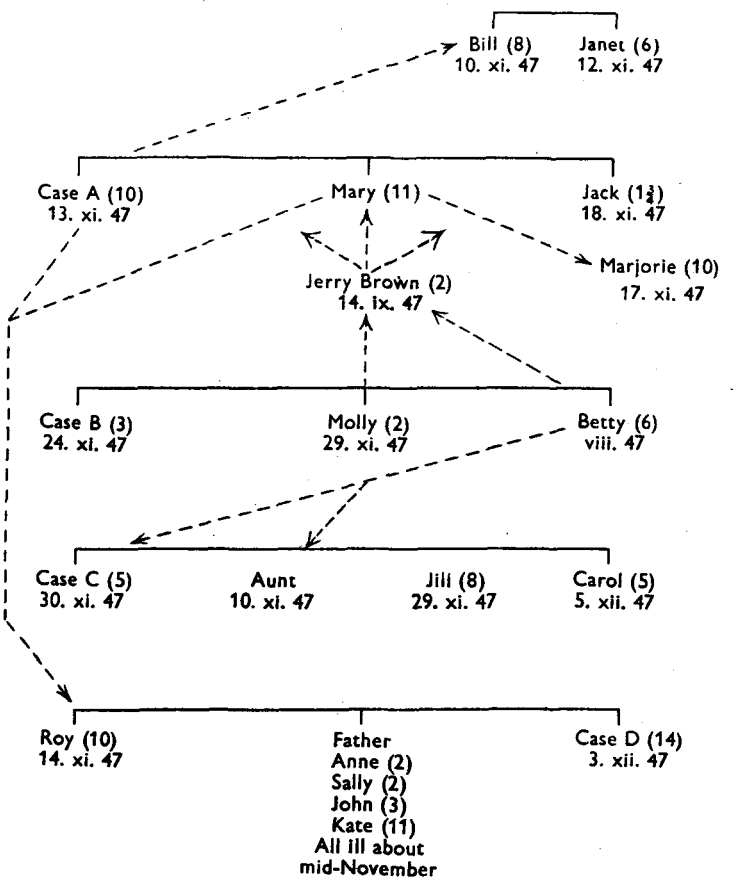

Fig. 2. Possible linkage between four positive Cases and sixteen other suspicious illnesses. (Ages in brackets. Dates are of time of onset. Unaffected members of households not shown.)

every positive case one could discover numerous instances of minor illness of a suspicious nature, and several quite definite abortive cases came to light at an early stage. This was merely what previous knowledge of the disease had led one to expect. It was not difficult in some instances to link one case with another through a network of suspicious illnesses in contacts, and it seemed not impossible that diligent inquiry might have extended the network to include most of the cases in the city. Even if this could be done, however, it would still be open to question whether the whole structure could not be accounted for by chance. There was a good deal of

II. NETWORK LINKING FOUR POSITIVE CASES

One of the earliest notified cases could be connected rather neatly with another in the same area, and two more living 5 miles away on the other side of the city. Sixteen suspicious illnesses, including at least one definite abortive case, were also involved.

Fig. 2 shows the relationship between these cases, the horizontal lines connecting members of the same family, and the broken lines indicating possible transference by contact. Fictitious names have been employed. Except for Mary, sister of case A, only

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persons who had a suspicious illness are shown, the dates are the times of onset.

Case A was a boy whose parents first realized he was ill on 13 November, but for a week before that his elder sister, Mary, had given him a pick-a-back home from school because of pain and weakness in his right leg. This girl had no history of any recent illness. The boy's classmate, Bill, was ill during the week before he took to bed, and a playmate of Mary's, Marjorie, had a suspicious illness shortly afterwards.

Next door to case A lived a child aged 2 called Jerry Brown. There was said to be little contact between the two families, but both houses were notably fly-infested owing to the activities of a senile neighbour who was fond of throwing night-soil on her garden. Jerry stayed with the family of case B from 25 August to 10 September, and on returning home had an illness lasting for a week with vomiting, diarrhoea and lassitude.

Betty, 6-year-old sister of case B, had 'influenza' about August, but remained well thereafter. She was a classmate and friend of case C and often visited his home. The aunt of this case, who lived with him, was in bed for about a week in the middle of November with vomiting, diarrhoea and a high temperature.

There was a double link between the families of cases A and D. Roy, who was in the same class as Mary, sister of case A, generally walked home from school with her, and was ill at about the same time as case A, with a sore throat, high temperature and marked drowsiness. Within the succeeding 10 days, five other members of the family went down with similar symptoms; only one girl, aged 14, escaped at this time, but early in December, after a very heavy day's gardening, she was admitted to the hospital as a positive case with severe paralysis (case D). The other link was the fact that, for 6 days from 12 November, just before case A took ill, the father of case A was working on repairs in the house of case D.

Study of Fig. 2 will show how difficult it is to be certain of the reality of the supposed relationship between these cases. The earliest illness noted was in Betty, sister of case B. If this had made her a carrier, she might possibly have infected Jerry Brown and case C as well as her sister (case B), and Jerry Brown might have conveyed the infection to the family of case A, who in turn might have passed it on to the family of case D; but it is difficult to account for the discrepancies in time. Jerry Brown had left the case B home before he took ill on 14 September; yet it was over 2 months before case B fell ill, and a similar interval separated Jerry's illness and the onset of case A. The more closely one studies it the more flimsy the whole structure appears.

Similar difficulties arose when other tentative networks linking positive cases were examined with a critical eye. There seemed to be no order or reason about the way in which these illnesses sprang up amongst a group of susceptibles. Two cases occurred, for example, in families living next door to each other. The victims were children with a marked difference in age who seldom came in contact, but were linked through the agency of playmates of their own ages, a sister of one and a brother of the other. Yet these two cases fell ill on the same day, and the illnesses of the 'links' occurred afterwards—one 2 days later, the other 8. In other instances a family of children of susceptible age in close daily contact would develop their illnesses not simultaneously, but successively, often with a considerable interval between them. It was all so haphazard that one was almost tempted to reject altogether the idea that these minor illnesses had any important connexion with the epidemic. At this stage in our inquiries we were the more inclined to scepticism about it, because of an almost involuntary tendency to regard droplet infection as the prevalent mode of spread. We were, of course, aware of the stress which has been laid in recent years on other means of transfer, especially intestinal, but we found it difficult to believe that an outbreak so widespread and increasing so rapidly could be other than a droplet infection. But surely no droplet infection ever behaved like this!

It was obvious that an investigation of the kind just described could only be of very limited value. It tends to overlook those who, despite close contact, escape unscathed; yet such people are far more interesting than those who fall ill. Why, how, and for how long do they continue to escape? A study of illness amongst contacts is handicapped, also, when one does not know what particular types of contact are of most importance. One naturally thinks of a school-child's classmate, the boy or girl who shares a desk with him, as a close contact; as indeed he is, if it is droplet infection that matters. But other less obvious contacts, much less easily traced, may be more interesting. For example, the child 'Mary' (Fig. 2) and her friend 'Marjorie' were in different classes, did not know each other's surnames, and never met outside school; neither could have named the other as a contact; but they met each day at lunch-time, and their routine was interesting. First they would play together for a while with a hand-ball, tossing it from one to the other. Then they sat side by side on a bank and ate their sandwiches. Before meeting they had, of course, visited the lavatory, and neither ever bothered to wash her hands; and in this school, as in many others, the children had an amiable habit of exchanging their more interesting sandwiches with their friends. There may not have been much interchange of droplets between these two, but the mechanism for the transfer of faecal organisms was complete.

III. PLAN FOR AN INVESTIGATION

What we really wanted to know was what was going on in the population at large while these positive cases were bobbing up so haphazardly; and particularly what had happened in the month or two before recognizable cases began to appear, and especially in the affected households. How did it all begin? What happened afterwards? What about the family contacts who had no history of illness? Did they eventually fall sick with a minor attack; and, if so, why did not all the susceptibles in close contact get it at about the same time?

It was with these and similar questions in mind that the investigation about to be described was undertaken.

It was noted at an early stage that few suspect cases and no positives had been reported from the North Shore. This seemed strange, because, although this portion of Auckland is separated from the remainder by the Waitemata Harbour, a considerable proportion of its population of 30,000 odd are employed on the city side; continual daily contact is maintained through the ferry service, and the North Shore beaches attract innumerable car-loads of children from south of the harbour every week-end. It was decided to regard the North Shore, so long as its freedom from cases continued, as a control area for the study of conditions on the city side. Even if the epidemic eventually spread there, it was hoped that its late commencement would enable us to find out what sort of conditions immediately precede an outbreak.

As it turned out, the date of onset of the first case on the North Shore was 24 December, nearly 2 months after the earliest recognized case fell ill. By this time over 50 cases had already occurred on the city side. By the end of March the North Shore had furnished only four cases, as against 91 in the rest of the metropolitan area (ratio 1:23), the population ratios being about 1:8. The method adopted was simple. The investigation was divided into two parts:

(1) The homes of cases occurring in the metropolitan area were visited as soon as possible after notification, and careful inquire was made into (*a*) the recent health of the patient, and the symptoms of onset of the attack; (*b*) the health of all other members of the household. Leading questions were avoided, an attempt made to interest the family in the problem and, by unhurried and sympathetic questioning, to obtain a complete history. Full allowance was made for the difficulty of recalling the details of family illnesses; few people find it easy to remember much about the health of their families more than a week or so previously, and the present writer found that he himself was quite unable to describe the health of his own household during the preceding month. The symptoms and duration of all illnesses possibly related to poliomyelitis were recorded and the date of onset was fixed as closely as possible in every case. Special note was made of any of the following:

Fever	Sore throat
Vomiting	Pain or stiffness of the neck
Diarrhoea	Abdominal pain
Headache	Drowsiness Delirium

(2) The first 16 confirmed cases in the metropolitan area were selected as nuclei for the study of the background to the epidemic. House-to-house visits were carried out in the area surrounding each of these cases, the houses visited being grouped, as far as possible, symmetrically about the house in which the case had occurred. A note was made of any address where admittance had not been obtained, and about twenty houses were entered in each area. As soon as possible after completing an area, the same investigator crossed to the North Shore, selected a district which appeared to be of similar social and economic type, and visited twenty homes. These served as controls.

The distribution of the test areas is shown in the sketch map (Fig. 3).

It was agreed between members of the team* that in all cases they would try to gain admittance to the house, and sit down quietly in the living-room with the housewife, before commencing detailed inquiries; information gained on the doorstep could not be of equal value. They were instructed to use some such formula as: 'I am Dr—— of the Health Department. We are making some inquiries in connexion with the infantile paralysis outbreak. We are trying to find out how much illness there is in Auckland at present. I mean illness of any kind, including quite minor illnesses. Do you mind giving me some particulars?'

Instructions to the investigator continued as follows:

'Information must be extracted by direct and persistent questioning. If necessary, give reassurance that it is not intended to apply any restrictions as a result of this investigation. Do not accept a reply that a person has not been ill lately without further probing.

'*Nature of illness.* Ask about each member of the household in turn, inquiring about "flu", feverish attacks, stomach upsets, headaches, sore throat, pains in limbs, etc. Has he been in bed ill, even for one day, in the last month or so? Note details of any illness even remotely related (but not other illnesses). Note if a doctor was called, and his diagnosis. Make note of date of onset in all cases.'

* The bulk of this part of the investigation was carried out by Dr G. A. de Lautour and Miss D. F. Gatenby.

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IV. RELIABILITY OF FINDINGS

Study of the household contacts of positive cases was based on 40 cases in the metropolitan area. The dates of onset ranged from 25 October to 29 December. Consecutive cases were taken, except that those which were rapidly fatal were not included. All homes were visited on three occasions, the first visit following immediately upon notification, the second and third being made after intervals of from 6 to 8 weeks.

There were fifteen test areas to the south of the Waitemata and fifteen control areas on the North Shore; in one instance an area was based on two cases in the same street which occurred on the same day. In 15 of these cases the dates of onset fell between 12 November and 1 December, while one commenced on 18 December. Visits to the test areas were completed between 1 and 23 December, the control areas being completed between the 3rd and 23rd. Intervals between visits to test and control areas averaged less than 3 days and were never longer than 5. A second visit was paid about the end of January to all homes in which there were children, and a third at the end of March. These families were therefore under fairly continuous observation throughout the period of the epidemic.

With regard to the well-known fallacy in such investigations, that houses where there happened to be nobody at home were not included, it is not thought that this factor was of much importance in the present instance. Throughout the period in question all schools were closed and the public had been warned to keep children segregated at home and not to allow them to go about the streets or enter shops. These instructions were, on the whole, very carefully observed, and most parents seldom allowed their children to leave their homes or gardens. Houses which included persons under the age of 16 were therefore ha dly ever found to be empty.

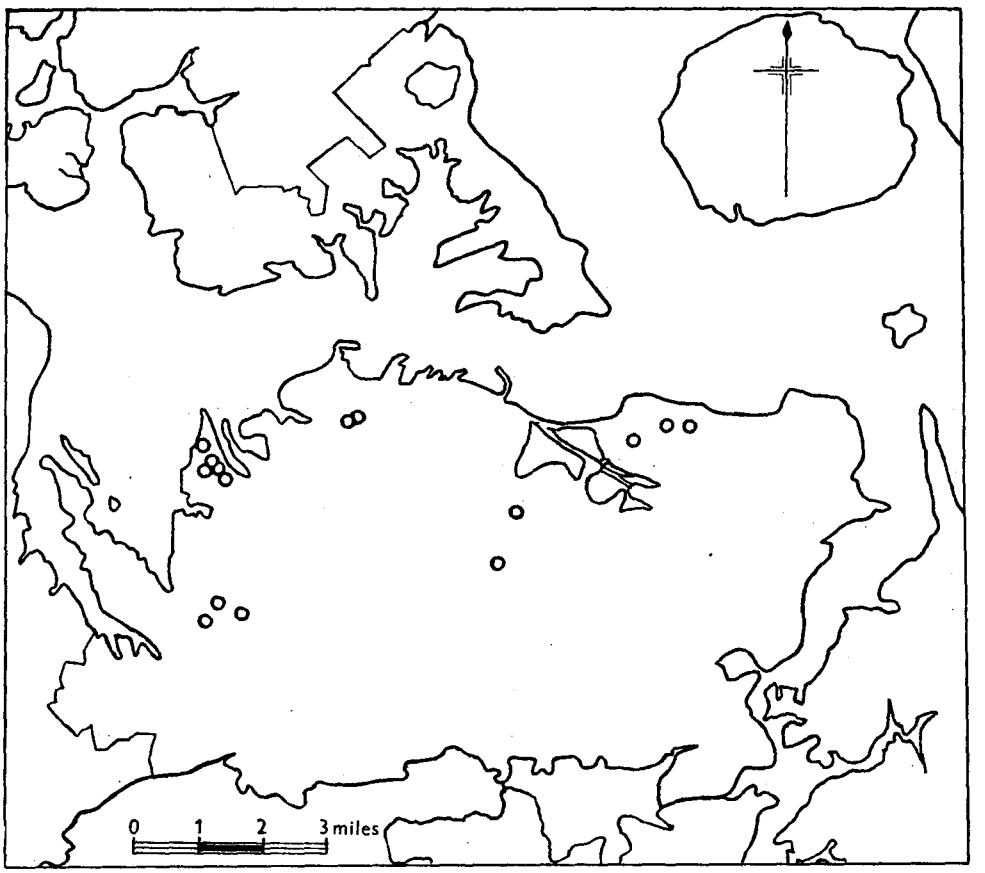

Fig. 3. Distribution of test areas: control areas (not marked) were all north of the Waitemata harbour 'North Shore'. ○ Test areas.

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Most of the inhabitants of missed houses must have been adults, and therefore, so far as households consisting of adults only are concerned, our results cannot be accepted without reserve.

In all cases the occupation of the head of the household was noted, and the house was placed in one of four social and economic classes (see Appendix, Table 1). Comparable control areas were not found without difficulty, and in two instances surveys of test areas had to be excluded because similar districts could not be found on the North Shore. In the final result, however, the two groups selected were practically identical in social and economic status.

In the Appendix, Tables 1 and 2, details will be found of the types of houses visited and of the composition of the families living in them, in both test and control areas. It will be seen that about 300 families (1100 persons) were included on each side of the Waitemata, families being subdived into those without children, those in which there were only pre-school children, and those including schoolchildren. There were minor differences in the density and composition of these families, but not sufficient to invalidate comparison.

The investigators were well received, but it was noticeable that where there were only adults in the household information was not so readily obtained as elsewhere. Mothers of young families were, without exception, most willing to co-operate. There was no reason to suppose that persons living on the North Shore were any less 'polio-conscious' than those on the city side.

A possible source of inaccuracy was the fact that in most cases information was obtained from the housewife, who might be expected to have a better recollection of her own illnesses than of those of the remainder of the household. It is possible, therefore, that the incidence of illnesses amongst adult females might be somewhat exaggerated under this method of inquiry.

V. THE BACKGROUND OF THE EPIDEMIC: THE EARLY STAGES

As has already been explained, during their first visits to test and control areas the investigators collected information about the health of persons in all types of families, including those consisting only of adults. The epidemic itself did not get properly under way until the third week in November, and it was decided, therefore, to separate illnesses in which the onset occurred before 1 November from those commencing later.

To simplify an assessment of the results of this part of the inquiry, the populations concerned were reduced to equal numbers. This was done by ignoring the excess persons in each age group, care being taken to ensure that the resulting populations would have the same composition in respect of each type of family.* The following comparisons are therefore concerned with equal test and control populations, composed as follows:

	Total	Type of family: Adults only	Only pre-school-children	With school-children
Adults	717	295	79	343
Schoolchildren	246	—	—	246
Pre-schoolchildren	128	—	47	81
Persons	1091	295	126	670

Considering, then, these equal populations of similar composition, we found that 164 persons in the test areas had a recent history of suspicious illness and 114 in the control areas—i.e. 15 % of the test population and 10·4 % of the controls.

These illnesses varied enormously in severity, and their analysis presented a difficult problem. To classify them as either 'possibly' or 'probably' related to poliomyelitis (the first method which was tried) would be too open to personal bias. It was finally decided that the only way to achieve an objective analysis was to classify them according to the number of suggestive symptoms actually recorded by the investigator. For this purpose:

(*a*) The following were designated 'cardinal' symptoms: fever, vomiting, diarrhoea, headache, sore throat.

(*b*) The following were regarded as suggestive: pain or stiffness in the neck, abdominal pain, drowsiness, delirium.

All illnesses recorded were then classified according to the number of 'cardinal' symptoms mentioned, but if two or more of the features listed under (*b*) were also present, the illness was placed in the next category above. All illnesses were further subdivided into those of less than 4 days' duration and those lasting for 4 days or more.

The results are shown in Fig. 4. Figures will be found in the Appendix, Table 3.

In Fig. 4, persons ill in the test areas are indicated by the black bars, those in the control areas being uncoloured. The situation before 1 November is shown in the upper half of the diagram, that after 1 November in the lower half. The following inferences may be drawn:

(*a*) *Before 1 November* suggestive illnesses of long

* It was necessary, for example, to reduce the number of adults in the test areas from 732 to 717 in order to make them equal to the total in the control areas. This was done by ignoring the last two adults in families without children, the last 10 in families including only pre-schoolchildren, and the last three families with school-children. This made the number of adults *in each type of family* the same in both populations.

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duration were practically identical in the two areas studied, both in frequency and severity.

(*b*) Suggestive illnesses of short duration were nearly twice as common in the test areas as in the control areas (1·8:1).

(*c*) *After* 1 *November* there was a considerable increase in illnesses of both types in both test and control areas. (Increase was 2·1:1 in test areas and 2·8:1 in the controls.)

the number of illnesses in *adults*, especially adults in families without children. After 1 November, families including children were much more severely affected in test than in control areas, particularly where there were schoolchildren, and there the increase was especially marked in respect of illnesses of long duration (3·2:1). After 1 November, illnesses in wholly adult families increased in the control areas, while tending to subside in the test areas.

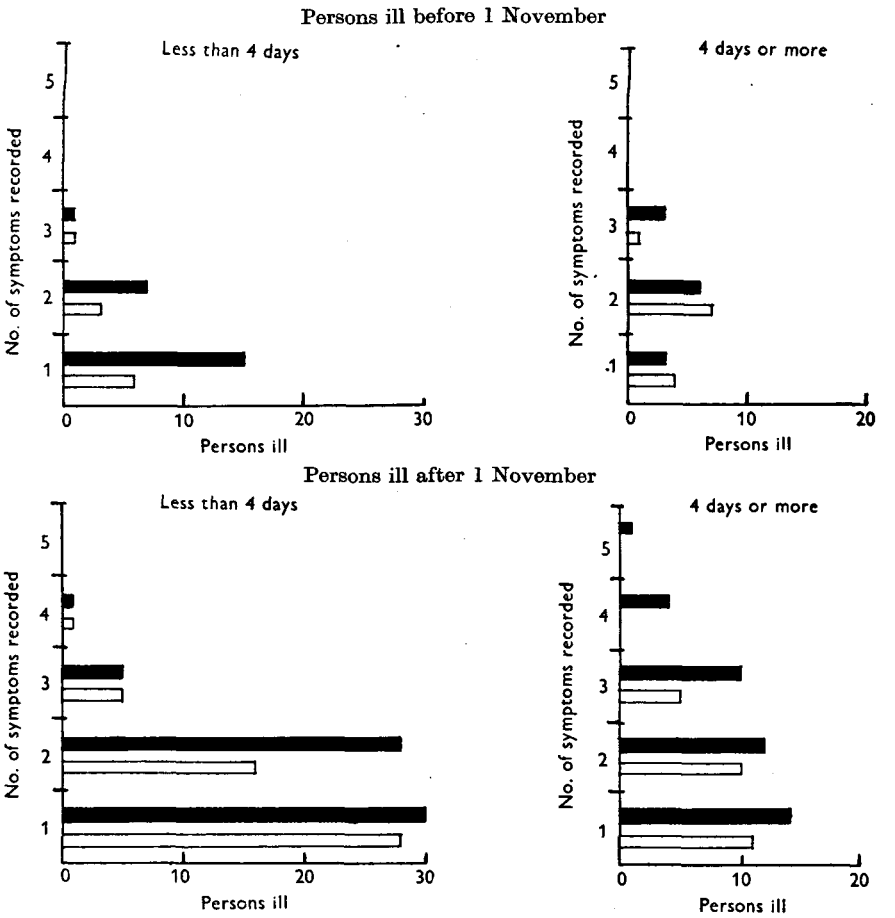

Fig. 4. Illnesses amongst equal populations (1,091 each) visited in December, 1947.
Test areas. Controls.

(*d*) The most notable feature after 1 November was that suggestive illnesses of long duration were now much commoner in test than in control areas (1·7:1), and the preponderance was greatest in illnesses showing three or more 'cardinal' symptoms (3:1).

A further analysis was made to discover what age groups and what types of family were most affected. Figures are given in the Appendix, Table 4. It was found that in the period before 1 November the excess of illnesses of short duration noted above was due to

This part of the investigation suggested, therefore, that the epidemic had been preceded by an increase of illnesses of short duration amongst adults, particularly adults in families without children. As the epidemic developed, illnesses in childless households tended to become less frequent in districts where polio cases were actually occurring; but in areas more remote, childless households now began to show an increase. Concurrently with the early stages of the epidemic, families including children suffered a considerable amount of the kind of illness

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which has in the past been regarded as possibly related to poliomyelitis. This was much commoner in the neighbourhood of polio cases, and most obvious and definite where there were schoolchildren. Even parts of the city where no positive cases had yet been identified seemed, however, to be going through a similar process at a less advanced stage.

VI. ILLNESSES IN AFFECTED HOUSEHOLDS

Forty houses in which positive cases had occurred were visited (*a*) in November and December, immediately after notification of the case, (*b*) in January, and (*c*) at the end of March. At the outset more than 150 homes were under observation, but many of these cases proved to be negative, and eventually it was decided to concentrate on cases in the metropolitan area which had commenced before the end of December 1947. Conditions of spread in rural areas must obviously be rather different from those in the city.

These 40 households included a total of 218 persons, distributed as follows:

	Primary cases		'Secondary' cases		Household contacts	
Age	M	F	M	F	M	F
0 and under 5	8	4	—	2	11	14
5 and under 10	15	3	—	—	12	13
10 and under 15	3	5	—	—	9	10
15 and over ('adults')	2	—	1	1	49	56
Totals	28	12	1	3	81	93

A second positive case occurred in four different houses, one of which commenced on the same day as the original case, another 2 days afterwards, the others after intervals of 10 days. Of the remaining 174 contacts, 77 (43 %) had an illness during the period under review which might possibly have been regarded as poliomyelitis in a mild form. Six were seen during the attack and were definitely diagnosable as abortive cases. The remainder varied enormously in severity, but only cases which displayed some genuinely suspicious feature have been included in this survey.

The onset of the positive case in the household was taken as the focal point for this part of the investigation, suspicious illnesses in the contacts being studied in their time relation to the positive case. Suspect illnesses whose dates of onset fell within 6 days on either side of that of the positive case were regarded as concurrent with it, as neither was likely to have been responsible for the infection of the other, assuming the generally accepted incubation period of not less than 7 days.

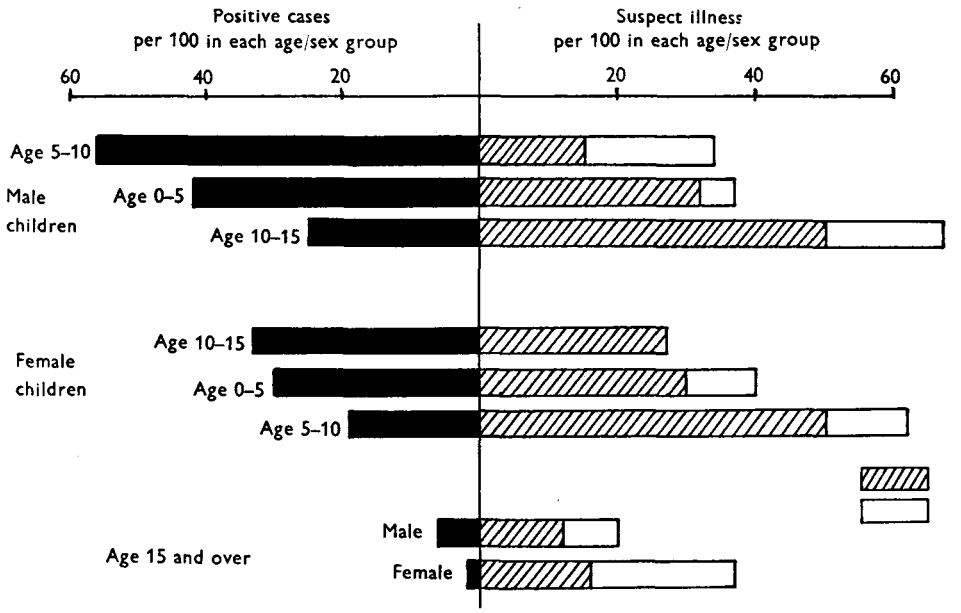

Fig. 5. Forty affected households: relationship between positive cases and suspect illnesses in age/sex groups. ▨ Before and concurrent. □ After.

As soon as suspect illnesses amongst contacts were divided into those concurrent with or commencing before the positive case and those occurring afterwards, it became obvious that there was probably a mathematical relationship between positive cases and suspect illnesses. This is shown in Fig 5, details being given in the Appendix, Table 5.

It will be noted that in each of the groups, male children, female children, and persons aged 15 years or over, the more positive cases there were, the less

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suspect illness was found*. This is true whether we consider all suspect illnesses or only those occurring before the onset of the positive case or concurrently with it. The coefficient of correlation is very high (from −0·88 to −1 in the latter case, −0·67 to −1 in the former) and confirms this apparent relationship.

This is an important observation. It is seldom possible—in ordinary circumstances, never possible—to prove that a particular minor illness in a polio contact has any real connexion with poliomyelitis. The relationship just described is, however, very strong evidence that the illnesses recorded in these contacts were definitely linked with the positive cases in their friends.

Up to this point in the investigation I must confess that I was often doubtful whether the whole basis of the inquiry was not fallacious. From here onwards one felt at liberty to discuss the behaviour of these contact illnesses with much more confidence than would have been justified by their clinical study above.

The next fact of importance shown in Fig. 5 is that there is no *general* correlation between positive cases and suspect illnesses, regardless of age and sex. If we arrange all the age/sex bars in Fig. 5 so that the left-hand portions (percentage of positive cases) are in order by lengths, the right-hand columns are thrown into confusion. To bring out the relationship described above it is necessary not only to consider 'adults' separately from the rest, but to divide children under 15 into the two sex groups.†

The meaning of this appears to be that there is some fundamental difference between the effect on male children of a poliomyelitis risk as compared with female children. It is well known that in all large outbreaks there are more boy positives than girls. This has sometimes been attributed merely to a preponderance of males over females in the younger age groups (see, for example, Sydney Smith's report on the Wellington epidemic of 1916, p. 21), but study of Fig. 5 will show that the pattern of positives and suspects is quite different in the two sexes. We will return to this question later.

Let us now see how this state of affairs developed. In Fig. 6 (see Appendix, Table 6) the progress of suspect illnesses amongst the different age groups is shown. Let us look, first, at the period up to 7 days before the onset of the positive case. This should demonstrate which age group was most liable to introduce the infection into the family.

It will be seen that more than 4 weeks before the onset of the positive case, members of both school age groups in each sex developed suspect illnesses. Other groups were affected as time went on, but the most striking feature is the steady advance of suspect illnesses amongst male children aged 10–15

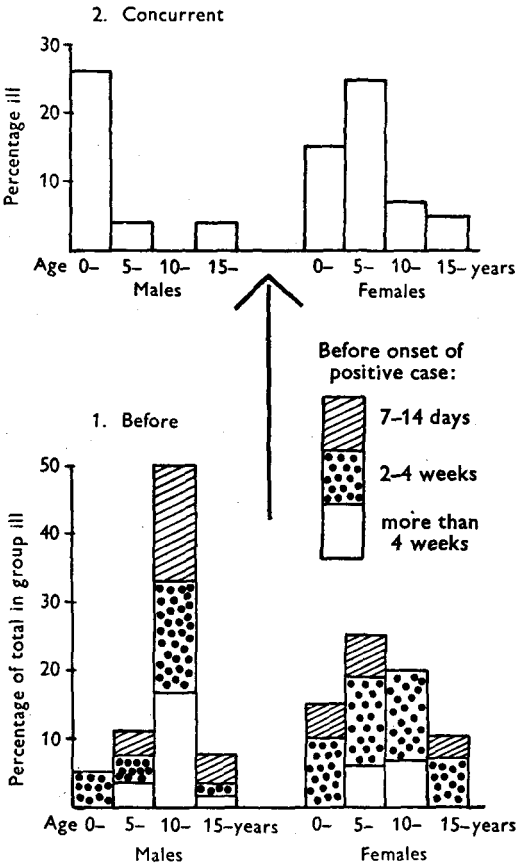

Fig. 6. Forty affected households: development of suspect illnesses 1 before, 2 concurrent with positive cases.

years, 50% of whom had already had an attack by the time the positive case commenced.

Now observe what happened at the time when the positive case fell ill. So far as suspect illnesses in male children are concerned, the situation is reversed. Schoolboys aged 10 or over developed no concurrent illnesses; pre-school males, least affected before, now had several. In females, however, concurrent illnesses followed the same pattern as before, presumably because the process of salting had not previously advanced so far as in males. Add con-

* By 'suspect illnesses' we really mean *persons* who developed a suspect illness, as only one illness was recorded for any individual.

† If, for example, we consider suspect illnesses before and concurrent, the coefficient of correlation is reduced from −0·88 for male children and −0·88 for females to 0·81 for both sexes combined. This is still a very high degree of correlation, but the reduction supports the view that there is some essential difference between male and female children in their response to the polio risk.

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current illnesses to those already recorded, and the distribution of positive cases in children of either sex can be predicted by simply applying the rule

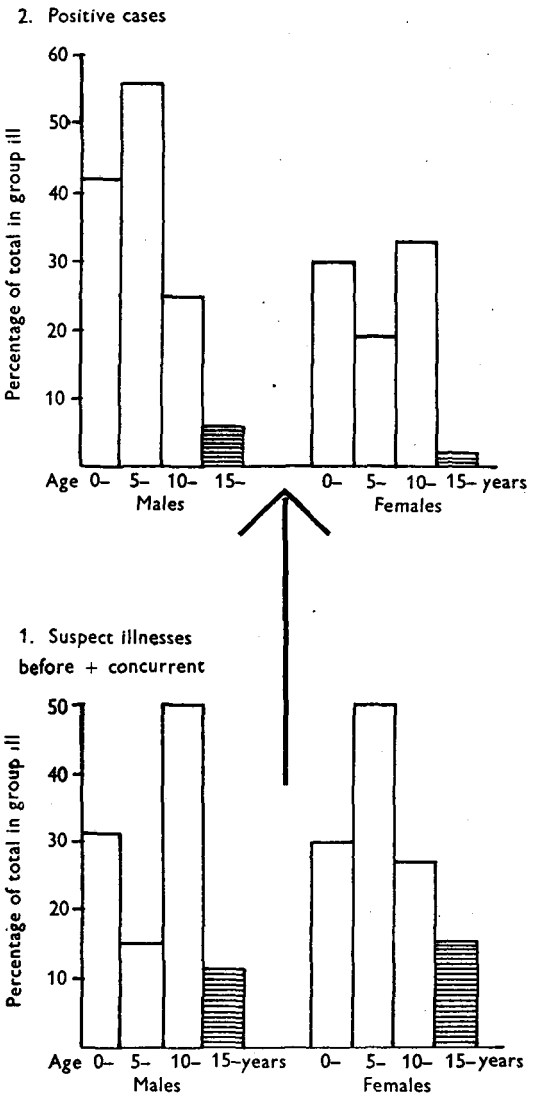

Fig. 7. Forty affected households: inverse relationship between suspect illnesses and positive cases in age/sex groups. ▤ Age 15 and over.

that the less suspect illness there has been, the greater will be the incidence of positive cases. This is shown in Fig. 7.

Indeed, it would almost be possible to forecast the actual number of positive cases in each age group, because of the high proportion of male children aged 10–15 years and of females aged 5–10 who either have a suspect illness or become positive cases. This is shown in Fig. 8, in which all suspect illnesses, including those occurring after the positive case, have been included.

It will be seen that the gap in each group, representing the percentage of persons for whom nothing was recorded in the period covered, increases from male children (where it is small, 8–21 %) through female children to adults.

Since some 'suspect illnesses' were only mild, a proportion may have been overlooked or forgotten. It is clear from Fig. 8, however, that we cannot close all the gaps by adding equitable proportions to represent missed illnesses. In other words, it is evident that, even in the homes of positive cases, all children exposed to infection do not suffer either a clinical or subclinical attack; for some reason most adults, and certain female children, seem to escape completely.

In the case of adults it would be natural to assume that the gap might be due to immunity acquired in a previous epidemic. But what of the difference between male and female children? In only one age group—the pre-schoolchild—does Fig. 8 suggest an identity of response. In the next group, aged 5–10 years, illnesses in girls seemed to be both less prevalent and less severe than in boys. In the 10–15 group, girls had less than half as much suspect illness as had boys of the same age, but rather more positives, and this is the group with the largest gap in female children and the shortest in males. We conclude, therefore, that, on the whole, female schoolchildren are either less susceptible to polio than males or are less frequently or less heavily infected. At a later stage we may find a clue as to which explanation is the more likely.

We have established that the majority, at least, of these suspect illnesses amongst contacts are related to the poliomyelitis virus. The next question is the interval between successive cases in the same household. When several of these illnesses have occurred in a family it is reasonable to suppose that in most cases those which have not become ill simultaneously have been infected by the patient next before in the family chain.*

There were 81 contact illnesses in our 40 households. The intervals between successive illnesses (including positive cases) in the same household ranged from 1 to 110 days and fell into two practically equal groups:

Thirty-seven with a short interval, from 1 to 10 days.

* I am aware that some authorities would dispute this assumption, arguing that individual variations in the incubation period may account for the stringing-out of several cases infected at the same time. The assumption is, however, justified for present purposes, at least until the alternative is proved.

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Thirty-six with a long interval, from 12 to 110 days.

Ignoring for the moment 16 cases in which the interval was 4 weeks or more, let us consider the 57 cases with an interval of 1–23 days. These are plotted in Fig. 9.

It will be seen that they fall into two groups, 37 with a short interval (1–10 days) and 20 with a long interval (12–23 days). The mean interval for the first group was just under 5 days, with a standard deviation of 2·51. The 20 cases falling between 12 and 23 days gave a mean interval of about 18 days, standard deviation 1·34. An obvious hypothesis is that these may represent the effects of different modes of transmission. May not the short interval imply droplet infection, and the long interval infection by faecal organisms? In the latter case one would expect considerable variation in the interval, which

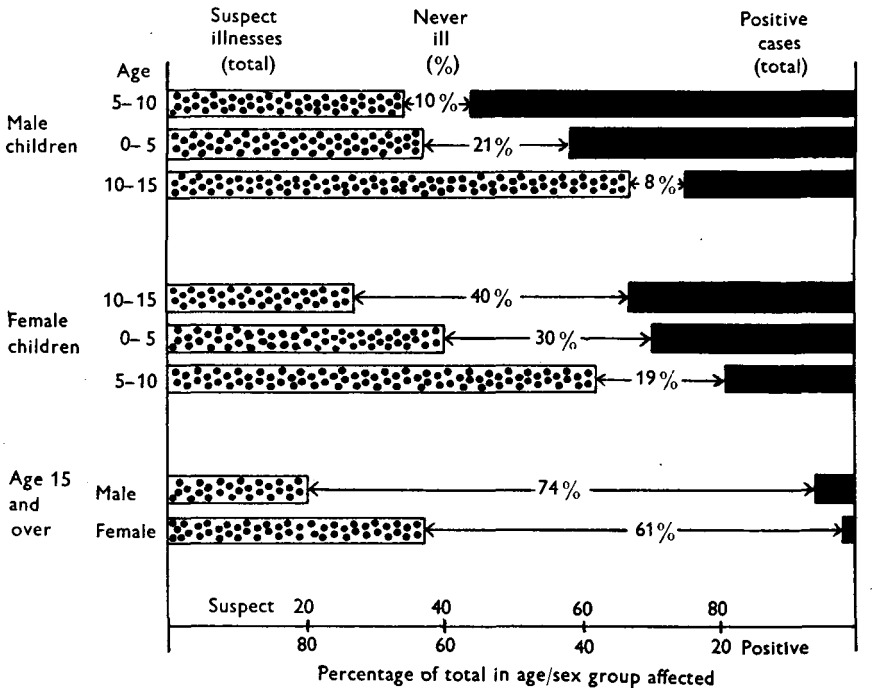

Fig. 8. Forty affected households: to show the percentage in each age/sex group not affected (either by suspect or positive illness) during period of observation.

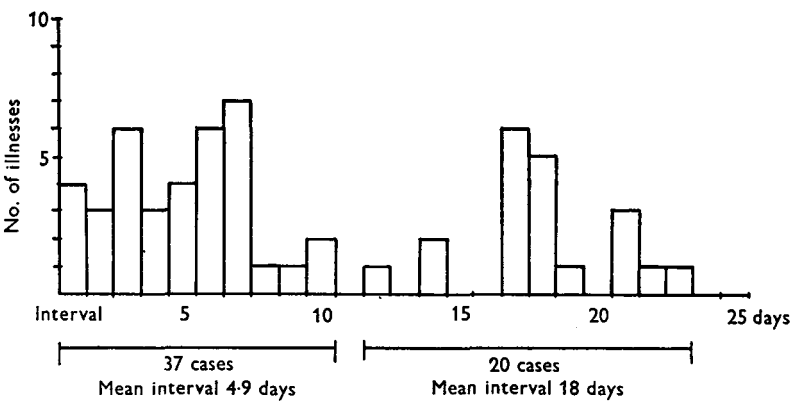

Fig. 9. Forty affected households: interval, in days, between successive illnesses in same household.

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might well be very long indeed, depending upon the function in the household of the source case and the standard of personal hygiene. When an attempt was made to 'correct' Fig. 5 by excluding the 16 cases in which the interval seemed excessively long (28–110 days), it was found that the neat correlation already noted between positive cases and suspect illnesses was upset, suggesting that even these cases were genuine instances of aberrant polio; but not, of course, excluding the possibility that they might have acquired their infection outside the home, so that the incubation period may not have been so long as it appeared to be.

A study was made of the age and sex of individuals who feel ill from 1 to 10 days after a previous case in the household, and those in whose case the interval was 12 days or more. There were 48 of the former and 28 of the latter, and the following table shows the percentage in each age/sex group:

	Males				Females			
	0–	5–	10–	15–	0–	5–	10–	15–
Short-interval infections: percentage in each group	*20·8*	20·8	8·3	6·3	12·5	10·4	*6·3*	14·6
Long-interval infections: percentage in each group	10·7	17·8	7·2	7·2	17·8	10·7	3·6	*25*

From this it would appear that male pre-school-children and girls aged 10–15 years have an excess of short-interval and female adults an excess of long-interval infections. The notion that the long interval denotes faecal infection might fit in with this observation, because it is on the mother that the duty of attending to the young or the sick child mainly falls, with a more than average risk of contamination with faecal organisms.

Forty cases may seem a small number upon which to base conclusions, however tentative. These cases, however, represented 74 % of the total notified in the metropolitan area in that period. It is probable, therefore, that, while it would be foolish to assume that the results have any wider application, they do represent with considerable accuracy what happened in the homes of positive cases falling ill in Auckland while the epidemic was at its height.

VII. THE BACKGROUND: PEAK AND DECLINE

(*a*) *Incidence in age and sex groups*

The Auckland urban area, with which this study has been concerned, has a population of just over a quarter of a million. During the 5 months November to March these produced a total of 94 confirmed cases of poliomyelitis, the peak in the first week of December (16 cases) being followed by an irregular and rather tardy decline.

It would be unwise to discuss the progress of an epidemic of poliomyelitis without having regard to the age and sex composition of the population concerned. I have tried to spare the reader by relegating the actual figures to the Appendix (Table 7), but in Figs. 10A and B he will find a picture of the course of the epidemic showing the number of cases per 15,000 in each age/sex group occurring each month. Males are shown in Fig. 10A, females in Fig. 10B. These charts also show, in parallel with the positive cases, the incidence of suspect illnesses in all families with children in the test and control areas. Suspect illnesses are shown per 100 in each age/sex group of the population concerned, the scale for positive cases having been exaggerated 150-fold for comparison.

It will be seen that the curves of incidence for all three are very similar. Control areas produced less illness than test areas, and it subsided more rapidly. It is tempting to attribute this, and the dearth of positive cases on the North Shore, to the measures imposed from the end of November onwards, but we cannot hope to prove it.

Table 8 shows the incidence of cases in the months November to March, by types of district.

The ratios for 'rural districts' probably err on the low side, as the population figure upon which they were based includes a large number of districts from which no cases were reported. Similarly, the 'semi-rural' ratios are probably too high, as the areas drawn upon are not completely covered by the population figures quoted. These two combined (4 and 5 in Table 8) give an incidence of 61·6 per 100,000.

This relatively high incidence in the rural population has been noted again and again in many parts of the world. An unusual feature in this epidemic is, however, the fact that the percentage of cases amongst persons aged 15 years or more is *lower* in the rural and semi-rural districts than in the city and its environs. This is the reverse of the usual finding.

(*b*) *Ratio of 'suspect' illnesses to positive cases*

Our test populations were deliberately selected in the near vicinity of positive cases. The controls were in parts of the urban area remote from the main incidence, and no positive cases occurred within their ambit. By averaging these suspect illnesses we get a fair picture of the whole area. In Table 9 this average incidence per 10,000 in each age/sex group has been compared with the incidence of positive cases, and the ratio of suspect to positive illnesses is shown.

It will be seen that the overall ratio is 300 suspect illnesses to every confirmed positive case, that the ratio is higher in females than in males and higher

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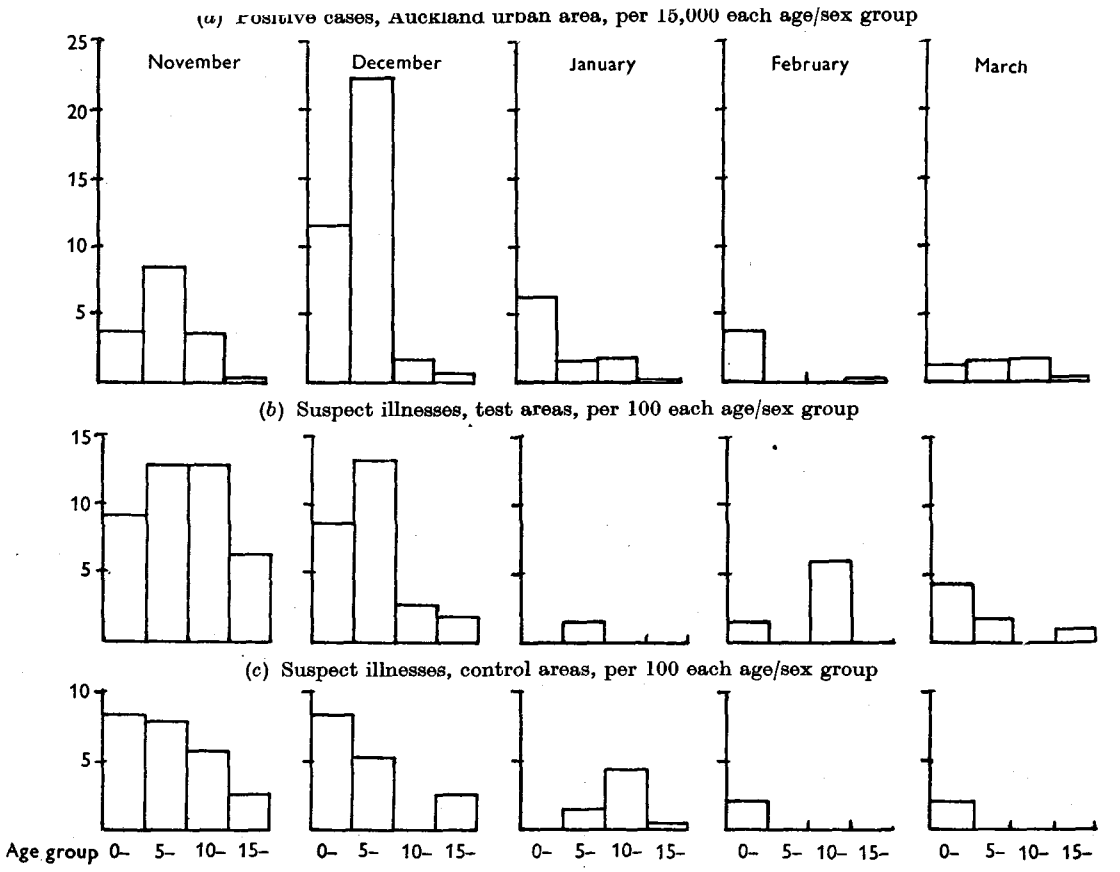

Fig. 10 (A). Males: positive cases in Auckland urban area, and suspect illnesses in test and control areas month by month
N.B. Scale for positive cases = 150 times scales for suspect cases.

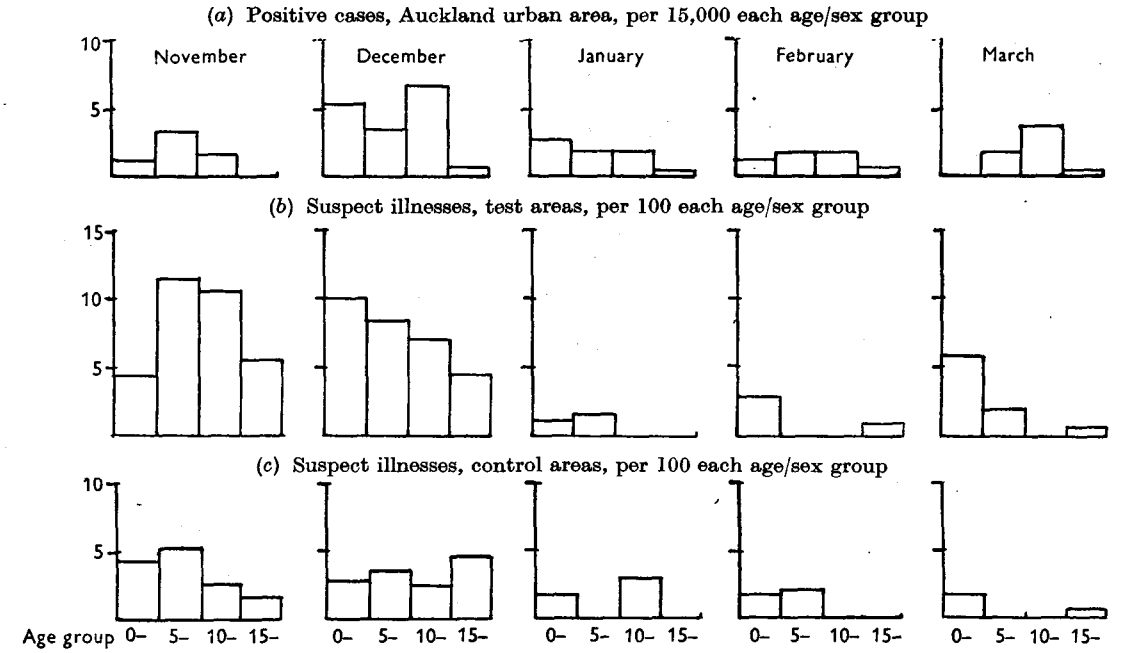

Fig. 10 (B). Females: positive cases in Auckland urban area and suspect illnesses in test and control areas month by month.
N.B. Scale for positive cases = 150 times scales for suspect cases.

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in adults than in children, and that it ranges from about 100:1 in young males to nearly 1000:1 in adult females. A curious feature is the comparatively low ratio amongst girls aged 10–15 years.

These ratios of suspect to positive cases are so high that it might be imagined that during the course of an epidemic the bulk of the population (at least in the lower age groups) must suffer from pliomyelitis in one form or another. This does not appear to be the case. In Fig. 11 the percentages in each age group affected by suspect and positive illnesses are shown, the scale for the latter being 100 times that of the former.

In the next diagram (Fig. 12), the populations in each age/sex group are charted and the calculated numbers of suspect illnesses in the period November to February are shown. On this scale the positive cases were too small in number to be indicated.

Table 8. *Incidence of positive cases in city, urban, semi-rural and country districts*

	Population 1 April 1947 (estimated)	Positive cases November to March	Ratio of cases to population	Cases per 100,000	Percentage of cases aged 15 years and over
1. Auckland Metropolitan Area, less North Shore	236,000	90	1 : 2,622	38·1	26·7
2. North Shore boroughs	29,660	4	1 : 7,415	13·5	25
3. Otahuhu and Papatoetoe	11,570	4	1 : 2,892	34·6	25
4. Other urban* (semi-rural) districts	14,950	20	1 : 747	133·8	20
5. Rural districts	54,860	23	1 : 2,385	41·9	17·4
Total	347,040	141	1 : 2,461	40·6	24·1

* The following districts have been excluded: Howick, Great Barrier Island, Waiheke, Warkworth and Helensville.

Table 9. *Ratio of suspect illnesses to positive cases, November to February inclusive*

Sex	Age	Suspect illnesses per 10,000	Positive cases per 10,000	Ratio Suspect positive cases	Round figures
Males	0–	1920	17·2	112 : 1	
	5–	2155	21·7	99 : 1	Male children: ratio 130 : 1
	10–	1595	5·0	319 : 1	
	15 and over	710	1·3	546 : 1	Males over 15 years: ratio 500 : 1
Females	0–	1460	7·2	203 : 1	
	5–	1630	7·1	230 : 1	Female children: ratio 200 : 1
	10–	1290	7·7	168 : 1	
	15 and over	875	0·9	972 : 1	Females over 15 years: ratio 1000 : 1
Total		991	3·29	301 : 1	Ratio 300 : 1

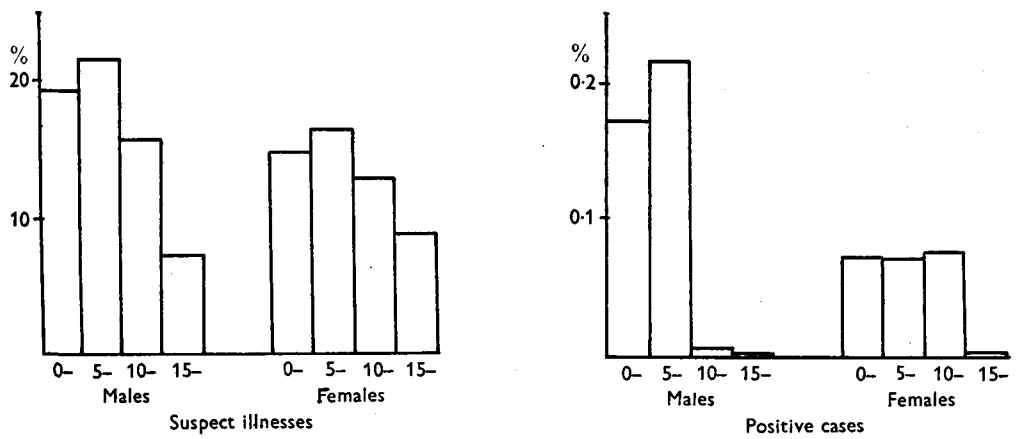

Fig. 11. Auckland urban area: percentage of each age/sex group affected by suspect or positive illness, November to February, 1947–48.

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It will be seen from Fig. 12 that even when the epidemic was at its most brisk, the proportion of each age group affected was so small that even with a generous allowance for missed cases it is impossible to believe that anything like a complete salting of *the population has taken place. By the end of July* there had been 131 confirmed cases. As a background to these there may have been about 40,000 minor illnesses related to polio. In 9 months, therefore, only about 15 % of the population were affected in any recognizable degree.

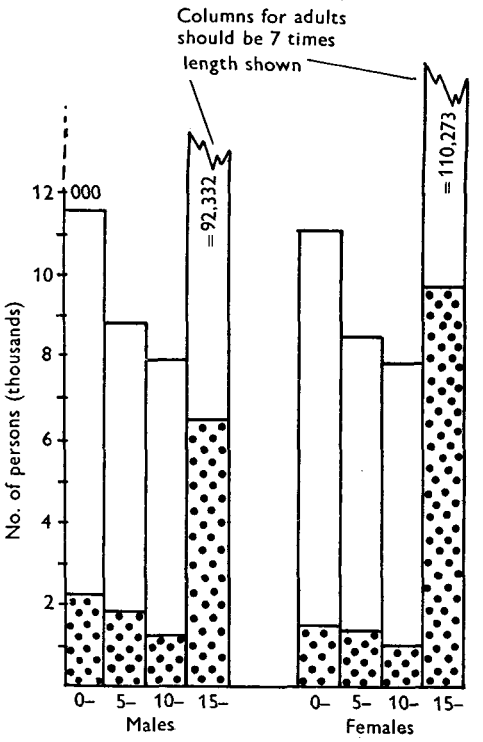

Fig. 12. Auckland urban area: number of persons in each age/sex group, and calculated number in each group who had a suspect illness November to February, 1947–8. (Scale too small to show positive cases.)

(c) *Leading symptoms, positive and suspect cases*

A study was made of the symptoms which had most impressed the parents or other members of the household in 50 positive cases, 78 illnesses in household contacts, and 170 illnesses in the test areas. These were the symptoms which were recalled by the relatives and mentioned to the investigator. The result is shown in Table 10.

An interesting feature was the infrequency of diarrhoea in positive cases (8 %) compared with suspect illnesses, and the small percentage of suspect cases in which nuchal pain or fever was noted as compared with positive cases. *In other words,* meningeal symptoms were commoner in positive cases, and intestinal symptoms in suspect illnesses. Drowsiness was only noted in 7 % of the test-area cases, or less than one-third as frequently as in the affected families.

VIII. DISCUSSION AND A SUGGESTED LINE OF INQUIRY

We have seen that what really took place in Auckland in 1947–8 was a very widespread epidemic of a comparatively trivial illness, totalling perhaps some 40,000 cases in the first 9 months. In the course of this otherwise unimportant outbreak, positive cases of poliomyelitis came to light as dramatic but infrequent episodes. It is further apparent that during the 4 months when positive cases were occurring in epidemic numbers, only comparatively small percentages of the total population at risk were affected. The most heavily attacked age group only suffered a little more than 20 % of casualties, and even in families in which positive cases had occurred we found no indication that *all* children in close contact had been affected. Some female schoolchildren in particular seemed to escape completely.

It has long been recognized that poliomyelitis tends predominantly to affect males. In 1932 an analysis of published data relating to 36,000 cases gave a male:female ratio of 1·3:1. Rhodes (1947) comments, however, that 'while this ratio holds for

Table 10. *Leading symptoms in positive and suspect illnesses*

	Positive cases		Contact illnesses		Test-area illnesses	
		%		%		%
In 30 % of cases or more	*Fever*	78	*Fever*	43	*Fever*	34
	Headache	44	Headache	30	Headache	30
	Pain in the neck	44	Sore throat	30	Sore throat	30
	Vomiting	38				
In 20–29 % of cases	Sore throat	24	Drowsiness	22	Vomiting	26
	Drowsiness	24			*Diarrhoea*	25
In 10–19 % of cases	Abdominal pain	12	Vomiting	19	Abdominal pain	16
			Diarrhoea	18	*Pain in neck*	16
			Pain in neck	15		

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younger children, it has been stated that at ages over 20 years there are more cases in females than in males'.

In our series the male : female ratio was 2·1 : 1 under the age of 15, and 1·2 : 1 above. With 'suspect' illnesses the ratios were 1·3 : 1 and 1 : 1·5 respectively.

Scrutiny of Figs. 11 and 12 will show that, in whatever form the epidemic is considered, male children were more heavily attacked than female children. The schoolgirl aged 10–15 years is in a peculiar position; this group produced fewer suspect illnesses than the corresponding male group, but more positive cases. In other words, her resistance (immunity?) is lower, but she is less frequently attacked.

It is clear, therefore, that poliomyelitis has a peculiar age and sex incidence which is not dependent on local or temporary conditions. In this investigation we have shown that the male schoolchild is especially liable to bring the infection into the household, and that once it is introduced it tends to pass from one member to another, not affecting all at the same time, but in turn, and that eventually a very high proportion of family contacts are attacked. When pondering on these facts, it suddenly occurred to the writer that there is another affection of childhood which behaves in a somewhat similar manner. I refer to infestation with threadworms. Recent research has thrown much light on this condition. It is now known to be far more common than was formerly believed. Reliable statistics of its age and sex incidence in normal population groups (as opposed to selected social groups, hospital patients, etc.) are difficult to obtain, but some interesting figures are available from the Peckham Health Centre (Pearse & Crocker, 1943). The population concerned is a good cross-section of a middle-class London suburban community, only whole families being admitted to membership of the Centre, and medical overhauls being obligatory. In Table 11 the percentages of each age group in which worm infestation was confirmed by laboratory examination are shown alongside our figures for 'suspect' illnesses in Auckland.

Table 11. *Comparison between incidence of threadworm infestation, Peckham, and 'suspect' illnesses, Auckland*

Age group	Peckham Health Centre: worm infestation						Auckland urban area— 'suspect' illnesses: percentage affected	
	Population concerned		Number with worms		Percentage infested			
	Male	Female	Male	Female	Male	Female	Male	Female
0–5	248	248	45	39	18·1	15·8	19·2	14·6
6–10	200	197	83	46	41·5	23·4	21·5	16·3
11–15	212	199	55	29	25·9	14·6	15·9	12·9
16 and over	1323	1375	53	69	3·9	5·0	7·1	8·7

A preponderance of male children, especially after school entry, and a higher proportion of female adults affected than of males, are features alike of the Peckham figures and of our records of 'suspect' illnesses in Auckland. It will be noted, also, that Rhodes's comments on the general incidence of poliomyelitis, quoted above, would apply equally well to the Peckham figures for worm infestation. (Females : males = 1·3 : 1. Females exceed males after age 20).

The resemblance between the behaviour of *Enterobius vermicularis* and of the virus of poliomyelitis does not stop, however, at a mere similarity of age and sex incidence. If we consider the following facts about threadworm infestation we are reminded at every point of similar features of poliomyelitis. I hasten to add that it is not suggested at this stage that there is any connexion between them, but rather that the epidemiology of one may throw light on that of the other.

E. vermicularis appears, as a parasite, to be strictly confined to man as its host. Failure has attended all attempts to infest guinea-pigs, mice, dogs and rhesus monkeys (Cram, E. B., 1943). We have here, then, a bowel parasite of man alone which so far as is known has no intermediary. One would expect that its incidence could be correlated with defective sanitation, but such is not the case, and, like poliomyelitis, this is one of its most puzzling features. In America, for example, of about 4000 persons examined, the percentages found to be infested were:

2895 Whites	41·5 % infested
1099 Negroes	12·9 % infested

Other writers (Miller & Einhorn, 1944) enlarge on their failure to find infestation in negroes and mestizos in the proportion which would be expected from their low standards of cleanliness, and admit that to date no satisfactory explanation has been forthcoming.

A description of the incidence of threadworms in the United States of America has a familiar ring. It was found to be highest in schoolchildren, lowest in adults, and intermediate in pre-schoolchildren

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(Cram, E. B., 1944). There was evidence that if young children attended nursery schools, their incidence rose until it equalled that of children of school age. The highest incidence recorded was in a group of 504 boys aged 6–16 years, of whom 57 % were infested.

When families in which one member was known to be infested were investigated (cf. our studies of household contacts of poliomyelitis cases), an unusually high incidence was found. In 286 white families, 1353 persons were examined; 72 % of the children and 36 % of the adults were infested. In 34 negro families, 172 persons were examined; 51% of the children, *but only* 7 % *of the adults*, were infested. A leading authority concluded that 'even stringent measures cannot be relied on to control the infection'.

All this is reminiscent of some of the more puzzling aspects of poliomyelitis. Growing emphasis has been laid in recent years on the role of faecal organisms in the spread of the disease. It seems possible, therefore, that something might be learned, by analogy, if we look further into the threadworm question.

This leads us to a startling aspect of the epidemiology of threadworms, only recently recognized, but already well established as a fact on both sides of the Atlantic. It is now known that where threadworm infestation is common (and where is it not?) the eggs can be recovered in large numbers from the dust in schools and in homes, where they seem to be blown about everywhere. The height above the floor of the surface examined is immaterial. In the homes of infested families they have been found in all rooms, and at all levels from the floor to the ceiling lights. In the United States of America, in seven infested households, 91·7 % of 241 samples of dust yielded eggs. The largest numbers were found in the bedrooms, and about half of the eggs were viable or had recently been so.

In Amsterdam, where all children are said to be infested, the number of eggs found* on a square foot of surface was 119 in a school dining-hall, 305 in classrooms, and 5000 in closets. More eggs were found in girls' closets, but girls were not more heavily infested than boys. The main source of the eggs appeared to be the anal region, where they are deposited by the female worm during the hours of sleep. Movements of the clothing cause them to be rubbed off, and they are then spread about in the dust. Bedmaking was noted as being favourable to dissemination. Dust-borne spread has been accepted as an important source of light infestation, thereafter convertible by finger transference into a severe and active condition.

Does this throw any light on the poliomyelitis problem? I think it may. We know that the virus of poliomyelitis is principally eliminated in the faeces and that contacts and persons who have had abortive attacks may harbour it for weeks or months in the intestine. We know that it may retain its infectivity in dried faeces for a long time. If, therefore, the relatively enormous ova of *Enterobius* have been found so readily in the dust in schools and in private houses and have been proved to be blown about indoors and to be liable to inhalation or swallowing, surely it is reasonable to suppose that spread of the poliomyelitis virus may well take place in the same way—possibly at the same time. The ova themselves might be contaminated with infective virus; tiny particles of dried faeces, derived in a similar manner from the anal region of a poliomyelitis carrier, almost certainly must be, at least occasionally.

It is possible that if it can be shown that the dust in school closets during an epidemic contains appreciable quantities of the active virus, this may also supply a clue to another mysterious feature of poliomyelitis. Why do epidemics nearly always commence in summer? Might the reason not be that certain physical conditions existing then, of temperature, humidity, and dust promotion, may for a short time be particularly suited to the survival and dissemination of the virus in an infective form by the means suggested above?

Without disputing the importance of other modes of transfer, it is, I suggest, possible that indoor dust spread, in schools in particular, may be the type best suited to touching off an epidemic in a population happening to be ripe for it, by causing a more rapid and wider circulation of the virus. This might lead first (as we have seen) to an increasing number of minor illnesses in schoolchildren, especially in boys aged 10–15 years, and later, when the virulence became exalted by passage, to recognizable cases, falling most heavily on the age groups which previously suffered least from the disease in its milder form. Infections acquired in this manner might generally be light (as seems to be the case with threadworms), and neglected personal hygiene might be necessary in most cases to boost them to a clinical level. This might explain why the older schoolgirl, perhaps the most fastidious member of the community, usually escapes lightly, while the adult woman (who makes the beds and attends to the younger child) is relatively heavily attacked.

I submit that a *prima facie* case has been made out for considering that this manner of spread, analogous to that of threadworms, is a possibility, and perhaps an important one. That the threadworm itself might be intimately concerned in the life history of the virus may seem a fantastic suggestion, but it cannot be excluded on grounds of speculation. So far as I know, the idea now put forward has not been considered before, and it at least deserves inquiry.

If it should be found to contain any truth, it opens up a new line of attack upon the disease. If it turns

* See 'Threadworms', *Lancet*, 18 May, 1946, p. 742—various references.

out to be a really important factor, the whole of our policy of poliomyelitis prophylaxis may have to be revised.

IX. CONCLUSIONS

(*a*) The Auckland poliomyelitis epidemic of 1947–8 was preceded and accompanied by large numbers of cases of minor illness in the general population, of varying severity, characterized by (in order of frequency) fever, headache, sore throat, vomiting, diarrhoea, and sometimes pains in the abdomen or neck.

(*b*) In the homes of positive cases, these 'suspect illnesses' in contacts were found to bear a close inverse mathematical relationship to the positive cases in the same age/sex groups.

(*c*) The monthly curves of incidence of these 'suspect illnesses' in the general population followed a similar pattern to the incidence of positive cases, not only in districts where positive cases were occurring, but also in parts of the city where there was little or no obvious prevalence of poliomyelitis.

(*d*) The ratio of 'suspect illnesses' to positive cases was very high. The over-all ratio was about 300:1. In male children it ranged from 100 to 300:1, in female children from 150 to 250:1, and in adults from 500:1 (males) to 1000:1 (females).

(*e*) Notwithstanding these high ratios, only comparatively small proportions of the population suffered any obvious illness, 'suspect' or positive. In the four months when the epidemic was most active the male age group 5–10 years was the most heavily attacked, but it was calculated that only about 22% of children in that age group were affected during this time. This is despite the fact that the incidence of positive cases in Auckland urban area, November to March inclusive, reached 38 per 100,000, which is generally regarded as a high figure.

(*f*) The following facts suggest that when individuals come unscathed through an outbreak of poliomyelitis, the primary factor is *not* (as has generally been assumed) immunity gained in a previous epidemic:

(1) During this widespread and moderately severe epidemic less than 1:4 in any age group of the general population was affected in any way.

(2) Amongst those in closest contact with positive cases, significant numbers of young people could be found who, after searching inquiry, appeared to have escaped completely.

(3) Between boys and girls of the same age there appeared to be a notable difference in susceptibility in favour of the latter.

(4) In rural areas, although the incidence was much higher than in the city, the percentage of cases aged 15 years or over was lower.

(*g*) Of equal, or greater, importance may be the degree of exposure to infection, and possibly the stage of the epidemic at which infection is first encountered. It seems that infection may not be acquired so easily as is generally believed, and that mode of life and personal habits may be deciding factors. Some immunity appears to have been conferred by minimal infections early in the epidemic—e.g. amongst older schoolboys and possibly adults. See next paragraph, and (*k*) below.

(*h*) The epidemic was preceded by an increase of illnesses of the type mentioned in (*a*) above, at first mainly of short duration (under 4 days), the chief prevalence being amongst adults.* As the epidemic approached its peak, however, 'suspect' illnesses showed their highest prevalence in schoolchildren, especially in the vicinity of positive cases, and there was a marked increase in the duration of illness and in the number of suggestive symptoms observed.

(*i*) Serial spot maps of positive cases showed no indication of spread from any particular focus. It must be presumed, therefore, that in the form of 'suspect' illnesses the disease had already established itself widely before the appearance of positive cases revealed its presence. Investigation showed that this unobtrusive spread of 'suspect' illnesses had occupied a period of some weeks at least, during which only a single positive case occurred, date of onset 25 October; yet so widespread had the infection become by the middle of November that a very large number of individuals must already have been involved. In the first 4 weeks of the overt epidemic (9 November to 6 December), 30 positive cases occurred in the Auckland urban area, and the peak of the epidemic was reached in the fourth week. These facts would be consistent with an increase in the virulence of the organism during October and November.

(*j*) A very high percentage of the household contacts of positive cases had a 'suspect' illness during the period of observation. Before, or concurrent with, the onset of the positive case, 50% of two age groups (males aged 10–15 years, females aged 5–10 years) in the affected households had a 'suspect' illness. Schoolchildren, particularly boys aged 10–15 years, were most frequently responsible for introducing the infection into the family. Amongst children the age groups most affected by 'suspect' illnesses beforehand produced fewest positive cases, but females, on the whole, suffered less than males, and adults very much less than children.

(*k*) The intervals between the dates of onset of successive illnesses (suspect or positive) in the same household ranged from 1 day to as much as 110 days. Half of them ranged from 1 to 10 days, half were of 12 days or more. A tendency to grouping about the

* For reasons mentioned on p. 87, however, least reliance can be placed in this investigation on records of illness amongst adults in childless families.

fifth day and the eighteenth day was noted. The facts pointed either to a comparatively poor capacity of the organism to pass from person to person in the home, or to a remarkable degree of variation in the incubation period. The former was considered to be the more likely.

(*l*) 'Suspect' illnesses in the general population followed the usual poliomyelitis pattern in their age and sex incidence. Males under the age of 15 were more affected than females of the same age, the preponderance being more marked after school entry than before; over the age of 15, females suffered rather more severely than males. Attention is drawn (see preceding section) to the fact that a similar age and sex incidence has been noted in different parts of the world in regard to threadworm infestation. The latter has, epidemiologically, some curious points of resemblance to poliomyelitis, and it is suggested that recent discoveries about its mode of spread may supply clues to some hitherto unexplained features of the more serious disease.

The task of the investigator is to establish facts. It is not a primary object of this paper to suggest the manner in which its conclusions might be translated into practice. One could not help being impressed, however, with the emphasis which the findings throw on the role of the schoolchild in spreading infection and introducing it into the home—particularly the older schoolboy; on the likelihood of faecal organisms, rather than droplet infection, playing the major part in propagating the disease; and on the supreme importance of personal hygiene over all possible general measures of control. With regard to these last, however, the chief point of practical importance which emerges is that a particular kind of public assembly is especially to be avoided in epidemic times—that is, any in which children are collected together, use closets in common, and take food. Day schools and children's parties are therefore much more dangerous than casual contact in trams, buses, shops, or in the streets. It is to be noted, however, that we have shown that by the time we are made aware of the presence of an epidemic the infection has already become widespread. School closure, therefore, unless it is prompt, can only be of limited value. That it definitely was of value in the present instance I am convinced. Experience of this measure in England or the United States of America has little interest for us in New Zealand, because conditions here are different from those in any other country. I doubt if the public in Britain would respond as well to the requirements of the health authorities as they did in Auckland, and there is no city in England whose average living conditions are at all comparable or which has such an abundance of private gardens and open spaces where children can enjoy the fresh air without coming into contact with others outside the family circle.

When the schools reopened in Auckland, a note from the Health Department was read out to all classes warning the children (amongst other things) that they must be careful to wash their hands before lunch. When lunch-time came, in one school at least, some of the older boys were heard to say that '*they* weren't going to wash their hands—only sissies did that'. This, as we have seen, is the group chiefly responsible for spreading the infection—and no wonder!

X. SUMMARY

(*a*) A field investigation into an outbreak of poliomyelitis in Auckland in the summer of 1947–8 is described.

(*b*) The object of the investigation was to study the background of the epidemic in the general population, to discover how it had commenced, and how much minor illness related to poliomyelitis had occurred (i) amongst household contacts, (ii) in other persons residing in the same neighbourhood, and (iii) in those parts of the urban area least affected by the epidemic.

(*c*) Conclusions reached have been detailed in the preceding section. A relationship was established between minor ('suspect') illnesses and positive cases; the ratio of 'suspect' to positive cases at different age levels has been calculated, and an estimate is given of the extent to which the population as a whole was affected. Reasons are adduced for believing that, during an epidemic, mode of life and personal habits may be of more importance in determining the fate of the individual than previously acquired immunity.

(*d*) It is suggested that indoor dust-borne infection, at school and in the home, may be important, and that the results of recent research into threadworm infestation may throw light on this factor.

APPENDIX

Table 1. *Composition of test and control areas by types of dwellings and families* (see p. 84)

Areas	Total houses visited	Class of House I	II	III	IV	Persons Male	Female	Total	Adults	School-children	Pre-school-children
				A. All categories							
(*a*) Test	300	33	224	41	2	566	567	1133	732	246	155
(*b*) Controls	303	30	237	34	2	548	553	1101	717	256	128
				B. Families without children							
(*a*) Test	118	16	83	19	—	140	157	297	297	—	—
(*b*) Controls	126	17	99	10	—	136	159	295	295	—	—
				C. Families including only pre-schoolchildren							
(*a*) Test	39	4	31	4	—	72	71	143	89	—	54
(*b*) Controls	33	2	25	6	—	64	62	126	79	—	47
				D. Families including schoolchildren							
(*a*) Test	143	13	110	18	2	354	339	693	346	246	101
(*b*) Controls	144	11	113	18	2	348	332	680	343	256	81

NOTE. Classification of houses (social and economic): I, superlative; II, good; III, fair; IV, bad.

Table 2. *Age and sex composition of first* 1000 *persons in test and control areas* (see p. 84)

Age group	0–	1–	2–	3–	4–	5–	6–	7–	8–	9–	10–	11–	12–	13–	14–	Over 15
Males:																
Test areas	10	15	17	20	15	16	19	13	11	7	6	7	12	8	8	319
Control areas	9	11	16	10	12	13	16	12	9	20	11	11	7	7	9	323
Females:																
Test areas	13	14	14	9	9	12	12	18	13	7	8	2	5	2	5	354
Control areas	13	13	9	12	10	9	13	11	13	5	5	7	6	11	8	359

Table 3. *Analysis of illnesses recorded in test and control areas at first visit* (see p. 85)

No. of 'cardinal' symptoms	Before 1 November: Duration less than 4 days Test	Controls	Duration 4 days+ Test	Controls	After 1 November: Duration less than 4 days Test	Controls	Duration 4 days+ Test	Controls
Five	—	—	—	—	—	—	1	—
Four	—	—	—	—	1	1	4	—
Three	1	1	3	1	5	5	10	5
Two	7	3	6	7	28	16	12	10
One	15	6	3	4	30	28	14	11
Indefinite	7	7	3	1	10	7	4	1
Total	30	17	15	13	74	57	45	27

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Table 4. *Distribution of illnesses recorded at first visit, by types of family* (see p. 87)

Families ...	Illnesses, duration under 4 days						Illnesses, duration 4 days or over					
	Adults only	Pre-school only		With school-children			Adults only	Pre-school only		With school-children		
	A* (295)	A (79)	P (47)	A (343)	S (246)	P (81)	A (295)	A (79)	P (47)	A (343)	S (246)	P (81)
Before 1 November:												
Test areas	12	3	1	8	5	1	4	0	4	3	4	0
Controls	0	0	3	6	5	3	3	1	1	5	2	1
Tests	12	4		14			4	4		7		
Controls	0	3		14			3	2		8		
After 1 November:												
Test areas	6	9	8	15	34	2	4	4	2	11	18	6
Controls	10	0	3	15	21	8	8	4	4	3	8	0
Tests	6	17		51			4	6		35		
Controls	10	3		44			8	8		11		

* 'A' = adults; 'P' = pre-schoolchildren; 'S' = schoolchildren.

Table 5. *Forty affected households: positive cases, and suspect illnesses, in age/sex groups* (see p. 87)

		Number			Per 100 in each age/sex group		
			Contacts ill			Contacts ill	
	Age group	Positive cases	Before or concurrent	After positive case	Positive cases	Before or concurrent	After positive case
Male children	5–	15	4	5	56	15	19
	0–	8	6	1	42	32	5
	10–	3	6	2	25	50	17
Female children	10–	5	4	0	33	27	0
	0–	6	6	2	30	30	10
	5–	3	8	2	19	50	12
Aged 15 and over	Male	3	6	4	6	12	8
	Female	1	9	12	2	16	21

Table 6. *Forty affected households: time relationship between onset of positive case and illnesses in contacts* (see p. 88)

			Ill before positive case			Concurrent		
Sex	Age	Total persons	More than 2 weeks	2–4 weeks	7–14 days	Contacts	Cases	After
Males	0–	19	—	1 (5%)	—	5 (26%)	8 (42%)	1 (5%)
	5–	27	1 (3%)	1 (4%)	1 (4%)	1 (4%)	15 (56%)	5 (18%)
	10–	12	2 (17%)	2 (17%)	2 (17%)	0 —	3 (25%)	2 (17%)
	15–	52	1 (2%)	1 (2%)	2 (4%)	2 (4%)	3 (6%)	4 (8%)
Females	0–	20	—	2 (10%)	1 (5%)	3 (15%)	6 (30%)	2 (10%)
	5–	16	1 (6%)	2 (12%)	1 (6%)	4 (25%)	3 (19%)	2 (12%)
	10–	15	1 (7%)	2 (13%)	—	1 (7%)	5 (33%)	—
	15–	57	—	4 (7%)	2 (4%)	3 (5%)	1 (2%)	12 (21%)

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Table 7. *Positive cases and 'suspect' illnesses in Auckland urban area, month by month* (see p. 91)

(*a*) Positive cases, Auckland urban area

Sex	Age	Population (1945)	Positive cases					Cases per 15,000				
			Nov.	Dec.	Jan.	Feb.	Mar.	Nov.	Dec.	Jan.	Feb.	Mar.
Males	0–	11,619	3	9	5	3	1	3·9	11·5	6·4	3·9	1·3
	5–	8,772	5	13	1	—	1	8·5	22·2	1·6	—	1·6
	10–	7,965	2	1	1	—	1	3·7	1·9	1·9	—	1·9
	15–	92,330	2	6	1	3	2	0·3	0·9	0·1	0·4	0·3
Females	0–	11,184	1	4	2	1	—	1·3	5·4	2·7	1·3	—
	5–	8,500	2	2	1	1	1	3·6	3·6	1·8	1·8	1·8
	10–	7,822	1	3	1	1	2	1·9	5·7	1·9	1·9	3·9
	15–	110,273	—	4	2	4	1	—	0·6	0·3	0·6	0·1

(*b*) Suspect illnesses, per 100 in each age/sex group, test and control areas

Sex	Age	Test areas					Control areas				
		Nov.	Dec.	Jan.	Feb.	Mar.	Nov.	Dec.	Jan.	Feb.	Mar.
Males	0–	9·1	8·9	—	1·4	4·2	8·5	8·5	—	2·0	2·0
	5–	13·2	13·2	1·6	—	1·6	8·1	5·4	1·6	—	—
	10–	13·2	2·7	—	5·7	—	5·9	—	4·4	—	—
	15–	6·3	2·0	—	—	1·1	2·7	2·7	0·5	—	0·5
Females	0–	4·3	10	1·4	2·9	5·9	4·3	2·9	1·7	1·7	1·7
	5–	11·6	8·6	1·6	—	1·6	5·3	3·5	—	2	—
	10–	10·7	7·1	—	—	—	2·5	2·5	3	—	—
	15–	5·5	4·5	—	1·0	0·5	1·7	4·8	—	—	0·5

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