

ESTIMATES OF THE INFECTION RATES FOR POLIO-
MYELITIS VIRUS IN THE YEARS PRECEDING THE
POLIOMYELITIS EPIDEMICS OF 1916 IN NEW YORK
AND 1945 ON MAURITIUS

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(With 2 Figures in the Text)

INTRODUCTION

Three important considerations in the epidemiology of any contagious disease are:

- (1) The proportion of susceptibles required to permit spread of the infecting agent in a given population.
- (2) The infection rate resulting from spread of the agent in that population.
- (3) The proportion of susceptibles remaining in that population when spread of the agent terminates.

Evans, Chambers, Giedt & Wilson (1957), have presented data concerning the third point relating to poliomyelitis in Ketchikan, Alaska. It is noted that relatively little information of this kind is available in the literature about poliomyelitis. Most of the available data are derived from surveys of the incidence of antibodies to Type 2 virus which is rarely implicated as the cause of epidemics. A serious limitation to serological studies is the relatively small number of specimens tested. Simple statistical analyses of the inherent errors of random sampling have rarely been included in reports of antibody surveys. To do so would raise doubts as to some of the conclusions reached.

Criteria used in determining suitability of epidemics for analysis

Appropriate analyses of age-specific attack rates during properly selected epidemics provide another approach to the problem of estimating the three values referred to above. If age-specific attack rates are to be used for this purpose, there are several important considerations in selecting the epidemics to be analyzed:

- (1) Data should concern a population group that is clearly definable.
- (2) Cases of poliomyelitis should be so distributed as to give confidence that the virus was widely distributed in all major parts of the population.
- (3) Errors of random sampling should be as small as possible. To this end, the data should concern as many cases as possible and as large a population as possible.
- (4) Data for numbers of cases must be available for age groups of 1 year.
- (5) Estimates of population by age groups of 1 year must be sufficiently reliable so that the error in these data do not seriously alter the conclusions reached. A considerable latitude is permissible.

(6) It is preferable that analyses be based on paralytic cases only. The uncertainty of diagnosis is considerably greater for non-paralytic cases than for cases regarded as having paralytic involvement.

Selection of epidemics for inclusion in this study

In reviewing published information for purposes of this study, attention was concentrated chiefly on those epidemics in which the paralytic attack rate constituted more than 1% of the population of the age groups most heavily involved. Data concerning some of the epidemics considered are shown in Table 1.

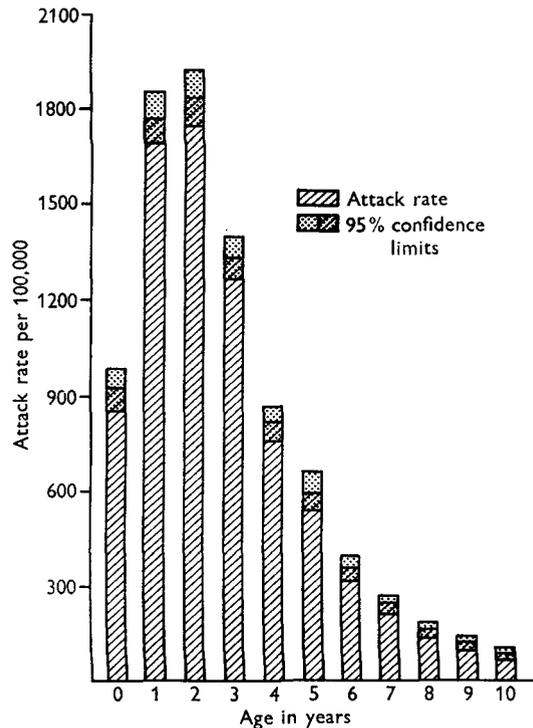


Fig. 1. Age-specific attack rates in the poliomyelitis epidemic of 1916 in New York.

It can be seen that most of the exceptionally severe epidemics occurred in small population groups. After considering the available data for each epidemic in the light of the six points listed previously, it was decided that the New York epidemic of 1916 and the Mauritius epidemic of 1945 were the most favourable for this study. The epidemic of 1916 in New York City represents the most cases of poliomyelitis that have ever occurred at one place and one time, and from a statistical viewpoint provides the most accurate age distribution of cases on record for a large population that is closely associated in a definable area. The epidemic on the Island of Mauritius in 1945 combined a high attack rate with a population of moderate size and from a statistical viewpoint gives data which are only fair, but nevertheless adequate for present purposes and useful in view of the paucity of better data. The attack rates for 1 year age groups and their 95% confidence limits for these two epidemics are presented in Tables 2 and 3 and Figs. 1 and 2.

Table 1. *Attack rates in the total population and in age groups most heavily involved in some exceptionally severe epidemics of poliomyelitis*

Epidemic	Reference	Total cases*	Total population	Total attack rate in %	Age groups with high attack rate	Cases these ages	Estimated population these ages	Attack rate these ages in %
Guam, 1899		—	10,000	3.4	All	—	10,000	3.4
Polk County, Nebraska, 1909	Grunwell, 1900 Anderson, 1910	279†	10,521	2.7	0.9 yr.	234‡	2,350‡	10
Nauru, 1910	Müller, 1910	470†	1,250	37.6	All	470	1,250	37.6
Sukkertoppen, Greenland, 1914	Hrolv, 1935	43	700	6.1	All	43	700	6.1
New York City, 1916	Lavinder, Freeman & Frost, 1918	9131	5,278,782	0.17	1-3 yr.	5370	326,004	1.6
Iceland, 1924	Sigurjonsson, 1950	463	98,483	0.5	0-4 yr.	148	11,523	1.3
Angmagssalik, Greenland, 1925	Høygaard, 1939	27	800	3.4	All	27	800	3.4
Sukkertoppen, Greenland, 1932	Hrolv, 1935	66	2,263	2.9	0-17 yr.	66	600‡	11
Malta & Gozo, 1942-43	Seddon, Agius, Bernstein & Turnbridge, 1945	426	271,359	0.16	0-3 yr.	364	16,000	2.3
Mauritius, 1945	McFarlan, Dick & Seddon, 1946	1018	419,185	0.24	1-5 yr.	614	47,990	1.3
St Helena, 1945-46	Nissen, 1947	77	4,000	1.9	0-29 yr.	72	1,700‡	4.2
Car Nicobar, 1947	Moses, 1948	566	8,722	6.5	0-35 yr.	564	6,857	8.2
Chesterfield Inlet, 1949	Pearl, 1949	57	275	20.7	All	57	275	20.7
Wythe County, Virginia, 1950	Previously unpublished data received from A. S. McGowan, M.D.; Department of Health, Commonwealth of Virginia	184†	23,327	0.8	0-12 yr.	145	6,757	2.1

* Unless noted otherwise, the cases were nearly all paralytic.

† Includes an appreciable proportion of non-paralytic cases.

‡ No specific data were found for total population in age groups with high attack rate in these epidemics. Figures listed are estimates of the present authors based on age ratios in other communities. Note that errors of 50% or more in this estimate would not make an important difference in the significance of these data relative to the present discussion.

Table 2. *Poliomyelitis age-specific attack rates in New York City, 1916*

Age in years	Population	Cases*	Attack rate per 100,000	95% confidence limits of attack rate
0	109,600	1014	930	868-984
1	104,800	1857	1770	1690-1850
2	112,200	2062	1840	1760-1920
3	108,900	1451	1330	1260-1400
4	103,800	847	820	760-870
5	102,400	607	590	545-640
6	101,700	360	350	320-390
7	100,300	241	240	209-271
8	97,700	157	160	135-187
9	95,000	118	124	101-147
10	96,800	80	83	64-102
10-14	452,600*	225	50*	—
15-19*	484,200*	78	16*	—

* These data are taken directly from Lavinder, Freeman & Frost (1917). Other data are calculated as explained in the text.

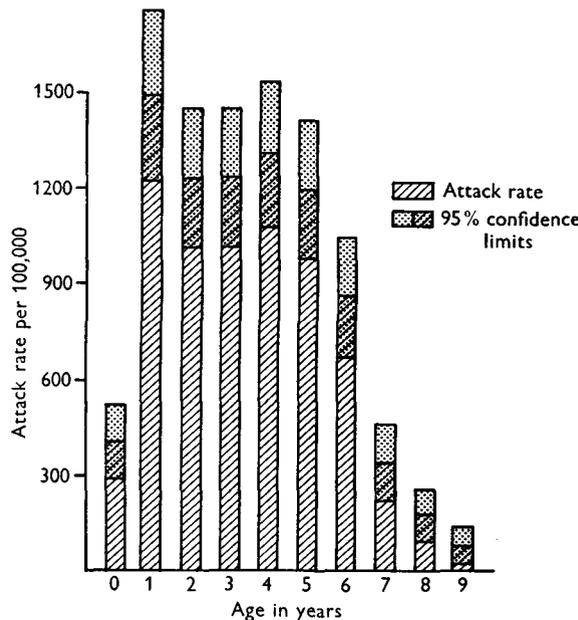


Fig. 2. Age-specific attack rates in the poliomyelitis epidemic of 1945 on Mauritius.

ANALYSIS OF THE NEW YORK CITY EPIDEMIC OF 1916

(a) Sources of data

Lavinder, Freeman & Frost (1918) in their monograph on the New York City epidemic of 1916 give data for the total population, number of cases, and attack rates for children in the following age groups—those under 1 year of age, those from 1 year to 4 years of age inclusive, and older children arranged in age groups of 5 years. These data are given for each of the boroughs and for the entire city. In addition, the authors give the number of cases by age groups of 1 year. They do

Table 3. *Poliomyelitis age-specific attack rates, Mauritius 1945*

Age in years	Cases	Attack rate per 100,000	95 % confidence limits of attack rate
0	48	400	285-515
1	119	1490	1220-1760
2	123	1230	1010-1450
3	123	1230	1010-1450
4	130	1300	1070-1530
5	119	1190	970-1410
6	86	860	675-1045
7	34	340	220-460
8	17	170	90-250
9	7	80	25-140
10-14	19	40	---
15-19	8	20	---
20-24	11	30	---
25-29	7	4	---

Data from McFarlan, Dick & Seddon (1946).

not give figures for total populations within age groups of 1 year or attack rates for age groups of 1 year.

In the present analysis the population of each age group in 1916 has been estimated by interpolation from the populations determined in the censuses of 1910 and 1920. For example, the number of children 1 year old in 1910 was 96,819; the number in 1920 was 110,192. Therefore, the estimate used for the population of 1-year-olds in 1916 is $96,819 + 0.6(110,192 - 96,819)$ or 104,843. This is rounded off to 104,800 in the tables. A high degree of accuracy in the estimates of population is not required for the present purposes. A change of 10,000, for example, in the estimated population of any age group or of all age groups in New York would not alter materially the conclusions that will be reached.

(b) *Calculation of infection rates* prior to 1916*

It is evident from the data in Table 2 and from Fig. 1 that the highest attack rate occurred in children 2 years old. In a total population of 112,000 2-year-olds, there were 2062 cases. This is an attack rate of 1840 per 100,000. The attack rate in 1-year-olds was slightly less than that in 2-year-olds. The difference was not significant ($\chi^2 = 1.42$, $P = 0.22$).

We are interested primarily in the explanation for the progressively lower attack rates in successive age groups of children 2 years old and older. The attack rate in 3-year-olds was 28 % lower than that in 2-year-olds. The attack rate in 4-year-olds was 38 % lower than that in 3-year-olds. Similarly, the attack rates in 5-, 6- and 7-year-olds were 28, 40 and 31 %, respectively, lower than those in the next younger age groups.

These data for our present purposes are to be interpreted as indicating that the

* Infection rate will be used to signify rates of infection that induced active immunity sufficient to protect from paralytic disease in 1916. Infections that did not immunize do not enter into these considerations.

immune component of the population in the successively older age groups was larger by virtue of immunizing infections during each of a series of previous years. The attack rate among 8-year-olds was 160, slightly less than 10% as large as that in 2-year-olds, 1840. It may be inferred that infections occurring over a period of 6 years immunized approximately 90% of the children born in 1907. The average annual infection rate required to immunize 90% of a population in 6 years (the difference in age between 2-year-olds and 8-year-olds) is slightly more than 30% per year. An infection rate of 30% per year would involve 88% of a population in 6 years. It is postulated that New York children who were 8 years old in 1916 had experienced annual periods of prevalence of poliomyelitis virus* during 6 successive years, and that on the average approximately 30% of those susceptible at the beginning of the year had been infected and immunized by the end of the year. Younger children had had the same degree of immunizing experience each year for proportionately fewer years.

ANALYSIS OF THE MAURITIUS EPIDEMIC OF 1945

(a) Source of data and description of Mauritius

An extensive description of the Mauritius epidemic of 1945 has been published by McFarlan, Dick & Seddon (1946), and our data are taken largely from their paper. More than 1000 cases occurred, 96% of which were paralytic. There were fifty-eight deaths.

Mauritius is an island, approximately 35 miles in average diameter, lying in the Indian Ocean about 600 miles east of Madagascar. Its population according to a census taken 11 June 1944, was 419,185. It was composed of three major groups of different origin—10,882 Chinese, 265,247 Indians (Hindus and Moslems), and 143,056 of the so-called 'general population' derived primarily from native African or Malagasy origin.

Sanitation was poor all over the island. Overcrowding in Mauritius was great, as was shown by the 1944 census. It was particularly marked in Port Louis where only the poorer classes lived.

(b) Description of the epidemic

The epidemic occurred principally in the month of March. Less than 10% of the recorded cases developed before 1 March, and more than 90% had their onset before 1 April. A group of investigators including A. M. McFarlan, G. W. A. Dick and H. J. Seddon visited the island, and a field survey was conducted 17 April–31 May. Data were obtained by personal interviews, by persons of this group or their representatives, with 773 patients. Records of a total of 1018 patients were obtained by interview and less direct means.

Analyses of attack rates in towns, in rural areas, and by districts showed that the disease was prevalent in all parts of the island with populations sufficiently large to make the data reliable. The observed differences in regional distribution of

* A discussion of the antigenic type or types of virus involved is presented in a later section of the paper.

the disease are not sufficient to interfere with the significance of an analysis of age-specific attack rates based on the entire population of the island. Attack rates for the various major racial groups are also presented. It is clear that persons of Chinese, Hindu and African origin all experienced high attack rates. Differences appear to be related to location of employment and residence rather than to hereditary factors.

The following statement is made concerning the incidence on the island prior to the epidemic of 1945. 'Poliomyelitis is endemic. A few cases have been reported from time to time since 1927 when the disease was made notifiable, and a considerable number of old cases were seen clinically, one dating from 1891.'

(c) *Calculation of infection rates prior to 1945*

In their paper McFarlan *et al.* (1946) give the number of cases and the attack rates for age groups of 1 year to 9 years inclusive and for quinquennia to the age of 29 years inclusive. It is evident from Table 3 and Fig. 2 that children 1 year old had the highest attack rate. Those 2-5 years old experienced attack rates that were essentially equal and were approximately 20% lower than that in 1-year-olds. χ^2 -analysis shows that chance variations could easily account for the differences in attack rates among the five youngest age groups ($\chi^2 = 3.88$, $P = 0.43$). Attack rates among those over 5 years of age were progressively lower with increasing age.

Estimates of the prevalence of immunizing infections in the years prior to the epidemic, as reflected by the age-specific attack rates, indicate that in 1940, 1941 and 1942 the virus was absent or at least so restricted in its distribution as to leave no detectable increment to the immune population among infants and very young children. In 1943 there may have been an appreciable but low incidence of immunizing infections in view of the fact that there was a higher incidence of poliomyelitis in 1945 among 1-year-olds than among children 2, 3 or 4 years old. However, as demonstrated by the calculated 95% confidence limits, this difference may well be due to random distribution of cases in the epidemic. Our chief concern is with the incidence of disease in children who were 5-9-year-olds during the epidemic.

The attack rate in 6-year-olds was 28% lower than that in 5-year-olds. The attack rates in 7-, 8- 9-year-olds were 60, 50 and 53%, respectively, lower than the attack rates in the next younger age groups. The total decrease from a rate of 1190 in 5-year-olds to 80 in 9-year-olds represents a difference of 93%. This difference could be accounted for on the basis of an annual infection rate of approximately 45% during the 4 years represented by the difference in age of the 5-year-olds and the 9-year-olds. An annual infection rate of 45% would involve 91% of a population in 4 years. It is, therefore, postulated that children of Mauritius who were 9 years old in 1945 had experienced an annual prevalence of poliomyelitis virus* during four successive years prior to 1940, and that on the average approximately 45% of those susceptible at the beginning of the year had been infected and immunized by the end of the year. Children in the next three younger age groups had undergone a similar experience for fewer years.

* A discussion of the antigenic type or types of virus involved is presented in a later section of the paper.

DISCUSSION

The concept that the incidence of poliomyelitis among persons of different ages is determined largely by immunizing infections in previous years is relatively old. Frost (1913) pointed out evidence supporting the concept that the small number of cases of poliomyelitis seen in adults was the result of immunity 'acquired from previous unrecognized infection with the virus of poliomyelitis'. He referred to previous discussions along similar lines by Wernstedt. In spite of general acceptance of the importance of previous unrecognized infection in determining age-specific attack rates in poliomyelitis, we have been unable to find published estimates of annual infection rates made from this sort of epidemiological data. It seems probable that a reason for the absence of such published figures is that available information has seldom been adequate for this purpose. Records concerning a large number of epidemics have been examined with this in mind; the fourteen listed in Table 1 were regarded as among the most severe. The data from only the two epidemics selected for the present analyses proved to be adequate to give firm support to the interpretation that immunizing infections were prevalent during each of 4 or more successive years prior to the epidemic. Analysis of the data from the other epidemics revealed an inadequacy of the numbers of cases or of the total population involved or other factors which introduced an undesirable degree of unreliability to calculations of this sort.

(a) *Other possible explanations for the age-specific attack rates in these two epidemics*

There are probably few who would doubt that previous immunizing infection was the chief cause of the low incidence of illness among children 10 years old and older during the New York epidemic of 1916 and the Mauritius epidemic of 1945. One need only consider the high incidence of paralytic disease in children of these ages during recent years in many parts of the world to rule out physiological factors associated with increasing age as a major protective influence. It is equally improbable to suggest that children 10 years old and older were shielded from virus through lack of exposure, while their younger siblings and neighbours were suffering the effects of devastating epidemics.

Likewise there is no reason why reporting of paralytic disease should be so markedly less with progressive increase of age by 1 year increments as to account for the observed age distribution of reported cases.

(b) *Antigenic type or types of virus causing immunizing infections*

In view of the size of the populations involved and the numbers of cases, it appears reasonable to infer not merely that attack rates in older age groups were low as a result of previous infection, but to conclude in addition that such immunizing infections occurred each year for at least 6 years (probably 1908-13) in New York and for at least 4 years (probably 1936-39) on Mauritius. The previous immunizing infections presumably were caused by virus of the same type or types as caused the subsequent epidemics. The possibility that heterotypic immunity

may have been a factor cannot be disregarded completely (Salk, 1955). More data are needed to evaluate this possibility.

It is evident that the postulated infection rates must be regarded as minimum figures in view of the possibility that multiple types of virus were involved in one or both epidemics, or that heterotypic immunity may have been operative to some degree. If, for example, Types 1 and 3 were responsible for equal numbers of cases in Mauritius, then the immunizing infections in previous years for each virus must have been approximately the number indicated, and the total number of infections was double the number calculated. Experience to date would, however, favour the idea that epidemics were caused principally by virus of a single type.

(c) *The similar attack rates in several of the youngest age groups*

For our present purposes, it is unnecessary to explain why the attack rate in New York was essentially similar in 1- and 2-year-olds and showed no significant difference among children from 1 to 5 years of age on Mauritius. However, it is of some interest to consider possible implications of these data.

The fact that in New York the attack rate was essentially the same in 1-year-olds and 2-year-olds may have resulted from any of several causes. Among those that appear plausible are:

(1) Decreased prevalence of virus and of immunizing infections in New York in 1915 as compared with preceding years.

(2) Lesser exposure of children under age 1 during the epidemic.

(3) Greater difficulty in diagnosing the illness in children under age 2.

The essentially similar attack rates in the Mauritius epidemic in the age groups from 2 to 5 years inclusive could reasonably be attributed to failure of homotypic virus to be prevalent during a 3 year period, probably 1940–42, before the epidemic. Most of the children who were 5 years old at the time of the epidemic in March 1945 were born in 1939. If immunizing infections had been prevalent in 1940, many in this age group would have been protected and the attack rate in 1945 would have been lower among 5-year-olds than among those 4, 3, or 2 years old. Such was not the case. While one cannot regard the absence of homotypic virus in the years 1940–42 as an assured explanation for the essentially similar attack rates in the age groups 2–5 years, it is the explanation that appears most plausible. In at least one other instance there is evidence of absence of one of the poliomyelitis viruses despite high population density and a very low level of sanitation. The age distribution of the 566 paralytic cases in the epidemic of 1947 on Car Nicobar, an island in the Bay of Bengal (Moses, 1948), indicated that the homotypic virus had not been prevalent in the population of 8700 for more than 20 years.

(d) *Estimates of total immune and susceptible populations at the beginning and end of each poliomyelitis season*

If one accepts the concept that poliomyelitis virus of a type involved in the epidemic infected and immunized approximately 30% of susceptible children in New York during 6 or more years, one can readily calculate the immune component of the population at the beginning and end of each poliomyelitis season.

To avoid the unnecessary complication of estimating the extent of infection among those under 2 years of age during years prior to 1916, we will designate successive age groups and their susceptibility just prior to a 'poliomyelitis season' as follows:

Youngest age group in which virus will be fully prevalent during the impending poliomyelitis season	100 % susceptible
1 year older	70 % susceptible
2 years older	49 % susceptible
3 years older	34 % susceptible
4 years older	24 % susceptible
5 years older	16 % susceptible
6 years older	11 % susceptible
Total of the above 7 age groups	30 % susceptible

From the above calculation it may be inferred that in New York, over a period of several years, spread of poliomyelitis virus began each year in a population in which approximately 30 % of children in these younger age groups were susceptible. During the annual period of prevalence it infected and immunized about one-third of susceptibles. Therefore, its spread terminated with 20 % of these children still susceptible.

A similar analysis for Mauritius during the years when there was an average annual infection rate of 45 % gives the following results:

Susceptibles at the beginning of a poliomyelitis season or year

Youngest age group	100 % susceptible
1 year older	55 % susceptible
2 years older	30 % susceptible
3 years older	17 % susceptible
4 years older	9 % susceptible
Total of the above 5 age groups	21 % susceptible

Therefore, spread of poliomyelitis on Mauritius appears to have started when approximately 21 % of those in the indicated age groups were susceptible and terminated its spread with approximately 12 % of the children still susceptible. It is possible that on Mauritius, spread of the virus was continuous throughout the year and occurred among a population with about 15 % of the younger age groups susceptible at any time.

The role of reinfection in the maintenance and spread of poliomyelitis virus in a population is not yet known. Probably the most significant information is that in the preliminary report of Fox, Gelfand, LeBlanc & Conwell (1956). These authors made periodic studies of 150 representative households in southern Louisiana for an average period of 27 months. Their findings indicate that when reinfection occurs 'it is not associated with prolonged epidemiologically significant shedding of virus'

They also stated 'our data suggest that the cyclic occurrence of Type 3 and Type 1 virus may be conditioned by specific deficiencies in immunity among children under four years of age'

SUMMARY AND CONCLUSIONS

Data concerning numerous severe epidemics of poliomyelitis were surveyed for information useful in estimating the size of the immune component of the population by age groups of 1 year. The New York epidemic of 1916 and the Mauritius epidemic of 1945 were chosen as the most suitable for this purpose. It is shown that there was a regularly progressive decline in attack rates for successively older age groups from 2 to 8 years in New York. The attack rate in 8-year-olds was less than 10% as large as that in 2-year-olds. It is noted that a difference of this magnitude could be accounted for by immunizing infections amounting to 30% per year for 6 years.

A similar analysis of age specific attack rates during the Mauritius epidemic shows progressive declines of 28, 60, 50 and 53% for the successive age groups 5-9. An average annual infection rate of 45% over a 4 year period could account for the ten-fold difference in infection rates between 5-year-olds and 9-year-olds.

An immunizing infection rate of 30% a year would lead to a pattern of immunity in which the seven youngest age groups had a total susceptible component of 30% at the beginning of a 'poliomyelitis season'. Twenty per cent would remain susceptible at the end of the season when spread of virus terminated.

An annual immunizing infection rate of 45% would bring about a situation in which 21% of the five youngest age groups were susceptible at the start of a period of viral prevalence and 12% at the end. Alternatively, one could consider that on Mauritius there may have been a continuous prevalence of virus in a population in which approximately 15% of the five youngest age groups were susceptible at any time.

It is suggested that the essentially similar attack rates among children 3-5 years old on Mauritius may have reflected a 3-year period during which homotypic virus was not prevalent, in contrast to its great prevalence during the years prior to that time.

A more detailed discussion and analysis of additional data concerning age-specific attack rates in poliomyelitis will be found in the thesis submitted by Dr Sample to the University of Washington School of Medicine entitled, 'Some observations on statistical and theoretical epidemiology of infectious diseases, principally poliomyelitis', in 1955. This is obtainable by Inter-library Loan from the Health Sciences Library, University of Washington, Seattle.

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REFERENCES

- ANDERSON, C. A. (1910). Report of an epidemic of two hundred and seventy-nine cases of acute poliomyelitis. *Pediatrics, Springfield*, **22**, 543.
- AYCOCK, W. L. (1928). The significance of the age distribution of poliomyelitis. Evidence of transmission through contact. *Amer. J. Hyg.* **8**, 35.
- EVANS, C. A., CHAMBERS, VELMA, C., GIEDT, W. R. & WILSON, A. N. (1957). The immune status of a population at the termination of a severe epidemic of poliomyelitis. *J. Hyg., Camb.*, **55**, 239.
- FOX, J. P., GELFAND, H. M., LEBLANC, D. R. & CONWELL, D. P. (1956). A continuing study of the acquisition of natural immunity to poliomyelitis in representative Louisiana households. *Amer. J. publ. Hlth*, **46**, 283.
- FROST, W. H. (1913). Epidemiologic studies of acute anterior poliomyelitis. *Hyg. Lab. Bull., Wash.*, no. 90. U.S. Govt. Printing Office, Washington, D.C.
- GRUNWELL, A. G. (1900). Report of an epidemic of acute anterior poliomyelitis of adults on the Island of Guam, Ladrone Islands. *Rep. Surg. Gen. U.S. Navy*, no. 224.
- HROLV, K. (1935). Observations from an epidemic of acute poliomyelitis in Sukkertoppen, Greenland. *Canad. publ. Hlth J.* **26**, 575.
- HØYGAARD, A. (1939). Acute epidemic diseases among Eskimos in Angmagssalik. *Lancet*, **1**, 245.
- LAVINDER, C. H., FREEMAN, A. W. & FROST, W. H. (1918). Epidemiologic studies of poliomyelitis in New York City and the northeastern United States during the year 1916. *Publ. Hlth Bull., Wash.*, no. 91. U.S. Govt. Printing Office, Washington, D.C.
- McFARLAN, A. M., DICK, G. W. A. & SEDDON, H. J. (1946). The epidemiology of the 1945 outbreak of poliomyelitis in Mauritius. *Quart. J. Med.* **15**, 183.
- MOSES, S. H. (1948). Report on the epidemic of anterior poliomyelitis in the Nicobar Islands. *Indian med. Gaz.* **83**, 355.
- MÜLLER, A. (1910). Eine epidemisch auftretende Erkrankung des Nervensystems auf Nauru [An epidemic of disease of the nervous system occurring in Nauru]. *Arch. Schiffs-u. Tropenhyg.* **14**, 535.
- NISSEN, K. I. (1947). Poliomyelitis on St Helena, 1945-6. *Proc. R. Soc. Med.* **40**, 923.
- PEART, A. F. W. (1949). An outbreak of poliomyelitis in Canadian Eskimos in wintertime; epidemiological features. *Canad. J. publ. Hlth*, **40**, 405.
- SALK, J. E. (1955). A concept of the mechanism of immunity for preventing poliomyelitis. *Ann. N.Y. Acad. Sci.* **61**, 1023.
- SEDDON, H. J., AGIUS, T., BERNSTEIN, H. G. G. & TURNBRIDGE, R. E. (1945). The poliomyelitis epidemic in Malta, 1942-3. *Quart. J. Med.* **14**, 1.
- SIGURJONSSON, J. (1950). Epidemiologic characteristics of poliomyelitis in Iceland. *Amer. J. Hyg.* **51**, 109.

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