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THE INFECTIOUSNESS OF MEASLES

BY MAJOR GREENWOOD

Measles, once a deadly, still a very common disease, is a worthy object of study for the biometrician. Although it is not now a very important cause of death or invalidity, it has been and may again become important. Then it is a virus disease, and virus diseases, for instance, influenza and poliomyelitis, are very important killing or maiming diseases; if we really knew precisely how measles spreads, that knowledge might help us to understand how these more serious illnesses are passed on. The statistical literature of measles is enormous, but, as I shall illustrate, it is easy to misinterpret unhomogeneous data, and really precise observations of the natural history of measles are rare.

Some opinions on the aetiology of measles are old and universally held by physicians. The first is that the length of time during which an infected person can pass the virus to another person is short and is over when the patient shows the characteristic signs of the disease—the typical rash, etc. The second is that the interval between the moment of infection, viz. reception of an effective dose of virus and the appearance of symptoms or signs of illness, is about 14 days; 'From 7 to 18 days; oftenest 14' is a common statement. The third is that droplet infection by coughing, spitting or contamination from sputum, etc., is responsible for the immense majority of infections. I see no reason to doubt that these statements are, broadly speaking, true; they are, however, vague. According to *N.E.D.*, incubation is 'The process or phase through which the germs of disease pass between contagion or inoculation and the development of first symptoms'. Symptoms, of course, are subjective, but probably the lexicographer includes physical signs—e.g. running from eyes or nose, rash, etc. This interval can only be *precisely* determined when the child has had but one exposure to another infected child.

Such precise information is not abundant in the enormous 'literature' of measles because it implies exact knowledge of contacts, which is only to be had in country districts where the medical practitioner is fully acquainted with the social habits of his patients. Excellent examples are to be found in Dr W. N. Pickles's book, *Epidemiology in Country Practice* (1939, see pp. 32–6). On the other hand, abundant data of intervals between successive cases in families are available. In the second column of Table 1 are the figures provided by Stocks & Karn (1928); these are obtained from the record cards of all cases of measles notified in the Metropolitan Borough of St Pancras from March 1924 to March 1927. The authors say explicitly that the interval is that between the first appearance of the rash in successive cases. In this frequency distribution all the data are included. A family with two infected children could only provide a single item, but a family with three would provide two and so on. A medical reader would hold that none of the first four or five frequencies included children really infected within the family, but that they, like the first child in family to go down, caught the disease elsewhere. Then, noting that from the sixth day onwards the frequencies increase to a maximum and decrease, he would say that, from the sixth day onwards, the proportion really infected within the family increased. Of course a biometrician wishes to do better than this; he would like to dissect the compound frequency into two components.

The easiest of all dissections is into two Poisson frequencies (see W. Schilling, 1947). I tried this on the St Pancras data but the result was execrable; the summed frequencies from interval 9 onwards were quite good fits, but from 1 to 8 hopeless—the computed frequencies were 310, 143, 56, 47, 80, 136, 204, 266. A better result was reached by pure empiricism. I used all the frequencies from interval 7 as they stood, guessed values—4, 10, 22, 43, 74, 116—for the first six and then fitted a Pearsonian type III to the product of my cookery. It was not too bad, but of no scientific value. My algebra is not equal to fitting a type III curve scientifically from its tail. But even when that problem has been solved, it does not seem

Table 1. *Intervals (days) between successive cases of measles in families (intervals of 0 or of more than 20 days included)*

Interval	St Pancras	%	Providence, R.I.	%	Providence, R.I. (between first and second in families of three)	%
1	341	9·88	253	6·07	61	8·54
2	246	7·13	184	4·42	43	6·02
3	160	4·64	108	2·59	21	2·94
4	117	3·39	110	2·64	25	3·50
5	96	2·78	111	2·66	20	2·80
6	90	2·61	114	2·74	20	2·80
7	99	2·87	189	4·54	40	5·60
8	173	5·01	247	5·92	41	5·74
9	206	5·97	388	9·31	75	10·50
10	318	9·22	519	12·46	110	15·41
11	353	10·23	511	12·27	72	10·08
12	329	9·54	451	10·83	66	9·24
13	269	7·80	344	8·26	53	7·42
14	205	5·94	244	5·86	39	5·46
15	153	4·43	142	3·41	14	1·96
16	104	3·01	105	2·52	8	1·12
17	70	2·03	58	1·39	2	0·28
18	48	1·39	37	0·89	0	0·00
19	38	1·10	25	0·60	2	0·28
20	35	1·01	26	0·62	2	0·28
Totals	3450		4166		714	

that we should have a unique frequency distribution of intervals. The third column of the table gives the findings of Wilson, Bennett, Allen & Worcester (1938) for Providence, Rhode Island, 1929–34, and the fifth column their counts of intervals between first and second cases in families in which three children were affected. Wilson *et al.* do not explicitly say how they measured interval—they may, perhaps, have taken the interval between first signs of illness instead of between dates of rash—but I do not think the point material. It is obvious that the total Providence frequency is not congruent with the St Pancras frequency, and that the Providence selection is not congruent with the total. I am speaking from the biometric standpoint, i.e. that no two of them could be regarded (using the χ^2 test) as drawn from a common population (naturally in comparing the Providence sets one has to subtract the frequencies of the selection from those of the total). But the unstatistical epidemiologist would say, with respect to the totals, that they are very similar; it is true that the St Pancras

mode is a day later than the Providence mode and much taller, but at least we can say that the most frequent interval is 10 or 11 days and the decline on the short side faster than on the long side. This is arithmetically vague, but good enough to justify us in thinking that in very few cases following a 'primary' within 5 or 6 days the patients were infected by the 'primary', and a great majority of those which were more than 8 days later were so infected. That information is enough to show how easily one may draw false conclusions from incomplete information, as I shall illustrate on a blunder of my own.

Eighteen years ago I interested myself in the question whether any light could be thrown on the mechanics of infection by studying arithmetically the frequency distribution of multiple cases of disease in families. There should be, for instance, a contrast between multiple cases of enteric fever, which is certainly not conveyed by coughing and spitting, and multiple cases of measles, which almost certainly is. Unfortunately, data of multiple cases of enteric in families are hard to come by; but those of measles are common. I knew Dr Stocks was working on St Pancras records of measles and asked him to let me have some data. He kindly supplied me with four sets in which there were respectively 2, 3, 4 and 5 children under 10, in addition to the child first infected, and three shorter series in which the children exposed were known not to have had measles before. I cite only series in which 60 families were available. It was quite obvious that these data could not be fitted by 'straight' binomials for which the exponents were the numbers in family (other than the first child to go sick), and p was given by the ratio of total cases to total of exposed children. Then the idea of a chain suggested itself. Suppose the first child distributes infection binomially; he may infect 0 child or he may infect all the n exposed, with chances q^n and p^n (p being unknown); in these instances no chain arises. But every other term of his binomial provides an opportunity; if he has infected $n-1$ (his chance of doing so being $np^{n-1}q$); this leaves one child unaffected, who is exposed to infection from one of these $(n-1)$ secondaries who distributes binomially with chance p', and so one could have a family in which all n were infected. Now if each new binomial distributor does so with a different p, we shall have arithmetical difficulties. Biologically it seemed very unlikely that the chaining p's *would* all be equal. Dr Stocks had in fact made it probable that exposure to infection which produces no clinical signs or symptoms of disease may confer some immunity. Hence the $(n-1)$th child in series is not so likely to infect the nth as the first to infect the second. However, it is sound empiricism to begin with the simplest hypothesis and to see if it works. Now if p is constant, the algebra and arithmetic are simple enough. Take $n = 2$. Then q^2 should give the proportion of families with no cases other than the primary, $2q^2(1-q)$ that of one case, and $1-3q^2+2q^3$ that of two cases. The mean will be equal to $2-4q^2+2q^3$, so we can solve for q. If n were 3 the highest power of q in the equation would be 6, if n were 4 it would be 10, and so on.* The arithmetic would become tedious as n increased, but in real life, in these days of small families, it would be rare to have adequate data beyond families of 3!

It is important to note that the assumption is made that the distribution of children infected by the first child, the primary, must be strictly binomial; as we shall soon see, this assumption may not be justified.

I applied this process to Dr Stocks's data, and in every instance the fit was satisfactory. For $n = 2$, in the less select and longer series (358 families) P† was 0·75; in the smaller but

* See Greenwood (1931).

† Here and below P relates to the χ^2 test for goodness of fit, standing for the chance of a result which is as least as divergent as that observed.

more select (299 families), $P = 0{\cdot}58$. For $n = 3$, P was 0·36 for the longer and 0·42 for the shorter series; for $n = 4$, 0·61 and for $n = 5$ (only 61 families) 0·42. I must confess that the concordance pleased me and, no doubt, lulled a scepticism which, I think, is my usual habit of mind. *Really* to verify the theory, one should break up the groups into their constituents, thus in the simplest case, that of $n = 2$, the proportion due to the primary is $(1-q)^2$ and the proportion derived by chaining $2(1-q)^2q$. Well, *was* it? I do not know; it is possible that Dr Stocks could not have supplied the information and certain, in view of what I have already said of the difficulty of dissecting the frequency distributions of intervals, that the information could not be absolutely precise. But that is no excuse at all for not even asking!

In the excellent memoir of Wilson and his colleagues already quoted, it is conclusively demonstrated that, to their data, this simple hypothesis does not apply. Like me, they found usually excellent fits to the gross data; for 416 families with $n = 2$, the value of P was 0·94, for another set of 185 families, 0·68, and for 151 families with three susceptibles $P = 0{\cdot}48$.

But, when the clubbed frequencies were dissected, the chain theory was demolished; in every instance the proportion infected by the primary was far larger and the proportion derived from chains far smaller than it should have been. One example (Wilson *et al.* 1938, p. 449) will suffice.

The gross data consisted of 151 families with three susceptibles (other than the primary) giving 10 (9·4) 0's, 7 (6·5) 1's, 39 (33·1) 2's and 95 (101·0) 3's. The figures in brackets are the expected numbers using the chain method with $q = 0{\cdot}392$, and show an excellent fit, $P = 0{\cdot}48$. Now let us break up the 3's into their chain constituents. We have

$$6Np^3q^3 = 12{\cdot}7\ (4), \quad 3Np^3q^2 = 15{\cdot}6\ (3), \quad 3Np^3q = 39{\cdot}8\ (13), \quad Np^3 = 33{\cdot}9\ (75).$$

Perhaps it may be said that the allocation to the several groups depends on personal opinion; but in this instance—and, I have no doubt, in the others—different classifiers would not reach significantly different results. Dr Jane Worcester kindly sent me a copy of the working sheet allocating the 95 sets of three cases. Of the 75 attributed to the Np^3 group, the greatest interval between the first and third in series was 5 days (two instances) and there were eight instances of an interval of 4 days. On *any* reasonable hypothesis of short incubation most of these must be attributed to the primary, and even if all were classed as intrafamiliar, the fit to the chain hypothesis would not be materially improved. There is a still more conclusive argument. The simple chain hypothesis assumes that the distribution of cases due to the primary is a binomial distribution; in the Providence experience it is not.

This subject has been taken up in a recent paper by Prof. E. B. Wilson (1947). In a set of 519 families with two susceptibles other than the primary, there were 49 0's, 102 1's and 368 2's. Take the ratio of total cases to population, 0·807, as p and $n = 2$. The binomial distribution is 19, 162 and 338. In this particular set, the fit of the gross totals to the chain hypothesis was poor, $P = 0{\cdot}02$, but the binomial fails just as conspicuously when applied to the primary distribution for 416 families which gave $P = 0{\cdot}94$ for the chaining method when used on massed frequencies. Here the gross totals were 51, 67 and 298. Of the 298 sets of two cases, 36 were found to be due not to the primary, so that the distribution due to the primary is: 0, 51; 1, 103; 2, 262. The binomial distribution for $p = 0{\cdot}7536$ is 25·2, 154·5 and 236·3, which is quite hopeless. This at once suggested, what I should have seen before posing the hypothesis, that if the families are heterogeneous in respect of risk, the summed frequencies obtained by adding 0's, 1's, 2's, etc., and deducing a p from the massed data *could* not be a straight binomial. This was first pointed out, I think by Karl Pearson (1917). It is easy to

see that, if we add in this way, the variance obtained will not be npq, where p and q are the weighted means of the several values for the summed binomials but will exceed npq by

$$n(n-1)\left\{\frac{Sm_s p_s^2}{N} - \left(\frac{Sm_s p_s}{N}\right)^2\right\}, \tag{1}$$

where n is the exponent and m_s and p_s the number of observations and value of p for the sth binomial; $N = Sm_s$. Clearly this only vanishes if the variance of p vanishes.

Is this, however, anything more than a debating point? I think it is, for this reason. It is hard to doubt that measles is conveyed by droplet infection and, if it is, the contiguity of the susceptibles to the source of infection must be a factor of the attack rate. So far as I know, there are no published data giving *attack* rates on groups of n susceptibles, primary attack rates, tabulated by numbers of rooms occupied. That *mortality* rates are negatively correlated with social status is, of course, a common-place of epidemiological literature and the usual explanation is that in poor families children are exposed to infection at an earlier age than in the families of the well-to-do; the fatality rate of measles diminishes steeply with advancing age. No doubt it is implicit in this argument that infection is increased by overcrowding but without data we certainly cannot say that in economically homogeneous data, the distribution of primary infections is binomial. Without fresh data, we can do no more than test whether the, by hypothesis, heterogeneous aggregate *can* be subdivided into constituent binomials. At first this seems trivial; if there are s subgroups and s values of p and the only conditions imposed are that the mean value of p and the variance of p must be reproduced and, of course, that all the p's must be positive and less than unity, the number of solutions must be great. However, there are two other 'common sense' restrictions. The subfrequencies must be reasonable; the frequency distribution of persons per room must be unimodal and the p's must decrease as the number of persons per room decreases. Take Wilson's set of 519 families of two susceptibles. The variance of the aggregate (49 0's, 102 1's, 368 2's) is 0·42569 which exceeds the binomial variance—$2 \times 0{\cdot}807 \times 0{\cdot}193$ by 0·11419 so if this aggregate is a sum of binomial frequencies the variance of p is 0·05709. I split up the total of 519 families into six subfrequencies, 47, 189, 147, 81, 25, 30. This is obtained from the proportional frequencies of families of three living in 1, 2, 3, 4, 5 and 6 or more rooms in the Metropolitan Borough of St Pancras in 1931. I then adjusted the p's on this principle: That the first should be unity, i.e. that in the very crowded tenements both children should be infected and that in the most spacious tenements neither child should be infected and that the p's should decrease from 1 to 0. Actually if the intervening p's are 0·9, 0·87, 0·795 and 0·389, the variance of p is 0·057 and the aggregate subtotals closely reproduced (47, 106, 366). Of course, many other solutions are practicable; the *only* value of the trial is that there is nothing plainly preposterous in the run of the p's.

The obvious criticism from the practical point of view is that to reach homogeneity requires an enormous mass of data. Suppose what I am suggesting were done (in the records of public health departments in this country and in the United States an immense amount of information lies unpublished) there would *still* remain possibly relevant heterogeneity. Still, the problem *is* an interesting one; that the primary distribution should be binomial, or approximately binomial, is a seductive hypothesis.

But it may fairly be objected that we have the massed data and it is interesting to try to interpret them. Wilson has made two suggestions. We infer from the data that measles does *not* spread within the family as if the children were independently infected and there are at

least two ways of characterising the dependence. The first may be described in his own words (the illustration is the set of 519 just discussed).

> One way to express dependence of elements is to compute the number which would be required by the theory of chance to explain the observed standard deviation. The actual secondary attack rates in the families with 0, 1, 2 secondary cases are 0, 0·5, 1·0 respectively; their mean is 0·807 and their standard deviation squared (variance) is 0·106. If this be equated to pq/n we find $n = 1{\cdot}46$. Thus the two susceptibles in the family are behaving relative to the contracting or escaping the infection as though they were about one and one-half.

I have substituted 0·106 for 0·016 which is an evident misprint. Here p is the mean value of p. The plan is to substitute for the observed table a binomial the exponent of which is deduced from the variance of p. I have a certain awe of binomials the exponents of which are not integers, perhaps owing to a recollection of v. Bortkiewicz's furious polemic entitled *Realismus und Formalismus in der mathematischen Statistik* (1918) directed against a paper by L. Whitaker (1914). The last-named author had found better fits to some data used by v. Bortkiewicz to illustrate the Poisson series by using fractional or negative exponents to binomials.

Taking Wilson's example, we have:

$$(0{\cdot}8073 + 0{\cdot}1927)^{1{\cdot}46227} = 0{\cdot}73124\ (1 + 0{\cdot}2387)^{1{\cdot}46227},$$

and the successive terms are

$$0{\cdot}73124,\ 0{\cdot}25523,\ 0{\cdot}01408,\ -0{\cdot}000603,\ 000056,\ \text{etc.}$$

Now if we use these as frequencies for variates 1·462, 0·462, −0·538, −1·538, −2·538, etc., and compute the mean and variance, we reproduce the proper mean and variance but do not reproduce the frequencies. Ignoring terms after the third—the sum of the first three terms is 1·00055—we find for expected frequencies, 379·5, 132·5 and 7·3, not much better than the integral binomial.

Wilson gives a set of 100 families with three susceptibles, as follows:

No. attacked	3	2	1	0
Frequency	67	18	11	4

This time the mean p is 0·826667 and its variance 0·07884, so that $n = 1{\cdot}81745$, giving for the binomial $0{\cdot}70754\ (1 + 0{\cdot}20967)^{1{\cdot}81745}$. The sum of the first five terms is 1·00016; neglecting the rest, the mean and variance are correctly obtained to 3 places for the mean and 2 for variance, but the frequencies are poorly reproduced, namely 70·7 for 67, 26·9 for 18, 2·3 for 11 and a small negative value for 4.

A better method, as Wilson suggests, is to use the method of association. When there are two susceptibles, call them A and B and form the table

	+	−
+	(AB)	(αB)
−	$(A\beta)$	$(\alpha\beta)$

In this (AB) means the frequency of both susceptibles going down, (αB) the frequency of A not falling sick but B doing so, etc. In such a table as that containing the set of 519 already discussed one cannot distinguish A from B (which, as Wilson notes, might be done if one used, for instance, age of each member of a pair as a distinction) so A must be put equal to B. One can then proceed on Yule's lines for associated attributes. In the case before us

the secondary attack rate is 0·807, but if one of the pair is attacked, the chance the other will be attacked is 0·88, while if one of the pair escapes the chance the other will escape is 0·49.

It occurred to me that another way of bringing out Wilson's point would be to use the theorem that if the events are not independent, $\sigma^2 = npq(1+r(n-1))$, where r is the arithmetic mean of the $\frac{1}{2}n(n-1)$ correlations of the variables, viz. the product-moment correlations of variables restricted to the values 0 or 1.

Suppose we have to do with n correlated 'events' of this type, we are concerned with a succession of 0's and 1's and for each of the n items the mean is p and the variance pq. Hence, if we estimate the expectation of the tth event from the results of the preceding $t-1$ events, each regression coefficient will be a coefficient of partial correlation (for all variances are equal) and we have $x_t - p = \rho_{1t.23\ldots}(x_1-p) + \rho_{2t.13\ldots}(x_2-p) + \ldots$.

Now if we put in this equation the values of x_1, x_2, etc. (the values must all be 1's or 0's), we reach, let us say, the value k. This will be its expectation. But, as x_t must be either 0 or 1, this amounts to saying that the probability that x_t will be 1 is k and the probability it will be 0 is $1-k$. The equation is, however, useless unless we know the values of the partial correlations. Let us suppose they are all equal. In the particular case of $n = 3$—the only one I shall discuss—they would be each $r/(1+r)$. The first event 0 or 1 has expectation p or q. The regression equation of the next event x_2 on x_1 is $x_2 = rx_1 + p(1-r)$ giving for $x_1 = 1$, $r+p(1-r)$, for $x_1 = 0$, $p(1-r)$, so the probability of (11) is p^2+pqr, of (10) $pq(1-r)$, of (00) q^2+rpq and of (01) $pq(1-r)$.

If x_1 and x_2 are given, the regression of x_3 on them is

$$x_3 = x_1 r/(1+r) + x_2 r/(r+1) + p\{1-2r/(r+1)\},$$

and the 8 probabilities can be calculated. For instance, the probability of the succession (010) will be

$$pq(1-r)\{1-r/(1+r)-p(1-2r/(1+r)\}$$

$$= \frac{pq}{1+r}\{(1-r)(q+pr)\} = \frac{q}{1+r}\{(p-pr)(q+pr)\}.$$

The expectations of (010) (001) and (100) are, of course, the same, so the probability of 1 'success' is

$$\frac{3pq}{1+r}\{(1-r)(q+pr)\}.$$

In this way, we reach for the complete 'successes' distribution

$$0 \quad \frac{q}{1+r}(q+rp)(q+rp+r),$$

$$1 \quad \frac{3pq}{1+r}(q+rp)(1-r),$$

$$2 \quad \frac{3pq}{1+r}(p+rq)(1-r),$$

$$3 \quad \frac{p(p+qr)}{1+r}(p+qr+r).$$

Leading to an area of unity, a mean of $3p$ and a variance of $3pq(1+2r)$. One can proceed in this way step by step to deduce the 16 frequencies for the case $n = 4$ and so on.

Dr J. O. Irwin has, however, obtained an elegant general solution which he will, I hope, publish, for it may be of value in other investigations. In these days of small families, one will not often have sets of more than 3 susceptible children apart from the first infected in

measles and I confine myself to trying out the method on Wilson's 100 sets of 3 susceptibles beyond the first. Here $p = 0{\cdot}82667$, and $r = 0{\cdot}32538$ giving:

Cases	Observed	Expected
0	4	4·44
1	11	9·68
2	18	19·32
3	67	66·56

$\chi^2 = 0{\cdot}7091$ and P (for one degree of freedom) $= 0{\cdot}40$.

One has a temptation to speculate on the results of *not* assuming that the correlations are equal, but it would be pure speculation and, although the method interests me, I am not sure that it is better than the straightforward calculations Wilson proposes, although it does permit of deducing a χ^2 which, for old acquaintance sake, is pleasing to me. As I have indicated earlier in this paper, one needs more precise and homogeneous data than the aggregated results of a city survey furnish. The comparison of two aggregates is subject to another difficulty to which Wilson and his associates draw special attention (Wilson *et al.* 1938, pp. 443–4). Neither the St Pancras data of Stocks upon which I worked, nor those of Providence, R.I., are complete records of the occurrence of measles in the areas. Stocks estimated that as many as 70 % of the cases occurring were reported; Wilson and his colleagues put the proportion in Providence, R.I., no higher than 50 %. The secondary attack rates in Providence were a good deal higher than in St Pancras. *Prima facie* measles was much less infectious under the conditions of family life in St Pancras than under those of Providence, R.I., Wilson *et al.* write:

> One need not overlook the possibility that with only 47 per cent reported in Providence, it may be that those families with a large number of secondary cases in proportion to their total susceptibles are disproportionately frequent in the reported as compared with the unreported families. The discrepancy, however, between the secondary attack rate in St Pancras and Providence is so great that the rates cannot well be reconciled on the hypothesis that there is a differential in favour of higher attack rates in the reported families in Providence unless one assumes a differential in favour of lower attack rates in the reported as compared with the unreported families in St Pancras. This leaves a quite enigmatic situation and makes any comparisons problematical (op. cit. p. 444).

I have nothing to add to this clear statement. One might surmise that in a family in which many children come down together or within a short time, *i.e.* are infected from an extra familiar source or from the first in series, domestic help may be more urgently needed than when the disease is passed on by chaining. But I can see no reason why such a bias, *if* it exists, should be more effective in St Pancras than in Providence, R.I.

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