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The Critical Community Size for Measles in the United States†

By M. S. BARTLETT
Manchester University

SUMMARY

The critical community size for measles (the size for which measles is as likely as not to fade out after a major epidemic until reintroduced from outside, corresponding to a mean time to fade-out of about two years) is found for the United States to be about 250,000 to 300,000 in terms of total population, or about 30 in terms of average weekly notifications. These figures agree broadly with English statistics, provided notifications are corrected as far as possible for unreported cases. Comparison is also made with results calculated from theoretical models. The contrasting epidemiological patterns for measles and chickenpox are noted.

1. INTRODUCTION

A STOCHASTIC (probabilistic) reformulation (see Bartlett, 1955, 1956, 1957, 1959) of the theoretical model for recurrent measles epidemics originally put forward by Hamer and Soper (see Soper, 1929) emphasized two important features of the stochastic model. One was that in large communities the theoretical tendency of the successive epidemics to damp down could be offset by random variability, and thus give some possibility of representing actual statistics of measles incidence (cf. Wilson & Worcester, 1945). The second was the tendency in small communities for the infection to die out when the number of susceptibles had dropped below its threshold value. This "fade-out" tendency was discussed in some detail in relation to observational material in my 1957 paper; and it was noted that among the towns in England and Wales included in my sample, Bristol and Hull were of critical size in the sense that they were as liable as not to show fade-out after any measles epidemic.

During a visit to the United States in the autumn of 1958, I was able to refer to official American figures for measles notifications which Jane Worcester had previously collected for another purpose, and which she kindly made available to me. It was of some interest to see how far these American statistics supported my previous conclusions based on the statistics for England and Wales.

2. THE AMERICAN DATA

The data available were for thirty cities in N. America (including some in Canada), for which the measles notifications were recorded by calendar months for the period 1921 to 1940. Six of these I deleted because of incomplete records, leaving twenty-four. These ranged downwards in size from the largest (New York), and all had populations (for 1940) over 200,000, so that they do not contain a sample of towns of smaller size than this. However, reference to Fig. 1 (or Table 1) shows that fade-out of infection was clearly demonstrated at the lower end of the spectrum of population size, and this was amply supported by some separate data giving statistics for

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towns in New York State, including Buffalo, Rochester and Syracuse (which were also in the first series) and other smaller towns.

One minor difficulty was that the notifications were recorded by calendar month, whereas the statistics for England and Wales were weekly. In the latter case the criterion used was a gap of three weeks, whereas for the American figures the monthly interval had to be used (which implies a minimum interval of one month, and possibly a gap in notifications of up to seven or eight weeks). Fortunately, the steep rise in number of "fade-outs" over the entire twenty-year period when the average number of weekly notifications drops low suggests that the critical value is reasonably well defined in spite of this difficulty. There is also the problem of incomplete notifications, but, as was argued in my previous (1957) paper, this is unlikely to affect greatly the determination of the critical size for similar reasons. This is, of course, in connection

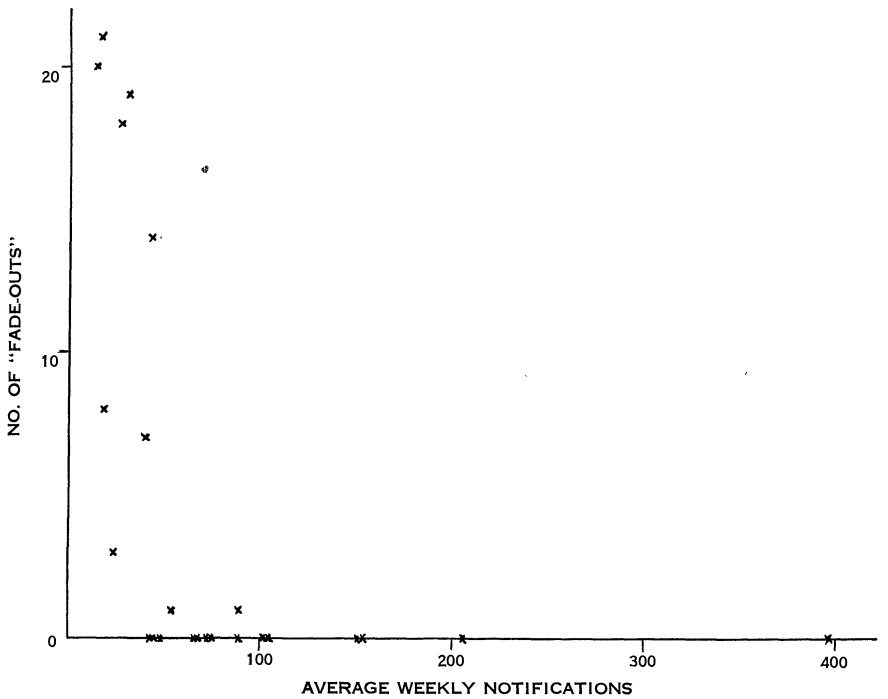

FIG. 1. Observed relation between average weekly measles notifications for N. American cities (1921–40) and the number of "fade-outs".

with the identification of towns of the right order of size; the question of the correct number of notifications for such towns is another matter which is discussed later. Deferring this question for the moment, we see from Table 1 (and Fig. 1) that the four cities which are most critical are Akron, Providence, Rochester and Winnipeg (Canada), with an average of weekly (reported) notifications of 31. This figure is rather lower than the average for the English towns (Bristol 67, Hull 57), and than the approximate theoretical figure arrived at in terms of a simplified epidemic model. This latter figure (see Bartlett, 1959) is, in terms of weekly notifications, of the order of 100. The probable reasons for the various differences are discussed in the next

sections; and in particular it is shown that the true American and English figures appear rather closer than the crude estimates quoted above.

TABLE 1

Measles notifications for North American cities, 1921–40

City	*Population* (1940)	*Weekly average*	*Number of "fade-outs"*
Akron	245,000	18	8
Baltimore	859,000	105	0
Birmingham	268,000	14	20
Boston	771,000	89	0
Buffalo	576,000	43	0
Chicago	3,400,000	206	0
Cleveland	878,000	89	1
Dallas	295,000	27	18
Detroit	1,623,000	154	0
Los Angeles	1,500,000	67	0
Milwaukee	587,000	102	0
Minneapolis	492,000	54	1
Montreal	1,022,000 (1954)	68	0
New York	7,455,000	397	0
Philadelphia	1,931,000	151	0
Pittsburgh	672,000	73	0
Providence	254,000	43	14
Rochester	325,000	24	3
San Francisco	635,000	49	0
Syracuse	206,000	31	19
Toronto	676,000 (1954)	75	0
Vancouver	344,833 (1954)	17	21
Washington, D.C.	663,000	45	0
Winnipeg	235,710 (1954)	40	7

3. THE "NOTIFICATIONS CORRECTION FACTOR"

The major correction to make to obtain a true notifications average is the ratio of actual cases to reported cases, which I shall call the "notifications correction factor". In the case of United States statistics, considerable attention was given to this point by Hedrich (1933), in his study of measles statistics for Baltimore from 1900 to 1931. It is possible to make an independent estimate of the average weekly notifications from the recorded births and deaths by age, making use of the known age-distribution for measles incidence, provided (and this is the main unknown datum) a definite percentage of children is assumed to have contracted measles by, say, their fifteenth birthday. Hedrich takes this figure to be 95 per cent., though it should be noted that there seems to be no complete unanimity over this estimate; the correction factor for Baltimore then decreases from 8 or 9 in 1900 to about 3 in 1920, remaining stable until 1930 (the end of the period investigated by Hedrich). An independent estimate of this factor for Newton (Mass.) by Chope (1940) was based on an actual survey of 6,325 school-children, among whom it was found that 3,245 were said by their parents to have had measles while in the district, compared with 1,670 reported cases. This gives a notifications correction factor of about 2, rather lower than Hedrich's earlier estimate for Baltimore. It is possible that some cases in Newton were still missed in spite of the questionnaire to parents, but as the

correction factor by Hedrich's method also gave a value of about 2, the difference is more likely to represent a real difference in reporting between the two areas. Such variability is also suggested by the variability in reported weekly notifications for the three selected United States cities, when compared with their total populations (Table 2).

A rough estimate of the correction factors for these towns (using local birth rates but a common United States set of infant mortality statistics, as the adjustment for deaths is comparatively slight) gave the values: Akron 3·9, Providence 2·2, Rochester 3·7. The corrected notification figures would correspondingly be 70, 95, and 81, with a mean of 82.

TABLE 2

Measles notifications for three North American cities, 1921–40

City	*Reported weekly notifications* (1921–40)	*Population* (1940)
Akron	18	245,000
Providence . . .	43	254,000
Rochester	24	325,000

A similar calculation for the English towns Bristol and Hull gave correction factors 1·8 and 1·6 and corrected notification figures of 121 and 91 respectively, with a mean of 106. These corrected means for England and the United States are rather nearer together than the original uncorrected means. It will be noticed that the English notifications correction factor appears rather lower on the average than that for the United States (it is, of course, for a more recent period). The mean factor for the two English towns is about 1·7, and this is reasonably consistent with other English estimates.† Thus Benjamin and Gore (1952) gave a figure of about 64 per cent. for official notifications before the age of 15 for London for the post-war years 1946–51; if we assumed a true value of 95 per cent., this would imply a factor of 1·5. An earlier percentage of notifications by the age of 15 for the whole of England and Wales calculated by Stocks (1949) for the period 1944–47 was 47 per cent. This would give a factor of 2 if compared with the assumed true value of 95 per cent., but Stocks, suggesting a true figure of 70 per cent. based on the number of school-children entering boarding-schools in 1930–34 who were reported to have had measles (Medical Research Council, 1938), arrived at the same numerical factor 1·5. However, it would seem at least advisable to correct the 70 per cent. for the children who later contracted measles at their boarding-schools. The calculation proposed is as follows: let P be the proportion who have had measles on entering (assumed immune to further attack), and let x be the annual entry as a proportion of the schools total N (assumed stationary). Then if the total number of cases at the schools is c, the number of immunes leaving school in r years is $rPxN+c$, and the final proportion of immunes is $P' = (rPxN+c)/(rxN) = P+c/(rxN)$. In the present instance r was 5 and x on average 0·235. c for all the boys' schools together was 1,801 compared with a total population of 10,270, giving an increase of P' over P of 0·15 (for boys and girls together

† I am indebted to J. Hajnal for references to these, and for some further helpful comments on the notifications question, including the point that recent developments in serological tests for measles antibodies may make possible direct assessment of the percentage of immunes in a population; see Black (1959).

it is about 0·14). This would lead to a final percentage nearer 85 per cent., and if this figure (which presumably can still be too low because of unreported cases before school entry) were used in place of the 70 per cent., Stocks's factor would change from 1·5 to 1·8.

4. Comparison with Theoretical Results

The corrected estimate of critical size for measles fade-out has become quite close to the approximate theoretical value already mentioned, about 100 in terms of average weekly notifications. This agreement is, however, almost too good if we recall the limitations in this latter figure. These limitations arise because the theoretical model for simplicity ignores certain complications in the actual epidemiological characteristics of measles, some of the more obvious of these being, to quote from my 1959 paper, "(i) the effect of a fairly well-defined incubation period and a limit to the persistence of infectivity from any one individual, (ii) the effect of spatial spread and diffusion of infection over an area, (iii) the effect of seasonal variation in incidence (probably largely due to summer dispersal and autumn re-assembly of school-children)".

To examine the effect of (i), we may consider a discrete-time or chain model of a type similar to ones considered previously for closed populations, by Reed and Frost in the United States and Greenwood in England (see Bailey, 1957). The extension to include the continual influx in susceptibles (the modification which permits the possibility of recurrent epidemics) is simple in principle; the consequent model is specified by

$$\begin{cases} S_{r+1} = S_r + m - I_{r+1}, \\ I_{r+1} = S_r P(I_r) + Z_r, \end{cases}$$

where I_r, S_r are the numbers of infective and susceptible persons at the rth time instant, and I_{r+1} is a binomial variable with probability $P(I_r)$, and number of "trials" S_r, where $P(i) \equiv 1-(1-p)^i$. Thus Z_r has zero mean and variance $S_r P(I_r)[1-P(I_r)]$. The influx m is assumed constant (not random), in contrast with the assumptions made in the "continuous-time" model (for details of which, see my previous papers); but it may be shown that the alternative specification of random influx does not affect the amplitude of the oscillations much if mp is small. Note that if we allow part, or all, of m to be available for infection with S_r by time $r+1$, we merely have to re-define S_r to include this increment and the equations are unaltered.

The above model is unfortunately even more difficult to handle theoretically than the continuous-time model, and even in the investigation of the approximate amplitude of "small" oscillations some care is necessary. Let us write

$$I_r = m(1+x_r), \qquad S_r = n(1+y_r),$$

so that the second equation becomes

$$m(1+x_{r+1}) = n(1+y_r)[1-(1-p)^m \, e^{mx_r \log(1-p)}] + Z.$$

It follows that

$$m \sim n[1-(1-p)^m],$$

which determines n in terms of m and p. If we write C for $-n(1-p)^m \log(1-p)$, the linearized form of the equations is

$$\begin{cases} y_{r+1} + mx_{r+1}/n = y_r, \\ x_{r+1} \sim y_r + Cx_r + Z/m, \end{cases}$$

whence by squaring or cross-multiplying, and averaging, we find on the assumption of stationarity (or quasi-stationarity; see Bartlett, 1959)

$$\begin{cases} 2\operatorname{cov}(x,y)+m\sigma/n_x^2=0 \\ \operatorname{cov}(x,y)+m\sigma_x^2/n\sim\sigma_y^2+C\operatorname{cov}(x,y) \\ \sigma_x^2\sim\sigma_y^2+2C\operatorname{cov}(x,y)+C^2\sigma_x^2+(1-p)^m/m. \end{cases}$$

The first of these equations is still correct for larger fluctuations and implies $\operatorname{cov}(I,S)/{\sigma_I}^2=-\frac{1}{2}$. We obtain also

$$\begin{cases} \sigma_x^2\sim(1-p)^m/\{m(1-C^2-m[1-C]/n)\} \\ \qquad\sim n/m^2, \\ \sigma_y^2\sim 1/m \end{cases}$$

for small m/n, a condition which is satisfied for measles (following Soper, I have taken $n/m=34{\cdot}1$) and under which the variance results are to this order the same as in the continuous-time model. This suggests that the time to fade-out of infection should be of the same order of magnitude in the two models. While this cannot be investigated theoretically much further, it is possible to handle the present model much more rapidly than the first by Monte Carlo methods, and the empirical formula was obtained from such results,

$$\log_{10}T=2{\cdot}1+0{\cdot}005\,m,$$

for the logarithm of the time (in weeks) to fade-out, when the time unit between "generations" of infections is taken as a fortnight. This empirical formula indicates a rather longer time to fade-out than the theoretical value mentioned above for the continuous-time model, giving even for $m=50$ (average weekly notifications, 25) an extinction time of over four years.

The effect of (iii) might be expected to offset this increase in time to fade-out, as fade-out is actually most likely in the summer months when seasonal variation enhances the probability of low numbers of infectives. This is still under investigation by an extension of the Monte Carlo work to include some where a seasonal variation in infectiveness is included.

The effect of (ii) seems less important than I thought at one time, for it may be shown theoretically that to the first approximation the critical size should be unaffected unless there is actual diffusion of individuals taking place in the population. Even moderate diffusion seems to have no very discernible effect, for some of the Monte Carlo discrete-time results referred to above were obtained by an electronic computer for a square grid of 6×6 cells (cf. Bartlett, 1957), with infection transmitted across cells by diffusion of the infectives, but these results did not appear to differ from the corresponding results for a model where no such spatial subdivisions were allowed for. A further point to note here on the observed statistics is that the actual communities are not by any means isolated, and fade-out is partly offset by diffusion from outside the area, a feature I discussed (Bartlett, 1957) in relation to individual ward figures for Manchester (England). The concept of a critical size is in consequence somewhat less well-defined in practice, for all communities have now become much more freely interconnected than formerly. Epidemiological precautions in the case of certain infections such as smallpox are an obvious example of this fact.

5. THE PROBLEM OF CHICKENPOX

In addition to a comparison of measles statistics in different countries, it would be of interest to cite another type of infection where the ideas already discussed could be examined. Unfortunately, measles occupies a rather unique position as being primarily a children's complaint for which the epidemiological characteristics, such as the susceptible population (and the new "recruits"), are fairly definite. It is, moreover, while incomplete reporting is a complication, officially notifiable, whereas another children's illness, chickenpox, which possesses many features in common with measles, is not, at least in England and Wales. However, it *is* notifiable in the United States, and I took the opportunity afforded by Jane Worcester's collections of statistics to compare the official U.S. notifications of measles and chickenpox for some representative cities.

TABLE 3

Measles and chickenpox monthly notifications for Philadelphia, 1941–43

Measles	*J*	*F*	*M*	*A*	*M*	*J*	*J*	*A*	*S*	*O*	*N*	*D*	*Total*
1941	2,906	4,770	6,991	5,457	2,203	527	77	8	7	10	19	16	22,991
1942	62	93	132	222	194	135	70	38	44	275	1,122	2,770	5,157
1943	4,923	4,759	3,583	1,428	1,198	821	235	10	14	22	23	22	17,038
Chickenpox													
1941	705	555	720	827	582	739	79	25	22	87	360	730	5,431
1942	1,021	991	1,167	1,144	974	531	92	16	18	124	227	387	6,692
1943	556	439	461	432	550	548	214	80	32	160	345	585	4,402

An immediately striking feature was the much more stable seasonal rhythm for chickenpox, in contrast with the more violent measles epidemics which flare-up on average only about once every two years in large communities. It will perhaps be sufficient to quote the monthly notifications for Philadelphia for 1941–43 for the two cases, the figures for New York City, Baltimore and other large towns showing similar characteristics (see Table 3).

There would seem to be two possible reasons for this contrast. One would be that there is no intrinsic epidemic oscillation in the case of chickenpox, in the sense envisaged by Soper (1929) for measles, but that the observed oscillation is due entirely to seasonal causes. The second would be that the intrinsic oscillation is still there, but coincides with any purely seasonal oscillation, and is also of smaller amplitude than in the case of measles. One function of a theoretical model should be to predict this difference in observed behaviour in the two cases. Let us consider the position briefly.

The infectiousness of chickenpox appears to be about 4/5 times that of measles, the period of transmission about 5/4 times (Hope Simpson, 1952). Two relevant characteristics of the stochastic model are (i) its period, (ii) the amplitude of intrinsic oscillations. For the continuous-time model (with no seasonal factor), the period is determined by two parameters, τ and σ (see Soper, 1929 or Bartlett, 1955), the last of which is unaltered by the above changes and the first of which is increased by 25 per cent. This leads to a theoretical diminution in length of period of about 12 per cent. The theoretical damping depends on σ and is therefore about the same; a better parameter for the stochastic model is the amplitude of oscillations, which depends approximately on $\tau^{-1}\sqrt{(\sigma/\nu)}$, where ν is the rate of influx of new susceptibles. The

amplitude should therefore be decreased by about 25 per cent. These changes are certainly in the right direction, but hardly appear sufficient by themselves to account for the observed change in epidemic pattern, and I suspect that there is a further characteristic of chickenpox infection which must be taken into account. This is the connection between chickenpox and shingles (herpes zoster), recent work† on which has indicated that the latter illness is accompanied by an eruption of chickenpox virus which may have been dormant in the individual for many years, and is capable of starting up a chickenpox epidemic in susceptible persons. Obviously such a situation may affect the characteristics of chickenpox incidence considerably, for it represents an auxiliary and long-term reservoir of infection which not only will affect the probability of fade-out of infection, but may be shown to lead to considerable further damping and contraction of the natural period of the epidemics. A more detailed discussion of these points will, however, be deferred until they have been more fully explored.

† See, for example, Hope Simpson (1954).

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