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Reconstruction of diffusion processes at local scales: the 1846, 1882 and 1904 measles epidemics in northwest Iceland

Andrew D. Cliff, Peter Haggett and Rosemary Graham

Because medical records of individuals contracting measles were not kept in northwest Iceland for epidemics before 1904, the spread of the disease in 1846 and 1882 has been traced from notifications of death recorded in burial registers and census returns. Using this evidence, a time-space matrix of measles deaths has been constructed and the dynamics of the diffusion process through a necklace of small communities strung along the coast has been analysed. In each of the three epidemics the mortality curve corresponded closely to an S-shaped logistic model, each new epidemic passing through the area more rapidly than its predecessor. The operation of a neighbourhood effect from a single point of introduction implies that the disease should move in a wave-like form through the area. Whereas the 1882 epidemic advanced steadily as a wave-front progression characteristic of the neighbourhood effect, those of 1846 and 1904 had strong spatial biases towards the parish of Eyri. The intense localization of the outbreaks in 1846 and 1904 appears not to be related directly to distinctive features in the demography or form of the settlement. In 1904 a confirmation service held in Eyri church brought many victims into contact with a measles carrier, but no special circumstances have been reported or can be deduced for 1846.

Introduction

In an earlier paper [1], we undertook a detailed analysis of an epidemic in a remote sub-arctic environment. The paper presented a case study in the historical reconstruction of the spatial and temporal links by which an infectious disease spreads through a human population. The choice of the area (northwest Iceland), the time (the summer of 1904) and the epidemic (measles) were all conditioned by the remarkable richness of the historical records for the events described. The records included not only textual accounts but also numerical information in the form of morbidity and mortality data collected by the local doctor and the parish priest. This richness allowed a rather complete reconstruction of the diffusion processes involved at different geographical scales. But what happens when the textual information is more flimsy and the numerical evidence more circumstantial? Can we use statistical models to throw light on the probable course of an epidemic in time and space?

In this second paper, we follow up that question and explore its implications for the reconstruction of diffusion processes. To allow comparability, we have concentrated on the same geographical area (the parishes of Eyri and Ögur in the Ísafjörður medical district of northwest Iceland) and the same diffusion process (the spread of a measles epidemic through a farming and fishing community). But

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to provide sharp contrast, we have searched back through the records to find the only two measles epidemics to have affected Iceland on any major scale in the nineteenth century – those of the summers of 1846 and 1882. Such waves pre-date the collection and reporting on a systematic basis of the morbidity data by doctors upon which we have relied so heavily in other studies [2]. This collection began in the last decade of the nineteenth century and gives morbidity estimates for a score of infectious diseases for more than fifty local medical districts. The analysis is thus forced back on to contemporary written reports and the parish records of mortality alone.

The study area

A detailed description of the environment in northwest Iceland was given in the preceding paper[3] and will not be repeated here. The two parishes of Eyri and Ögur lie on the southwestern side of the main fjord of northwest Iceland (Fig. 1).

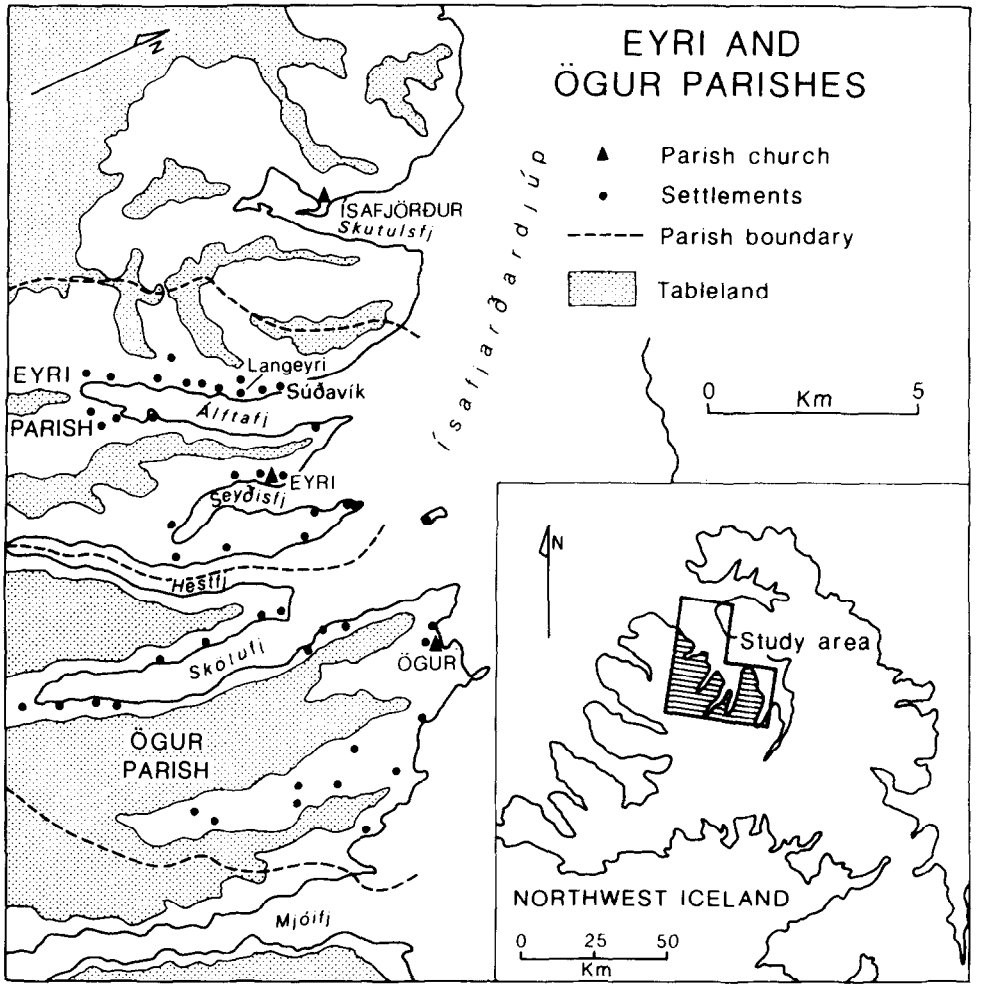

Figure 1. Location of settlements in Eyri and Ögur parishes at 31 December 1903. The stringing of the settlements around the fjord edges is striking. Inland from the fjord wall is tableland at an average elevation of *circa* 850 m. Data source: *Sóknarmannatal: Norður-Isafjarðarprófastsdæmi, Ögur og Eyri i Seyðisfirði* (Parish census: North Ísafjörður Deanery, Ögur and Eyri in Seyðisfjörður), 31 December 1903.

They are split by four smaller fjords (up to 8 km in length) penetrating a table land about 850 m in elevation. Patches of cultivable land are limited to the fjord heads and to narrow strips between the fjord walls and the sea. Small spits of shingle and glacial material form the main sites for local harbours.

At all three dates – 1846, 1882 and 1904 – the settlement pattern appears on the map as a necklace of small communities strung around the coast. In 1845, there were 43 settlements recorded in the parish censuses for Eyri and Ögur together; in 1881, this number was unchanged, but twenty new locations had been occupied by the end of 1903. Along with this increase went a steady growth of population from 526 at the start of the study period to 923 at the end. But as Figure 2 indicates, the picture of growth was not a uniform one. Both parishes had equal populations in 1845; by 1903 Eyri had more than doubled whereas Ögur had grown by less than one half. The spurt of growth in the more westerly parish stemmed from the increased importance of fishing and whaling and is reflected in the slightly larger settlements. From the epidemiological viewpoint, the emergence of four large communities (more than 25 inhabitants) in the western parish with external links through their marine activities was to prove important in the epidemic of 1904.

Textual accounts of the epidemics

It is convenient to divide the evidence for the spread of measles epidemics into two categories: the *textual* evidence considered in this section (doctors' reports, newspaper accounts and journals) and the *numerical* evidence analysed later (morbidity records and parish registers of deaths). The critical textual documents are the handwritten annual reports sent by the local medical officers to Reykjavík at the end of each year. These are full of geographical detail on the pattern of spread and are available from the formal organization of health services in 1896 to the end of World War II. They provide a unique 50-year-long window on to the operation of diffusion processes.

Before 1896, the number of physicians was so small, their geographical areas of responsibility so large, and the reports so brief and unquantified that only glimpses of the sequence of spread can be gained. The general picture of measles in Iceland in 1846 is given in the manuscript report of the Chief Physician (Landlæknir) for Iceland [4]. Iceland had been free of measles since 1788 and, since the physician himself had not met the disease during his student period at Copenhagen between 1812 and 1820, his printed instructions on how to cope with the epidemic had to be based on textbook accounts. Although the winter fishing season had been a good one, March and April were exceptionally cold and stormy, causing an unusual concentration of fishing boats and their crews in the harbours. The first Danish boat of the year arrived in Iceland on 3 April and reported that a measles epidemic was in progress in Copenhagen. Despite the quarantining of incoming boats, cases began to be reported in Iceland itself from early May. By late May, the Chief Physician reported that "there was now no possibility of stopping the disease"[5] and by the end of June it had spread over most of southern Iceland, claiming the Landlæknir himself as one of its victims.

Textual evidence on the spread of the measles epidemic in northwest Iceland comes from the report written to the Landlæknir from A. P. Jensen, the surgeon for Iceland's western country. Measles began to occur in the district in early July, appearing simultaneously at two places on opposite sides of the main fjord, about

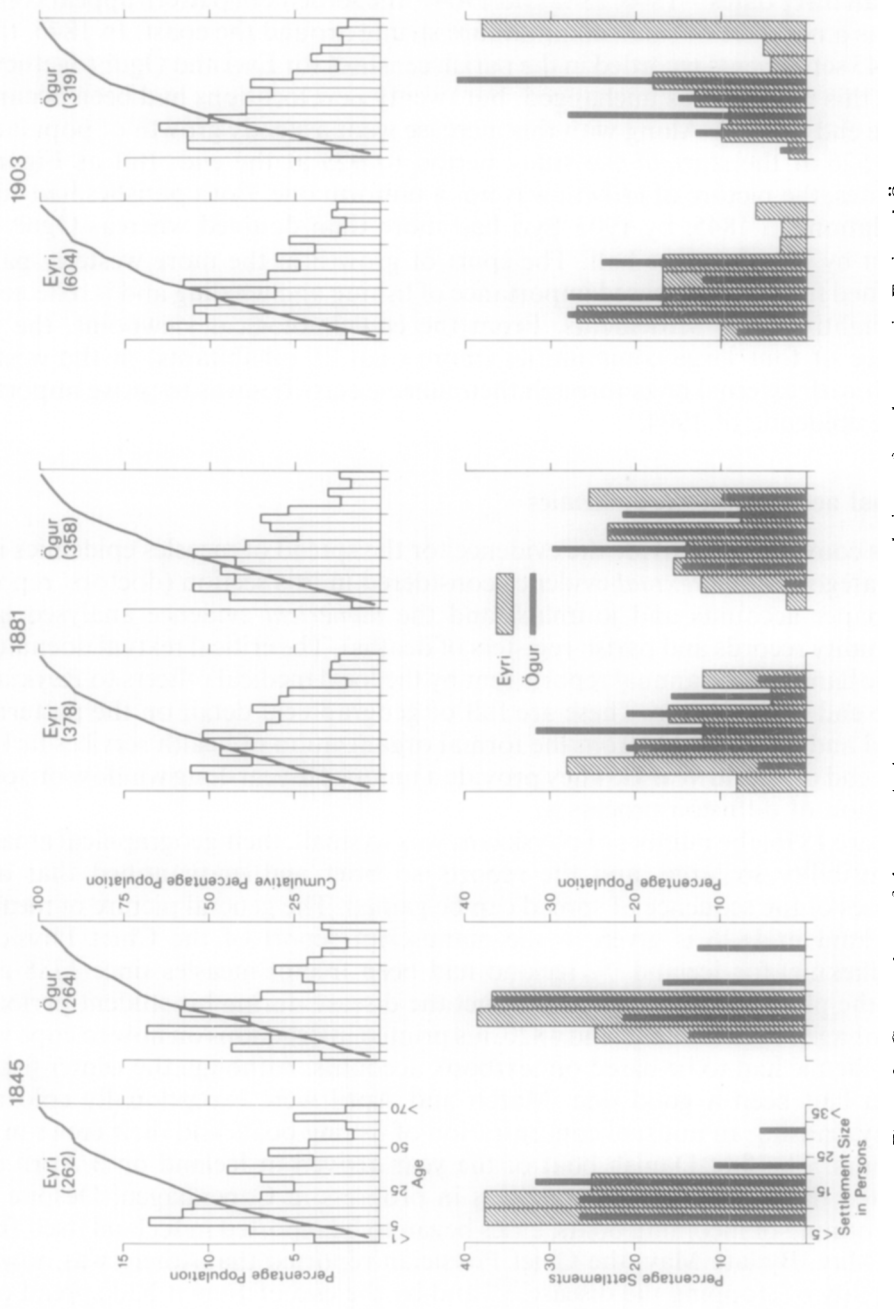

Figure 2. Comparison of the population age structures and sizes of settlements in Eyri and Ögur parishes at each of the census dates, 31 December 1845, 1881 and 1903. Data source: as Figure 1.

10 miles northwest of Ísafjörður. Although Jensen suggests that "the disease must therefore be assumed to have transmitted itself [across the fjord] by air"[6], it seems unlikely in the context of modern virological knowledge. Once established in the fjordland area, the disease spread very rapidly indeed. As Jensen comments "it would be easier to count the people who escaped the disease than to count those who were affected by it, for it spared very few. . . . If the disease has afflicted this part of the country before then it must have been very long ago, for the oldest men I talked to did not know of it and seventy to eighty year olds of both sexes were afflicted just as much as younger people."[7] Jensen notes that some of the very few to escape were Danes who had had measles before in their home country. No figures are included in Jensen's report and the surgeon comments that "my activity could only extend to patients in a couple of parishes; the far greater proportion used no medicine."[8]

Information on mortality caused by the epidemic is limited. The Landlæknir states that, for the fishing villages near Reykjavík where conditions were poor "the mortality there was supposed to have been about 70 of 1250 people [*circa* 5·6%] . . . of whom a large number . . . were certainly old and fairly elderly people, some very delicate children . . ."[9] In Reykjavík, "the mortality among the population of nearly 1500 was only 37 [2·5%], in all of June and July . . ."[10] In the Ísafjörður medical district, A. P. Jensen simply commented, "The mortality is unknown to me, but can hardly be supposed to be notably greater than usual."[11] As we shall see later, this was an understatement of affairs.

For the next epidemic in 1882, the only textual evidence to have survived are four handwritten pages of the local medical officer's report to the Landlæknir. The spring was exceptionally bitter with drift ice arriving off the coast in April while "June and July were very cold, with frost and drifting snow occasionally."[12] The bad weather affected the measles epidemic in several ways. First, it ensured that the "fishing villages were packed with fisherman"[13] so that when measles was brought into Ísafjörður by passengers on the mail boat from Reykjavík on 19 May it spread very quickly. Second, the severe cold and unsanitary, wet conditions led to many complications (diarrhoea and chronic bronchitis), so that the deaths directly or indirectly attributable to measles were extremely high. But third, and in some compensation, the local medical officer (Þorvaldur Jónsson) had two unexpected medical colleagues to help him a brother district officer for the northern region who was forced to stay in Ísafjörður by the pack ice, and a ship's doctor from the naval steamer *Diana* which was also stranded for some time.

Together the three medical officers faced an epidemic which between mid-May and late-August brought illness on a massive scale to this isolated region. From a total population of 5500, some 250 (*circa* 4·5%) died of measles in three months. Attention is drawn to the strong regional variations in mortality, viz:

> The mortality rate varied greatly in the various parishes. Thus only 37 people (or $2\frac{1}{2}$% of the total population of c. 1600) died of measles in the four most westerly parishes; here the populace lives mostly by farming and on the whole under better sanitary conditions, and there are no large fishing villages. On the other hand, the mortality rate in some parishes where the populace for the most part lives by fishing rose to 6% to 8% (in one, over 10%). In connection with this it must be noted that among those who died in the fishing villages are many who did not belong there, but in other parishes or districts and they

> were only staying there for the fishing, from which it follows that the mortality rate in the fishing places seems relatively to be very much higher compared with the hinterland parishes than it is in reality.[14]

The appointment of a new Landlæknir (Jónas Jónassen) in 1895 and the reorganization of medical districts in 1899 brought with it the accurate and regular reporting of the patterns of morbidity and mortality. By 1904, the manuscript accounts of the medical officers had grown in length and detail, and that for the Ísafjörður medical district contains an account of the summer measles epidemic which is remarkable for its geographical detail. The course of the epidemic has been described in full in our earlier paper[15] and will not be repeated here; sufficient to say that both Eyri and Ögur lay near the central and most heavily affected area. The pattern of social contacts (notably a Whit Saturday confirmation service) played a critical part in determining the geographical distribution and timing of the epidemic wave. Despite its size and intensity, the death rate in 1904 was lower than in the two previous outbreaks.

Numerical evidence for the epidemics

The textual record takes us thus far in unravelling the diffusion processes underlying the three epidemics. We now turn to the numerical record to see what additional information about the processes can be gleaned from it. In nineteenth century Iceland, the only quantitative sources which can be used for such a study are the parish censuses and returns of births, marriages, deaths, confirmations and in- and out-migration made annually by the Lutheran priests. The nature of these sources is considered fully in our earlier paper.

One way of analysing these data is graphically. As an example, it is evident that the descriptions of the three waves based upon the written reports of the various medical officers give only a general guide to patterns of mortality in the epidemics. However, using the records of the Lutheran priests, Figure 3 can be prepared for the parishes of Eyri and Ögur. The diagram makes clear, for the 1846 epidemic, the strong age bias in measles mortality towards the young and the old commented upon by the Landlæknir in the Reykjavík region. In contrast, the more severe epidemic of 1882 produced measles deaths indiscriminately across all ages up to about 50. In 1904, deaths from measles were confined to the under 20 years old population – that is, those born since the 1882 epidemic, which had been on such a scale that "on average all work stopped for three weeks [in the area] on account of the epidemic"[16] and "affected everyone who had not had the disease before (in 1846)."[17]

More sophisticated analysis of data such as these returns us to our original question: Can statistical models be used to throw light on the probable course of an epidemic in time and space? In order to answer this question, a methodology appropriate to the problem being studied is first outlined. The empirical findings produced by it for the Icelandic data are then presented; finally, an assessment of the approach is made.

Methodology

A wide variety of methods is available for the analysis of diffusion processes. For each of the epidemics studied here, a time-space matrix of mortalities from measles was constructed. Since mortality levels are small compared with case

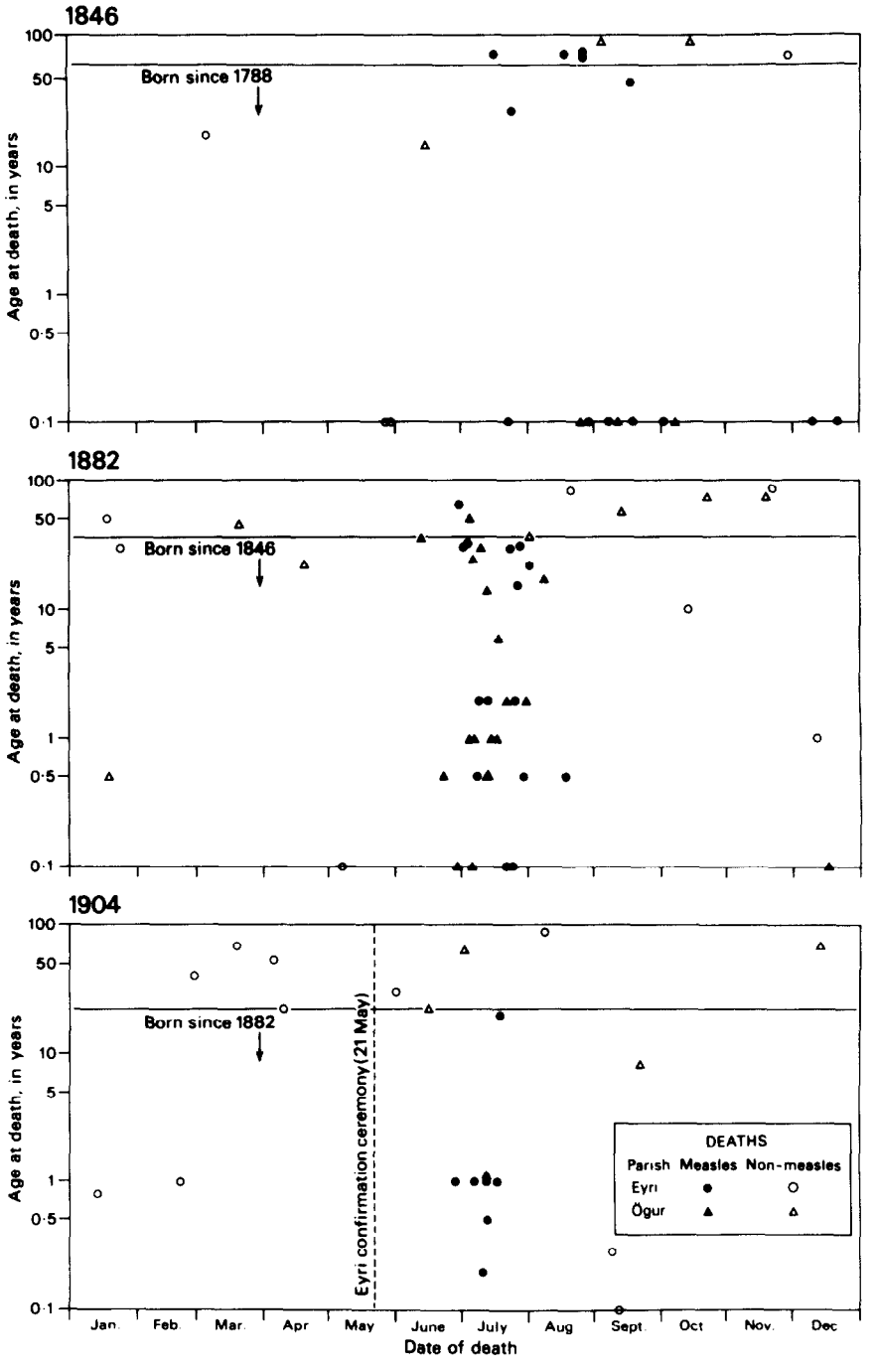

Figure 3. Temporal distribution of deaths in the study area in 1846, 1882 and 1904. The vertical axes of the graphs are logarithmic, with 0·1 indicating deaths within 35 days of birth. Lines indicating people born since the preceding measles epidemic are drawn. The date of the Eyri confirmation ceremony, which played a crucial role in the 1904 epidemic, is also marked. Data source: *Prestsþjónustubók, Norður-Ísafjarðarprófastsdæmi, Ögur og Eyri í Seyðisfirði* (Church register, North Ísafjörður Deanery, Ögur and Eyri in Seyðisfjörður) for the three years.

levels, the matrices were built on a binary basis, recording 1 if a death occurred at a given time-space location and 0 otherwise. For each epidemic, the day on which the first death from measles was recorded in the parish records was called day 1 and subsequent days numbered serially thereafter, up to and including the last recorded death. This procedure yielded the following time scales for the epidemics:

Epidemic	*Duration*	*Matrix time*
1846	16 July–21 December	1–159
1882	12 June–17 August	1–67
1904	27 June–31 July	1–35

Geographical location was recorded in the conventional way on a Cartesian co-ordinate system.

Such time-space matrices can be examined in three different ways. In *one-dimensional analysis*, spatial effects are ignored and the purely longitudinal time-series behaviour of the epidemic is the primary focus of interest. However, many geographical studies ignore the dynamics of systems and concentrate upon a *two-dimensional cross-sectional analysis;* each map is treated as a snapshot slice of the state of the system at a particular point in time. The most complex approach is to meld the previous two treatments into a combined time-space analysis (*three dimensions*), in which the time-based interaction of spatial units upon each other is examined.

In Table 1, we have summarized the models employed to analyse the three epidemics according to the divisions suggested above. It is generally accepted in diffusion studies that the temporal build-up of the item being diffused can be described by an S-shaped curve.[18] The logistic model is most commonly employed to characterize this shape. It is therefore of interest to know whether the build-up of an epidemic of an infectious disease displays the same pattern. An S-shaped curve would imply that case levels are low at first as the disease slowly becomes established in a susceptible population. When mixing between susceptibles and infectives occurs and the case load increases, a point of take-off will be reached and rapid spread of the disease will occur. Ultimately, the rate at which new infections happen will begin to level out as the population at risk is saturated by the disease, and the epidemic will begin to die away. To determine whether this is a reasonable description of disease behaviour in the Icelandic epidemics studied here, a logistic model has been fitted to each. Since we are working with mortality data rather than case loads, we are assuming that deaths are distributed through time at the same rate as cases.

One feature of infectious diseases which might cause departure from the logistic model is the nature of the *serial interval* for the disease in question. This is defined as the average time between the observation of disease symptoms in one individual and the observation of symptoms in a second case directly infected from the first. In the case of measles, this is about 12–14 days[19]. We might therefore expect the logistic shape to be complicated by a 12–14 day harmonic reflecting the serial interval of the disease. The temporal pattern of deaths may thus display a similar harmonic. Spectral analysis is a standard method for detecting harmonics in data and, as indicated in Table 1, it is used here for that purpose.

TABLE 1

Methods employed in the analysis of the 1846, 1882 and 1904 measles epidemics, Eyri and Ögur parishes, northwest Iceland

Data domain	Method	Epidemic		
		1846	1882	1904
Univariate time series (1 dimension)	Logistic model	X	X	X
	Spectral analysis	X	X	
Cross-sectional spatial analysis (2 dimensions)	Binomial test	X	X	X
	Kuhn-Kuenne centroid	X	X	
	Trend surface analysis	X	X	
Joint space-time analysis (3 dimensions)	Knox space-time clustering		X	

Blanks indicate use of method prevented by inadequate sample size. Reported deaths, n, in each of the epidemics were as follows: 1846, $n=15$; 1882, $n=34$; 1904, $n=9$.

We now consider the cross-sectional analysis of Table 1. The most commonly noted empirical regularity in the spatial build-up of diffusion phenomena is the neighbourhood effect[20]. The classic summary of this is provided by Hägerstrand[21]:

> The main spatial similarity is, briefly, that the probability of a new adoption is highest in the vicinity of an earlier one and decreases with increasing distance. Later ones seem to be dependent on earlier ones according to a principle for which the term "neighbourhood effect" would be apt.

For infectious diseases, the operation of a neighbourhood effect from a single point of introduction implies that the disease should move in a wave-like form through a study area[22]. Thus, for mortality data, a low-order trend surface of dates of death should be a reasonable model to employ if a simple wave-form exists. Similarly, the geographical centroid of the disease should migrate in a steady fashion with time across the map.

In the same way that the time-series behaviour of an epidemic may be distorted from the logistic shape by the serial interval of the disease, so the spatial transmission may be distorted from the neighbourhood effect most usually because of inhomogeneities in the population through which it is passing. Today, measles is assumed to be a childhood disease. In fact, anyone who has not had measles (or in recent times, been vaccinated) is at risk. The interval between epidemics is thus of crucial importance in determining the size of the population at risk. For the epidemics considered here these are 58 years (1788 to 1846), 36 years (1846 to 1882) and 22 years (1882 to 1904). In addition, as we have seen, mortality from measles tends to be greater in the very young and very old. Parish census records thus become important in establishing whether biases exist in the demographic characteristics of the parishes Eyri and Ögur. The binomial test can be used to determine the probability of a particular demographic bias being the cause of spatial distortions away from a wave form.

If the joint time-space analysis of Table 1 is considered, a neighbourhood effect on successive maps should theoretically result in a "ball" of infection moving through the time-space matrix. That is, there should be a high degree of

dependence of the map at time t upon that at $t-1$, and so on. A test of such space-time contagion has been developed by Knox[23] and is used here.

Numerical evidence: trends over time

We now report the results of the various analyses and assess the extent to which the diffusion regularities noted above exist for the data examined.

The logistic model fitted is given by the equation

$$p_t = (1 + e^{a-bt})^{-1},$$

where p_t is the proportion of deaths in the population at t, and a and b are parameters. The quantity, e, is the base of natural logarithms. The model is readily fitted by ordinary least squares since it reduces to the simple regression form.

$$\ell n\left(\frac{1}{p_t} - 1\right) = a - bt$$

on rearranging terms and taking natural logarithms. Thus b is directly proportional to the average change in mortality levels per unit time.

The results obtained from fitting the model appear in Figure 4 and Table 2.

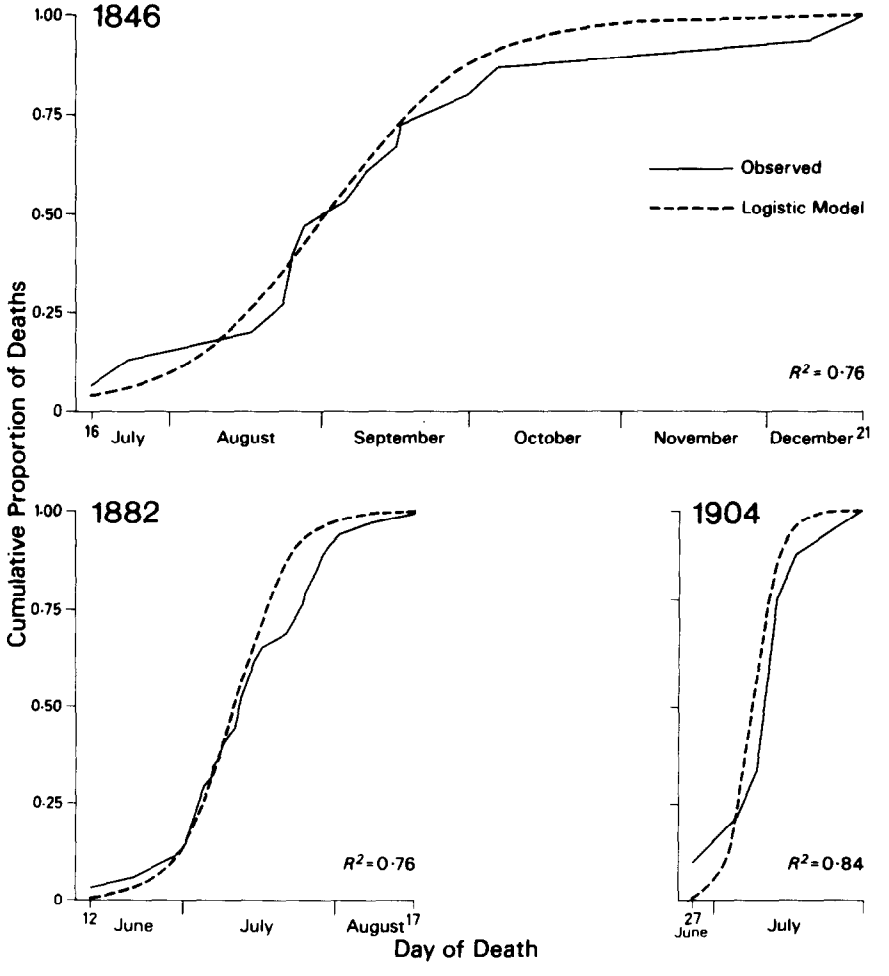

Figure 4. Cumulative diffusion curves for deaths from measles in the study area in the three epidemics. Note the increasing steepness of the curves from 1846 (the slowest wave) to 1904 (the fastest wave). Data source: as Figure 3.

TABLE 2
Results of fitting logistic model to measles mortality data for Eyri and Ögur parishes, northwest Iceland, 1846, 1882, 1904

Epidemic	Parameter values $\hat{a}$	$\hat{b}$	R^2	Sample size
1846	3·33	6·76* (1·11)	0·76	15
1882	5·33	17·5* (1·96)	0·76	34
1904	5·17	39·9* (7·06)	0·84	9

Standard errors for $\hat{b}$ appear in brackets. Both they and $\hat{b}$ are $\times 10^2$.
* Significant at $\alpha = 0{\cdot}01$ level (1-tailed test).

Figure 4 shows that there is a close degree of correspondence between all the epidemic mortality curves and the logistic model. It is also worth noting from Table 2 that the value of b increases with each epidemic; 1846 has the smallest value of b, 1882 the next largest and 1904 the largest. Since b measures the average rate of growth of the process to which the model is fitted, this rank-ordering implies that each new epidemic has passed more rapidly through the area than its predecessor[24]. As shown in Figure 2, the population of the two parishes had become more concentrated in larger settlements at each epidemic date; *ceteris paribus*, we would expect an epidemic to move more rapidly through a more densely populated area than a lightly populated one. This is commented upon by the medical officer of health for Ísafjörður in connection with the 1904 epidemic[25].

> ... it appears that proportionately more people would have died in Álftafjörður than elsewhere in the district. That is doubtless also the result of the fact that in the villages of Tröð and Súðavík it is rather thickly populated and there are many cottars who have worse premises than is the fashion elsewhere ...

In using spectral analysis to check for harmonics in the time-series of deaths from measles in the epidemics, the series were first all reduced to a ± 1 basis by a procedure known as infinite clipping.[26] Let x_t denote the number of deaths reported on day t of an epidemic. We redefine x_t as follows

$$x'_t = \begin{cases} +1 \text{ if } x_t > c \\ 0 \text{ if } x_t = c, \\ -1 \text{ if } x_t < c \end{cases}$$

where c is some selected constant. This is described as infinite clipping of x_t about c. Thus, if $0 < c < 1$, the series is "naturally" clipped into a "square" waveform using a death/no death criterion. The discontinuous nature of the periods with measles deaths (see Figs 5C and E), and small samples, suggest that analysis in terms of square waveforms may well be appropriate. In spectral analysis, waves of different wavelengths or frequencies are fitted to the data, and the percentage of the total variance in the data (power) accounted for by each wave is plotted against wavelength as in Figure 5. Calculation of the power spectrum for clipped

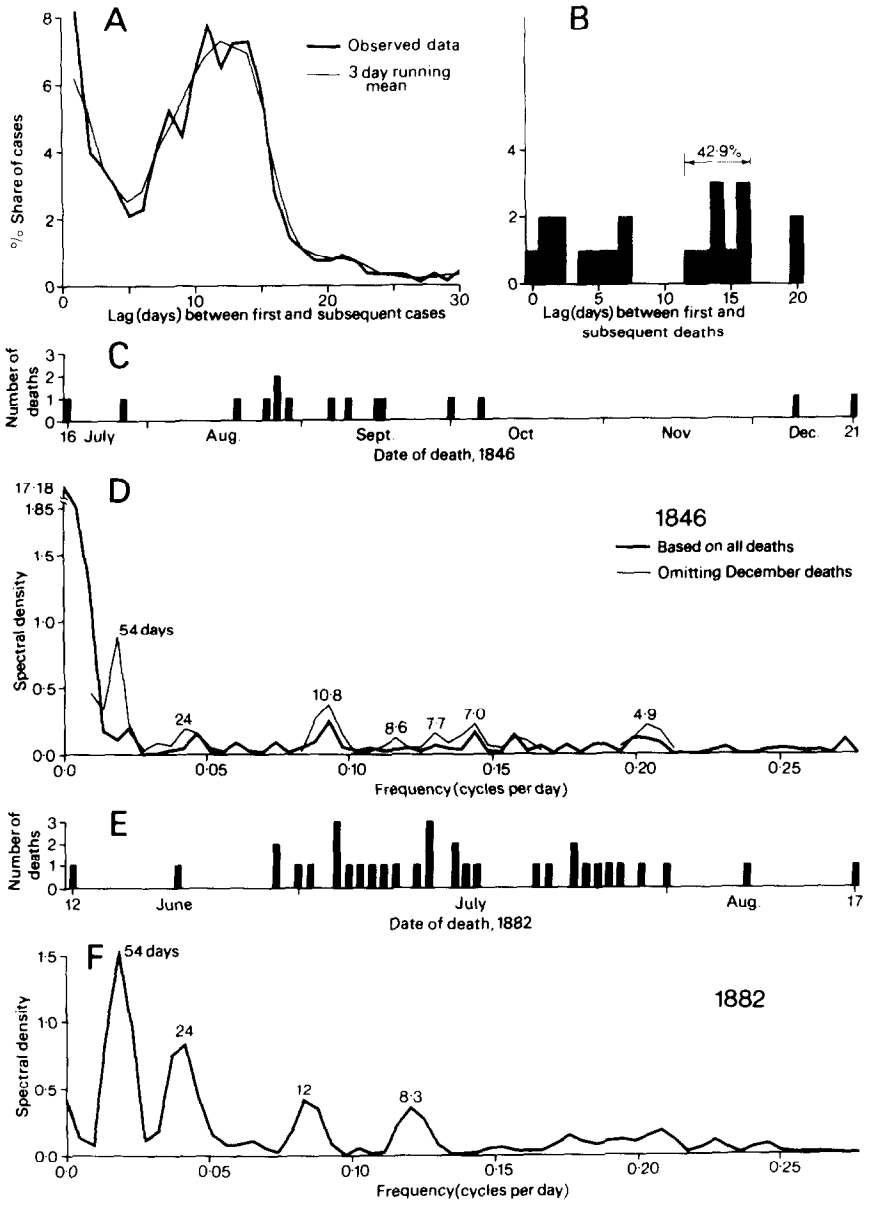

Figure 5. Time-series analysis of measles incidence. (A) Lag in days between first measles case in a family and first day of sickness for subsequent cases. Based on a study by Chapin[27] of 6,345 cases in Providence, Rhode Island. Light line is raw data, heavy line is three-day running mean. (B) Lag in days between first and subsequent deaths from measles in each settlement in the study area for the three epidemics. (C) Date of deaths from measles in the 1846 outbreak. (D) Power spectrum of data shown in (C). (E) Date of deaths from measles in the 1882 outbreak. (F) Power spectrum of data shown in (E). Data source: as Figure 3.

rather than pre-whitened data helps to emphasize any periodicities which may be present.

Figure 5A is based upon Chapin's classic data for Providence, Rhode Island, collected in 1925.[27] The secondary wave peaks between 10 and 15 days after the initial case in a family, a harmonic we would expect from our earlier discussion of the serial interval of measles. In Figure 5B, for every settlement in Eyri and Ögur which reported two or more measles deaths in a given epidemic, the lag in days between first and subsequent deaths was recorded. Results for the three epidemics were pooled to produce the diagram. Allowing for sample size, a secondary peak of linked deaths between days 11–16, separated from deaths caused by what were probably simultaneous infections (lags 0–7), is evident. Excluding the two December deaths in 1846, which are outliers from the main part of that epidemic (Fig. 5C), 42·9% of all linked deaths occurred between days 11–15; this implies that deaths display the same secondary wave feature as do case levels.

For the 1846 and 1882 epidemics, sufficient deaths are recorded to enable the power spectra based upon the clipped data series to be computed (Figs 5D and F). The peaks in the spectra at 10·8 days (1846) and at 12 days (1882) are again highly suggestive of a harmonic in the pattern of deaths which is tied to the serial interval of the disease. The large peaks at 24 and 54 days do not seem to be empirically interpretable. The minor peaks in the spectra between 7–8 days are also within the secondary wave zone of Chapin's data (Fig. 5A).

Numerical evidence: trends over space

The spatial pattern of deaths in the 1846 and 1882 epidemics appears in Figures 6A and 7A. The patterns shown are summarized in two ways in parts B of the same diagrams. Let z_t denote the number of deaths recorded on day t of a given epidemic at the Cartesian co-ordinate position, $\{x,y\}$ on the map. Then the polynomial trend surface model,

$$z_t = \sum_{i=0}^{p} \sum_{j=0}^{q} b_{ij} x^i y^j + \varepsilon_t, \; t = 1,2,\ldots,T$$

may be employed to produce an expected surface for date of death in the study area. An historical example is given in Cox and Demko[28] who examined the dates of occurrence of agrarian riots in parts of Russia, 1905–10. If the spread of an epidemic conforms to the simple wave model implied by the neighbourhood effect, a low-order trend surface should suffice to describe the progression from early to late in the study area. In the same way, the geographical centroid of deaths – here computed by the Kuhn-Kuenne method[29] should migrate steadily across the map.

Such a structure seems to exist for the 1882 epidemic. As shown on Figure 7A, the early deaths are concentrated in the eastern part of Ögur parish. By days 26–39, the centroid had moved westwards, and deaths occurred mainly in a zone along the western border of Ögur parish. In the final phase of the epidemic, deaths were concentrated around Alftafjörður. The cubic trend surface is shown in Figure 7B because it accommodates the slower spread in the central part of the area between days 30 and 40 more accurately than the lower order surfaces. In fact, however, the simple east to west drift is the main component in the pattern. The linear trend surface produced $R^2 = 0{\cdot}26$; the quadratic terms extracted an

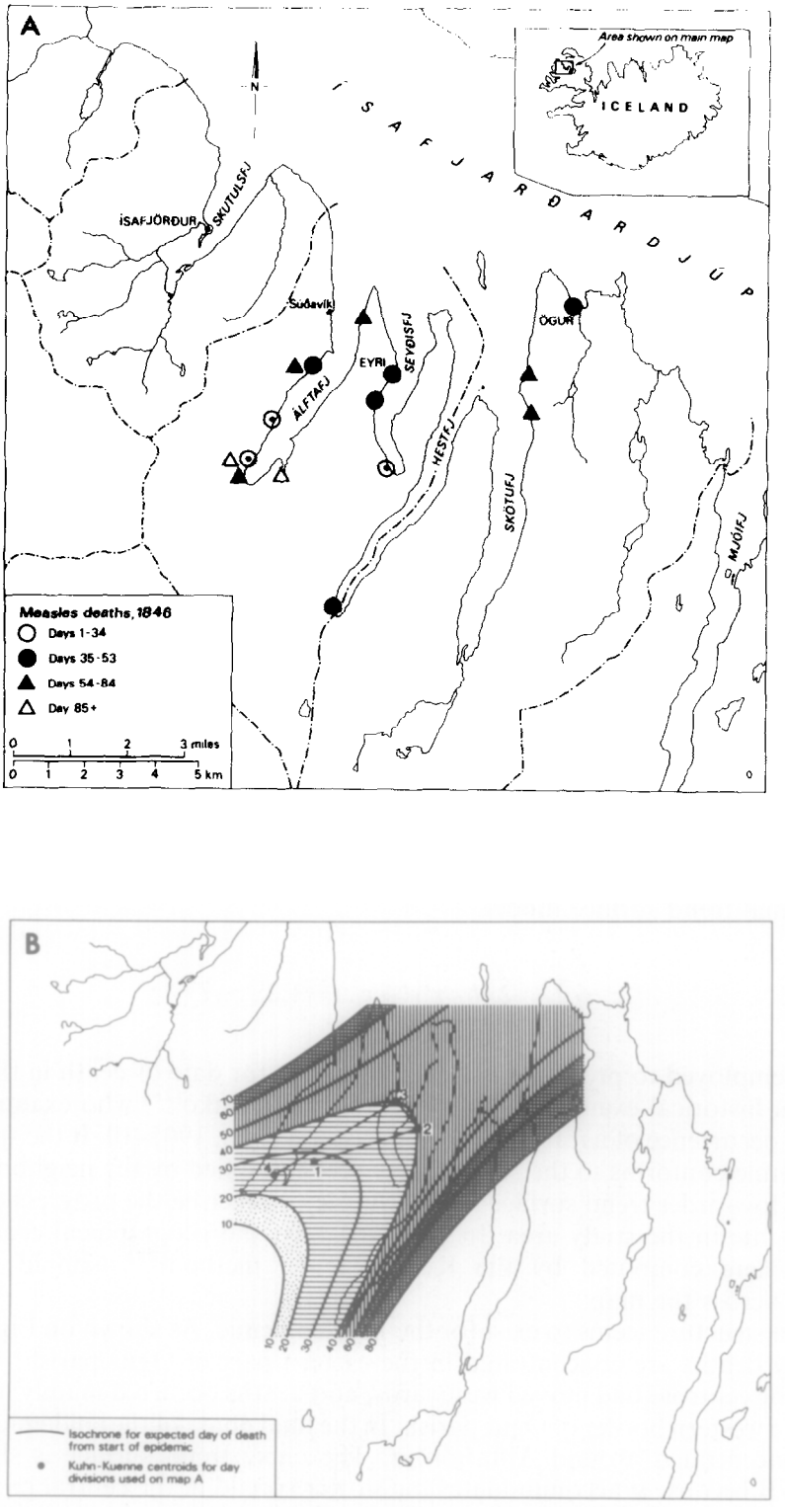

Figure 6. (A) Spatial distribution of measles deaths in the study area for 1846. Small dots indicate location of settlements. (B) Quadratic trend surface for expected day of death from start of the epidemic, with centroids based on day divisions used in (A). Data source: as Figure 3.

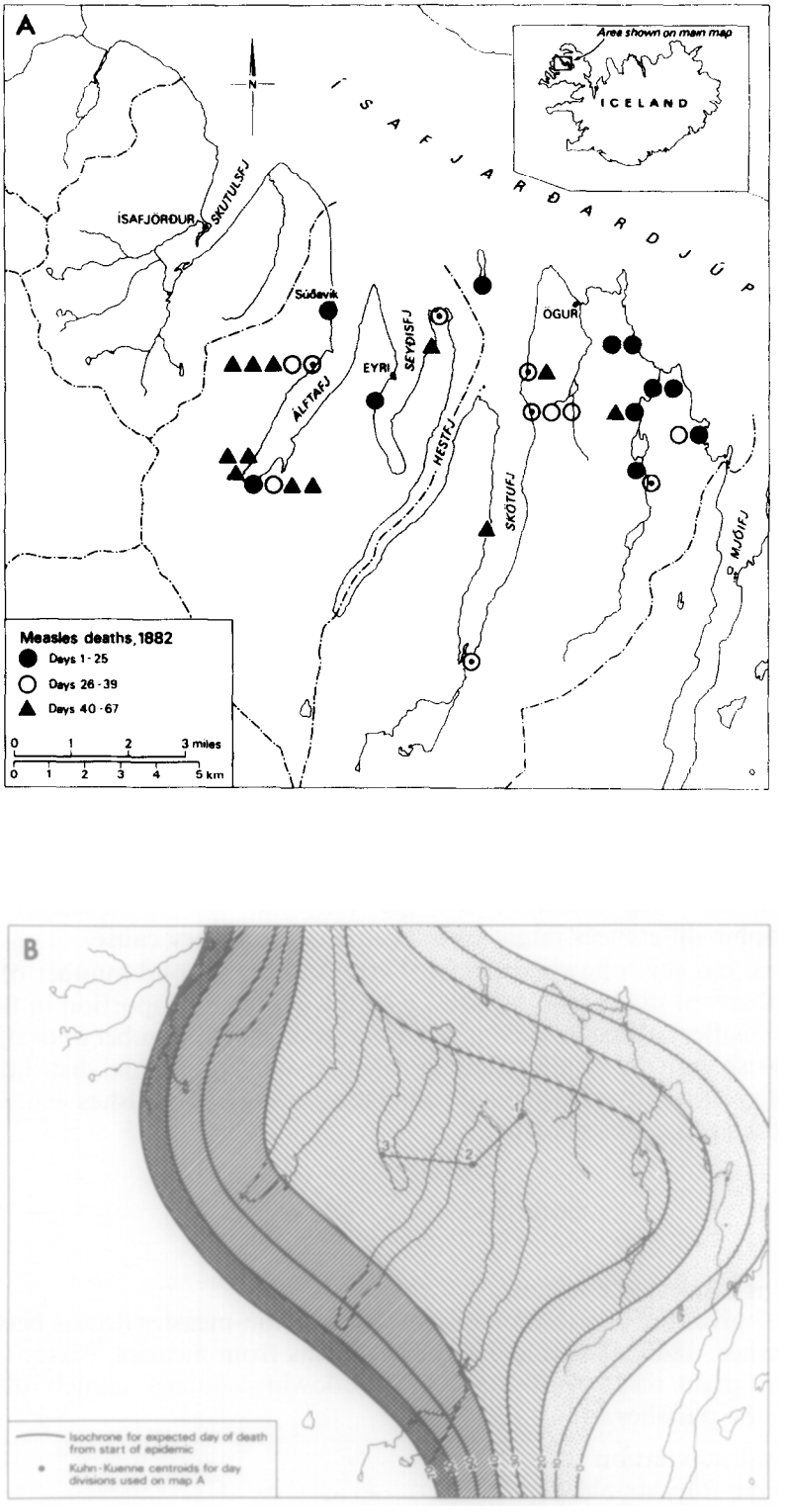

Figure 7. (A) Spatial distribution of measles deaths in the study area for 1882. Small dots indicate location of settlements. (B) Cubic trend surface for expected day of death from start of the epidemic, with centroids based on day divisions used in (A). Data source: as Figure 3.

additional 6·7% explanation; the cubic a further 2·3%. For the linear surface, the analysis of variance yielded

Sum of squares	*Degrees of freedom*	*Mean square*	*F*
Explained = 1583	2	791·5	5·31
Unexplained = 4618	31	149·0	

F is just significant at the $\alpha = 0{\cdot}01$ level (1-tailed test).

For the 1846 epidemic, the pattern is more complicated. Deaths are concentrated in Eyri parish. The epidemic began and finished around the head of Álftafjörður and, as shown in Figure 6B, seems to have pursued an anti-clockwise trajectory as it moved eastwards across the parish, into the western part of Ögur parish and back across Eyri. The quadratic trend surface is illustrated and emphasizes the movement outwards from the head of Álftafjörður.

Insufficient deaths occurred in the 1904 epidemic for the analysis to be repeated in that instance. However, it is clear that the 1846 and 1904 epidemics both stand in sharp contrast to that of 1882. Whereas the 1882 epidemic had the steady wave-front progression characteristic of the neighbourhood effect, those of 1846 and 1904 had strong spatial biases in the direction of Eyri parish. As discussed in our earlier paper, in the 1904 epidemic, this localized intensity may be attributed to the effects of a church confirmation ceremony held at the parish church on 21 May which involved many of the families in the parish and at which a measles carrier was present. The report of the medical officer of health for the area in 1846 gives no hint of special circumstances which might have caused the spatial bias in that epidemic. We therefore use the binomial test to establish whether demographic differences might have been a contributory cause.

The test has the following format. Let n denote the total number of measles deaths observed in the two parishes, p be the expected proportion in the parish with the smaller number of deaths, $q = 1 - p$, and x be the number of deaths, again in the parish with the smaller number. Then, from the binomial distribution, the probability of a split of measles deaths between the two parishes which is more extreme than that observed is

$$\text{prob}\,(x < k) = \sum_{i=0}^{k-1} \binom{n}{i} p^i q^{n-i},$$

where k is a selected constant.

Table 3 gives the distribution of measles and non-measles deaths between the two parishes, 1846, 1882 and 1904. For deaths from measles, biases in spatial incidence might result from any of the following sources, namely differences between the parishes in

(1) total population
(2) under 20 year-old population
(3) under 5 year-old population
(4) number of old people (over 50 years old)
(5) under 5 and over 50-year-olds taken together
(6) number born since the last epidemic.

TABLE 3
Number of measles deaths and deaths from other causes in Eyri and Ögur parishes, 1846, 1882 and 1904

Cause of death	Year	Parish	
		Eyri	Ögur
Measles	1846	12 (4·6)	3 (1·1)
Other		5 (1·9)	3 (1·1)
Measles	1882	16 (4·2)	18 (5·0)
Other		7 (1·9)	8 (2·2)
Measles	1904	8 (1·3)	1 (0·3)
Other		10 (1·7)	5 (1·6)

Deaths as percentage of total population at 31 December in the preceding year are given in brackets.

Sources (1)–(6) represent different ways of measuring the susceptible population. The young and the old have a greater probability of dying from measles than do young adults, while (1) and (6) are more general measures of those at risk. The demographic data were obtained from the parish censuses of 31 December 1845, 1881 and 1903[130].

To illustrate how p was estimated, the following example is given. The total population of Eyri in 1845 was 262 and that of Ögur, 264. Ögur had fewer (3) measles deaths than Eyri (12) in the 1846 epidemic. Thus $p = 264/(262 + 264) = 0{\cdot}502$. In equation (5), the other terms take on the following values: $q = 0{\cdot}498$, $x = 2$, $k = 3$ and $n = 15$. Equation (5) then yields

$$\text{prob}(x < k) = 0{\cdot}004.$$

In that 0·004 is substantially less than, say, 0·05 (the probability associated with the 95% significance level), we may say that the distribution of measles deaths between parishes is more extreme than can be accounted for by the difference in total populations of the two parishes. Application of this procedure enabled Table 4 to be constructed. The analysis was repeated for deaths from other causes to act as a control on the interpretation of results for deaths from measles.

The patterns in Table 4 are clear. The split of deaths from other causes between the two parishes can be accounted for by differences in the demographic structure of the two parishes and is not exceptional. For deaths from measles, however, the picture is quite different. The 1846 and 1904 epidemics display a strong spatial bias, in both cases in the direction of Eyri parish. Moreover, this bias cannot be accounted for by any of the demographic differences examined. In the case of the 1904 epidemic, this is not surprising given the crucial role played by the Eyri confirmation ceremony in the spread of the disease. For the 1846 epidemic we can offer no explanation as to why this bias should exist. Several other attempts were made by the authors to account for the bias by looking at differences in the settlement patterns of the two parishes, but without success.

The 1882 epidemic displays no parish bias, and this is consistent with the wave spread model suggested earlier. It was noted in the earlier discussion of Figure 1 that settlement in the two parishes is strung around the fjord edges like beads on a necklace. An alternative way of looking at the wave spread hypothesis is, therefore, to treat the settlements simply as a chain graph into which disease is introduced at its eastern end. A simple regression of day of reporting against

TABLE 4

Probabilities of observing a distribution of measles deaths and deaths from other causes more extreme than that actually occurring, Eyri and Ögur parishes, 1846, 1882, 1904

Demographic basis for p	Year	Probability of death	
		Measles	Other causes
Total population	1846	0·004*	0·142
	1882	0·249	0·266
	1904	0·022*	0·364
Under 20 year-old population	1846	0·009*	0·202
	1882	0·213	0·241
	1904	0·019*	0·334
Under 5 year-old population	1846	0·003*	0·125
	1882	0·100	0·152
	1904	0·021*	0·358
Over 50 year-old population	1846	0·002*	0·114
	1882	0·217	0·243
	1904	0·029*	0·428
Population under 5 and over 50 years old	1846	0·002*	0·119
	1882	0·160	0·201
	1904	0·027*	0·412
Population born since last epidemic	1846	0·003*	
	1882	0·268	Not
	1904	0·018*	applicable

* Significant at $\alpha = 0{\cdot}01$ level.

distance along the chain can then be attempted (Fig. 8). That is, we postulate the model

$$t_i = a + bd_i,\ i = 1,2,\ldots,n$$

where t_i is day of death from measles in settlement i and d_i is the distance settlement i is from the start of the chain. In Figure 8, we have defined the position of settlement i in the chain graph (x-axis) simply as its ranked distance from Súðavík (assigned rank 1), the westernmost settlement in Eyri parish. The quantities a and b are parameters and, given the coding adopted in Figure 9, we would expect an inverse relationship between t_i and d_i if simple spread along the linear graph from east to west exists. The regression line is shown on the diagram; the parameter values obtained appear in Table 5. A weakly significant inverse relationship was found which provides further justification for regarding the spread of the 1882 epidemic across the parishes as a wave-like phenomenon.

Numerical evidence: trends in time-space

The treatment of the 1882 epidemic as a wave process implies, in time-space, that there should be a high degree of correlation between deaths from measles on the map at t and the maps at $t+1$ and $t-1$; that is, we can imagine a ball of infection migrating through the space-time matrix. Knox [31] has proposed the following test of clustering of infection in time and space. His statistic, I, say, is defined as

$$I=\sum_{\substack{i\\ i\neq j}}\sum_{j} w_{ij}y_{ij}.$$

Here $w_{ij}=1$ if settlements i and j are contiguous in space and $w_{ij}=0$ otherwise; $y_{ij}=1$ if the event of interest (a measles death) occurs in settlements i and j in adjacent time periods, and $y_{ij}=0$ otherwise. The product, $w_{ij}y_{ij}$, will thus be one if and only if deaths occur in adjacent settlements in adjacent time periods. The summation is over all settlements, so that I counts the number of times on which such abutments occur. The sampling distribution of I is given in Cliff and Ord[32]. I is normally distributed for moderate and large numbers of abutments and Poisson-distributed for small numbers.

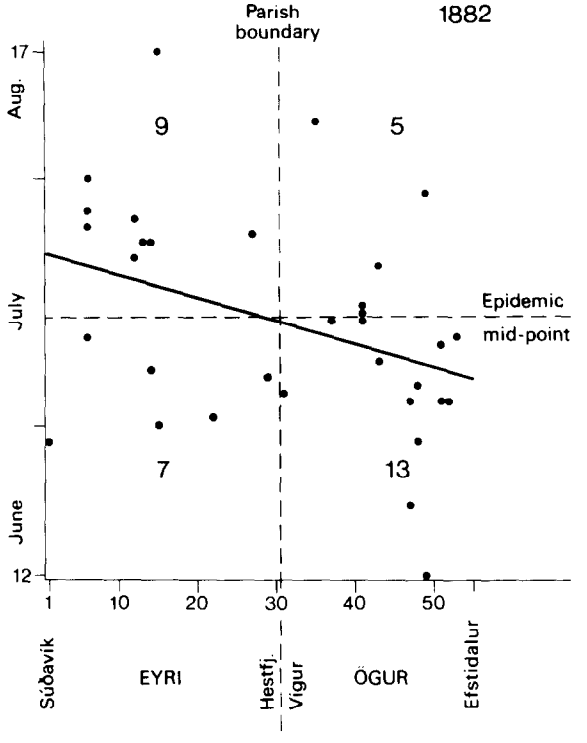

Figure 8. Date of deaths from measles in the 1882 outbreak plotted as a function of distance from Súðavík around the fjord coastline of the study parishes. The simple regression line of date of reporting against distance is shown. Numbers of deaths in each quadrant of the diagram are also given. Data source: as Figure 3.

TABLE 5

Results obtained from regressing day of death against distance from the origin of the chain graph of settlements in Eyri and Ögur parishes, 1882

Parameter values		R^2	Sample size (n)
$\hat{a}$	$\hat{b}$		
41·64	−0·29 (0·13)	0·14	34

The standard error of $\hat{b}$ is given in brackets; $\hat{b}$ is significant at the $\alpha=0{\cdot}05$ level (1-tailed test).

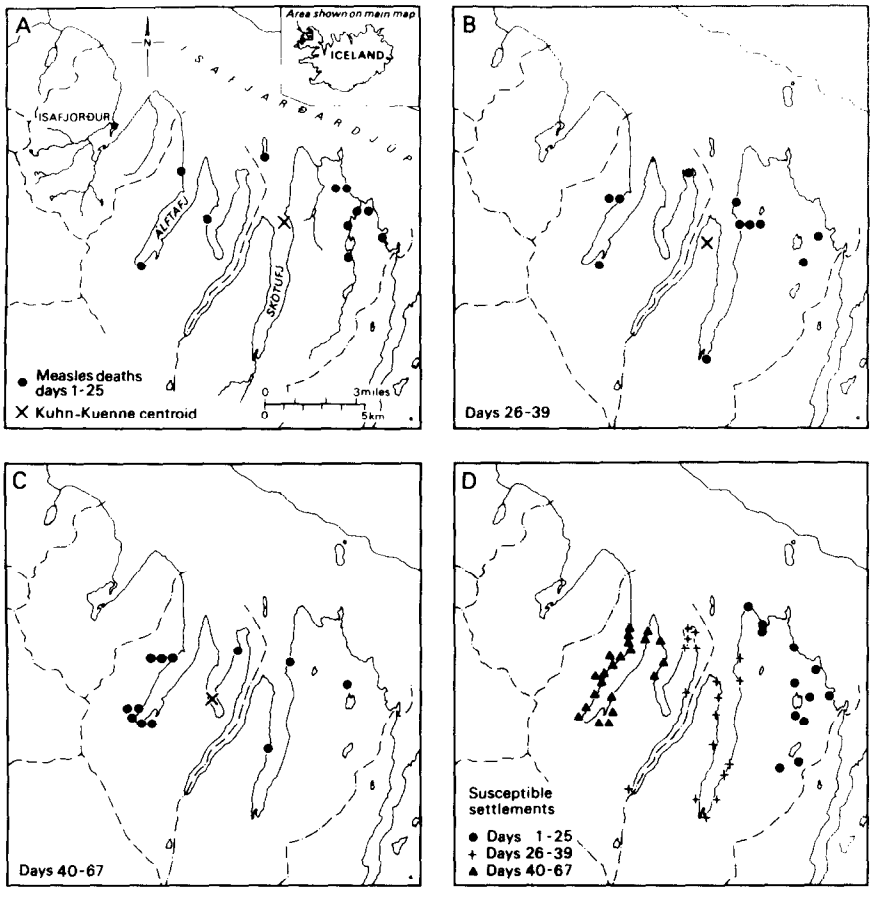

Figure 9. Spatial distribution of deaths from measles in the 1882 outbreak in the study parishes. (A)–(C) Timing of deaths in terms of three phases with centroids at each phase. (D) Settlements that might be regarded as at high risk in each of the three phases. The divisions shown are used as the basis for Knox's space-time clustering procedure described in the text. Data source: as Figure 3.

Figure 9D indicates how the time and space contiguities were defined for the 1882 epidemic. The settlements at risk at various stages of the epidemic are distinguished. In the spread process, we assume that those at risk on days 26–39 can have received the disease only from settlements in which measles death occurred on days 1–25; similarly those at risk on days 40–67 are assumed only to have received the disease from settlements in which measles deaths were reported on days 26–39. Using these definitions, the Knox coefficient was evaluated, yielding the results shown in Table 6. The coefficients obtained were significant at the $\alpha = 0{\cdot}05$ level (1-tailed test). Space-time contagion appears to exist in the data, and we conclude that wave-like transmission of the disease occurs in both time and space.

TABLE 6

Results obtained for Knox's space-time coefficient from measles data, Eyri and Ögur parishes, 1882

Dependent maps	*I*	Normal score	Poisson probability
Days 26–39 on 1–25	20	5·30	0·001
Days 40–67 on 26–39	20	2·92	0·046

Conclusion

Where qualitative evidence of the way in which a diffusion wave has moved exists alongside maps showing the stage-by-stage pattern of spread, the task of reconstruction is a straightforward one. Thus in the 1904 measles epidemic in northwest Iceland, the written records and the data can be woven together into a convincing canvas. But if we push the investigation in the same area back by a generation or two, then one or other of the sources may become weaker. In the case of the 1846 and 1882 epidemics, the mappable data at the micro-scale remain firm (indeed, through an increase in recorded deaths, they become firmer) but the accompanying written evidence on the process of spread of the disease is too generalized and inconclusive to buttress them.

This paper explores a series of ways in which statistical models, some of them elementary, and all used in geographical work, can provide guides to the source and nature of a diffusion process. Inevitably, such models cannot take us further than the stark facts of the mapped distribution permit, and insights (such as the role of the 1904 Whit Saturday confirmation service in Eyri described earlier) will always be denied. None the less, the analyses conducted have uncovered important regularities in the data. First, the temporal study shows that the speed of the three epidemics (as measured by logistic curves) increases substantially from one epidemic to the next, and that the pattern of deaths from measles in each settlement (as plotted by spectral peaks) has a serial interval or harmonic at just that time lag predictable from later epidemiological studies of morbidity from measles. Second, the spatial analysis of events indicates that the 1882 epidemic conforms to a simple wave model, but that those of 1846 and 1904 are more complicated, with a heavy bias towards Eyri parish. Comparison of the demographic and settlement structure of the two parishes provided a basis for explaining differences in non-measles deaths but did not provide a means of accounting for the spatial bias in epidemic deaths from measles. How important differences in occupational structure (a higher proportion of fishermen in Eyri) may be, with the attendant effect of local crew groupings and long-range contacts, cannot be determined from the data available. Finally, by combining the observations from the most severe epidemic (1882) into a time-space matrix and using a development of Knox's test, it is possible to establish that the passage of the disease through the parishes takes the classical form of a diffusion wave.

Despite small and non-extendable numbers of observations, it appears feasible to use statistical models to throw light on the movement of local epidemic waves through rural populations. While the presence of textual evidence (as in the case of the 1904 epidemic) greatly aids interpretation, its absence for earlier historical events (those of 1846 and 1882) does not debar the researcher from making some geographical sense of the spatial diffusion processes which appear to be operating.

Department of Geography, University of Cambridge

Department of Geography, University of Bristol

Trinity College Library, Cambridge

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