On the critical behaviour of simple epidemics

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SUMMARY

We show how ideas and models which were originally introduced to gain an understanding of critical phenomena can be used to interpret the dynamics of epidemics of communicable disease in real populations. Specifically, we present an analysis of the dynamics of disease outbreaks for three common communicable infections from a small isolated island population. The strongly fluctuating nature of the temporal incidence of disease is captured by the model, and comparisons between exponents calculated from the data and from simulations are made. A forest-fire model with sparks is used to classify the observed scaling dynamics of the epidemics and provides a unified picture of the epidemiology which conventional epidemiological analysis is unable to reproduce. This study suggests that power-law scaling can emerge in natural systems when they are driven on widely separated time-scales, in accordance with recent analytic renormalization group calculations.

1. INTRODUCTION

The behaviour of spatially extended, perturbatively driven dissipative systems can often be characterized by simple power laws which emerge from an analysis of the 'avalanches' of activity that propagate through such systems (Bak *et al*. 1988). Attention has focused particularly on the dynamics of sandpile models (Bak *et al*. 1988), forest-fire models (Bak *et al*. 1990; Grassberger & Kantz 1991; Drossel & Schwabl 1992; Mossner *et al*. 1992; Christensen *et al*. 1993; Drossel & Schwabl 1993; Grassberger 1993; Henley 1993; Clar *et al*. 1994; Drossel & Schwabl 1994; Clar *et al*. 1996) and spring-block models (Chen *et al*. 1991; Olami *et al*. 1992). Much analytic and computational effort has been expended in order to gain insight into the emergence of robust spatio-temporal scale-free dynamics which are so often a feature of these systems. In addition, experimental work has begun to test the generality and applicability of some of the models to the phenomena they purport to represent (Held *et al*. 1990; Frette *et al*. 1996).

A remarkable distinguishing feature that some of these highly non-equilibrate systems exhibit is an ability to evolve towards the same scale-free fixed point behaviour irrespective of the choice of initial conditions and without the need to adjust model parameters to specific values. Such behaviour, termed self-organized criticality (SOC), has been promoted as a concept of central importance in understanding the dynamical origin of the abundance of scale-free (fractal) phenomena observed in the natural world.

SOC is an appealing and potentially very powerful hypothesis, but its very generality has ensured a long-running and on-going debate on how it actually comes about, though recently Flyvberg (1996) has sought to calm these turbulent waters by stating the minimum criterion for SOC to be possible. However, lack of consensus need not serve as an obstacle to the application of these ideas to natural phenomena in the wider world.

Biological processes may well prove to be a fertile area in which to apply the SOC concept, as we require models that give robust dynamical behaviour without the need to adjust parameters to exact values (Solé & Manrubia 1995). For example, recent discussions concerning evolution and the 'punctuated equilibrium' hypothesis have been informed by results from Bak & Sneppen (1993) who introduced a simple (possibly oversimplified) model, which has subsequently been elaborated somewhat (De Boer *et al*. 1994; Sneppen 1995; Sneppen *et al*. 1995; Newman & Roberts 1995; Roberts & Newman 1996; Solé & Bascompte 1996), describing an ecology of interacting and evolving species which exhibits punctuated equilibrium behaviour. It has been proposed that evidence from the fossil record indicates that evolution occurs in an intermittent way, with occasional rapid bursts of evolutionary activity separated by longer episodes of relative stability.

Pursuing this theme, it is our aim to use ideas and models developed in a statistical, mechanical context to gain insight into biological phenomena and, in

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particular, to explain the observed patterns of epidemics of communicable diseases. As epidemiological models frequently act as templates for models in the wider field of population biology the conclusions we reach can have implications beyond the issues relating to the spread of disease. In this paper we make extensive use of a model initially introduced by Bak *et al.* (1990), in the context of turbulence, and later modified by Drossel & Schwabl (1992) in order to discuss SOC phenomena. This model, and its variants, are traditionally discussed in terms of forest fires but they are, of course, closely related to models of excitable media and therefore lend themselves quite naturally to interpretion as epidemic models; i.e. trees are equivalent to susceptibles, and fires are equivalent to infectives. In epidemiological language, the forest fire corresponds to the simplest possible spatially explicit susceptible infective (SI) model. Below, we show how the dynamical behaviour of the Drossel & Schwabl (1992) model (forest fire with sparks) can be used to explain the observed patterns of epidemics in small isolated populations. Specifically we describe how, under certain circumstances, epidemiological dynamics appear to exhibit scale-invariant (power-law) phenomena, implying that there is structure present in data sets which were, hitherto, thought to be dominated by largely stochastic effects. We suggest that it might be possible to classify certain highly transmissible diseases using distinct exponents which can, in turn, be calculated from the lattice model. Additionally, we believe that we can explain why less transmissible diseases have different exponents which can take a number of different values according to the location of the epidemic. Our analysis, in terms of scaling and the results of the lattice-based models, presents a unified picture which conventional epidemiological analysis is unable to match. Previous research has established the possibility of scaling dynamics in epidemiological systems (Rhodes & Anderson 1996*a*,*b*). Here we extend those studies to other diseases, showing scaling to be a more general phenomenon than first appreciated, and further, we demonstrate that lattice models exhibiting critical behaviour can be used to explain the observed patterns of infection in several situations.

In § 2 we recall the definition of the forest fire model and discuss how it can be used to interpret Type III epidemics (defined below) of a number of diseases in small populations. In § 3 we present the analysis of the scaling dynamics observed in the epidemiological data sets, and these are interpreted with the aid of results from lattice simulations in § 4. We conclude in § 5 with a discussion and suggestions for further research.

2. THE FOREST-FIRE MODEL

The forest-fire model was first introduced by Bak *et al.* (1990) in an attempt to introduce a new meta-model for turbulent phenomena, scaling and criticality. It is an extremely simple lattice-based model. The sites of the lattice can be in any one of three states; empty, occupied by a tree, or occupied by a burning tree. The system is updated in parallel with periodic boundary conditions using the following rules: (i) Trees grow at empty lattice sites with a probability p. (ii) Trees on nearest-neighbour sites to burning trees catch fire. (iii) Burning trees become empty sites on the next time-step. However, it turns out that on large lattices the model becomes increasingly deterministic in the limits that were expected to show critical behaviour, with the emergence of spiral fire-fronts. Extensive computational simulations have established that no self-organized critical behaviour occurs in this model. Johansen (1994, 1996) has used the model, and variants of it, as an instrument to illustrate dynamical processes in epidemiology. Principally he demonstrated that a simple spatially explicit epidemic model can exhibit sustained oscillations without the need for additional seasonal forcing.

This model is the latest in a number of lattice-based models which have been introduced in order to investigate disease dynamics in spatially distributed host populations. One of the earliest was formulated by Bailey (1965, 1975) in connection with percolation theory. This approach was developed by Mollison (1977) and Mollison & Kuulasmaa (1985) who proposed that the lattice technique could be used as a general framework within which to describe spatial heterogeneity in epidemic models. More recently Cox & Durrett (1988) and Durrett & Neuhauser (1991) have developed the spatial contact model for individual-based spatial models. A detailed summary of this work and how it compares with other approaches to modelling spatial heterogeneity can be found in Durrett & Levin (1994*a*,*b*) and Durrett (1995). Perhaps closer in spirit to the forest-fire models and the emergence of scale-free phenomena, which we discuss in this paper, are the early studies of Durrett & Cox (1988), Grassberger (1983), Cardy & Grassberger (1985) and Cardy (1983), who used scaling dynamics to characterize disease spread through stationary spatially distributed host populations. In an individual-based lattice model including host movement, Boccara & Cheong (1992, 1993) and Rhodes & Anderson (1996*c*) demonstrated the importance of host mobility on the rate of epidemic spread. In a similar fashion to Bocarra & Cheong (1993) we have also investigated the emergence of scaling dynamics in mobile host populations (Rhodes & Anderson 1996*d*). Lattice models of processes in population biology have also been extensively investigated in host–parasitoid systems, where spatial heterogeneity can stabilize otherwise unstable dynamics and generate coherent pattern formation (Hassell *et al.* 1991; Rand *et al.* 1995; Rohani & Miramontes 1995).

(*a*) *Critical dynamics*

A simple modification of the forest-fire model can have dramatic consequences to the spatio-temporal development of the system. The 'lightning' mechanism allows the forest-fire model to exhibit critical dynamics, and was introduced by Drossel & Schwabl (1992). The rules of the model (i)→(iii) are retained, as stated above, only now an extra rule is added which allows

any tree which has no nearest-neighbour burning trees to spontaneously catch fire with a probability f. With the addition of lightning, the dynamical behaviour of the forest-fire model is radically different to the basic model of Bak *et al.*(1990). In the next section we show how this type of model can, under certain circumstances, successfully account for the observed behaviour of communicable diseases in human populations. Specifically, we will be investigating Type III epidemics. These are epidemics triggered by the arrival of an infected individual in a largely susceptible community. The epidemics are of a relatively short duration and the disease eventually dies out in the population only for it to be possibly introduced again at a later date from outside.

The forest-fire model with sparks was primarily intended to provide an example of a self-organized critical system. Analysis in this model is done in terms of looking at the numbers of event sizes and durations. Each forest cluster burning event has a size, s, defined as the total number of trees burned during the fire and a duration, t, defined as the number of time-steps between the initiation of the fire and the time when the fire is completely extinguished. The critical dynamics of this model, characterized by various scaling exponents, are now well known but there still remains a question of interpretation as to whether it is a true example of SOC. There are two parameters in the model; the tree growth rate, p, and the lightning rate f. Critical behaviour occurs when the following condition is met;

$$T(s_{\max}) \ll p^{-1} \ll f^{-1}, \tag{1}$$

where $T(s_{\max})$ is the time taken for a 'large' (i.e. of the order of the lattice size) forest cluster to burn down. Thus, we are dealing with a series of well defined tree-cluster burning episodes which are separated by epochs of straightforward tree growth (Drossel & Schwabl 1992; Mossner *et al* 1992; Christensen *et al*. 1993; Drossel & Schwabl 1993; Grassberger 1993; Henley 1993). SOC behaviour occurs when, as the condition in equation (1) states, there is considerable separation of time-scale between the rate of tree growth and the rate of lightning strikes. It is argued that this is SOC because all that is specified is a separation of time-scales rather than a tuning of f/p to some precise value.

The central quantity of interest in our work is the size distribution of the burning clusters (i.e. the number, N, of burning clusters of size s), conventionally denoted by the scaling exponent τ, such that

$$N(s) \propto s^{-\tau} C(s/s_{\max}), \tag{2}$$

where $C(x)$ is a monotonically decreasing function with $C(x)$ being unity at $x < 1$ and $C(x) \to 0$ for $x \to \infty$. Other exponents are also defined, but it is not our purpose to provide an exhaustive review here. Simulation results satisfying equation (1) show that in two dimensions $\tau = 2.14 \pm 0.03$ (Grassberger 1993; Henley 1993; Clar *et al*. 1994) irrespective of precise choices of particular values of f and p.

Recent work on a real-space renormalization scheme for the forest-fire system has demonstrated how good approximations to the exponents can be calculated analytically (Rohani & Miramontes 1995; Loreto *et al*. 1995). However, from an analysis of the transformation's fixed point behaviour, this type of calculation indicates that the forest-fire model is more like a conventional critical system, with f/p (the measure of the separation of time-scales) acting as the tuning parameter. Similar calculations using this framework for other dynamical systems indicate that SOC can arise whenever phenomena are driven on very different time-scales (Loreto *et al*. 1995).

Whether or not this particular version of the forest-fire model is strictly within the class of SOC systems, from a biological perspective it is an extremely useful model. In many biological systems it is simply not possible to measure accurately the necessary rate parameters. However, it *is* usually possible to estimate the relative magnitudes of those rate parameters. Clearly, if we can identify a biological system whose dynamics is dominated by widely different time-scales we might have a chance of observing the non-trivial critical behaviour predicted by the forest-fire model. Then, as in models of other critical phenomena, it would be possible to use a simplified model of the epidemiology containing the most essential biological factors, which would then act, in turn, as a concise statement of the origins of the observed dynamics.

3. TYPE III EPIDEMICS

In epidemiological terms the lightning mechanism has an appealing interpretation. It represents the occasional immigration of an infective individual into a population. Such an individual is referred to as an index case. Providing that immigration of index cases occurs sufficiently infrequently it permits the possibility to realise the condition for the separation of time-scales. In a population of constant size, births are continually taking place. For example, in a population of N individuals with an average life expectancy of around T years, there will be roughly $N/T(365)$ births per day. For populations of around 30 000 individuals with a life expectancy of around 70 years there would be approximately one new birth per day. If, in addition, this population is geographically isolated, then contact with the rest of the world will be limited, providing restricted opportunities for infectious agents to enter the population. So an index case may arrive in the community on average only once per year. Thus, there is a clear separation of time-scales for the rate of appearance of susceptibles (births) and the rate of appearance of index cases, and the consequent potential to observe critical dynamics. However, equation (1) is not fully satisfied in that births *do* occur whilst an epidemic is in progress. Despite this, the arrival of susceptibles is sufficiently low so as not to disrupt the scaling pattern, and each epidemic outbreak remains a well defined event without the arrival of susceptibles significantly fuelling the epidemic whilst it is in progress.

To test whether critical dynamics can occur in an epidemiological system the following conditions need to be fulfilled:

(i) The host population must be 'small': this is to ensure that when disease does arrive in the population it does not maintain itself indefinitely, so the population must be well below the endemicity threshold for the disease we are considering (i.e. Type III epidemics occur). Also, from equation (1), to observe critical dynamics we need to ensure that during the progress of an epidemic new births of susceptibles are not so great as to fuel the persistence of that epidemic. This condition ensures that p is small.

(ii) The host population must be isolated: this is necessary to ensure that we can clearly understand by what routes infection can enter the population. An island population would be ideal since we would be dealing with a well defined population and isolation would ensure that infection arrives only infrequently. This condition means that f is small, and that $f/p \ll 1$.

(iii) The population we consider must have historically long running and accurate time series for various communicable diseases, so that we have a chance of measuring the distribution of epidemic sizes and durations.

Surprisingly there is one particularly good example of a population that meets all the above conditions. The people of the Faroe Islands are a small geographically isolated population of around 25 000 individuals situated in the North Atlantic, midway between Scotland and Iceland, and for which, fortuitously, there exists long-running and detailed epidemiological data sets. Historically, as a part of the Kingdom of Denmark, the Faroese adopted the Danish system of maintaining accurate records of the presence of disease in their community (Cliff *et al.* 1993). From these epidemiological records, from the pre-mass-vaccination era, we have established the total number of cases per month for three communicable diseases and undertaken an analysis to investigate whether scaling dynamics occurs.

(*a*) *Measles*

The monthly incidence of measles virus infection in the Faroe Islands is shown in figure 1*a*, for a time span of 100 years from 1870 to 1970. Data from 1866–1869 is entirely null; it is used in our analysis, but not shown on the graph for ease of presentation. Data from 1940 to 1943 is not recorded. Measles is an easily diagnosed, communicable disease with distinctive symptoms, so this time-series is believed to be very accurate. There are 1200 months of data with measles present for 291 (i.e. 24%) of those months. The time-series is made up of 60 distinct epidemics ranging in size from one case up to 4456 cases. For a population of the size of these islands', there will be approximately one birth per day ($p^{-1} = 1$), and given that there are 60 epidemics in 97 years $f^{-1} = 365 \times 97/60 = 590$. This gives a lower limit on f/p of *ca.* $1/600$.

We define an epidemic as having a duration t, where

$$t = M_{\text{end}} - M_{\text{start}}, \tag{3}$$

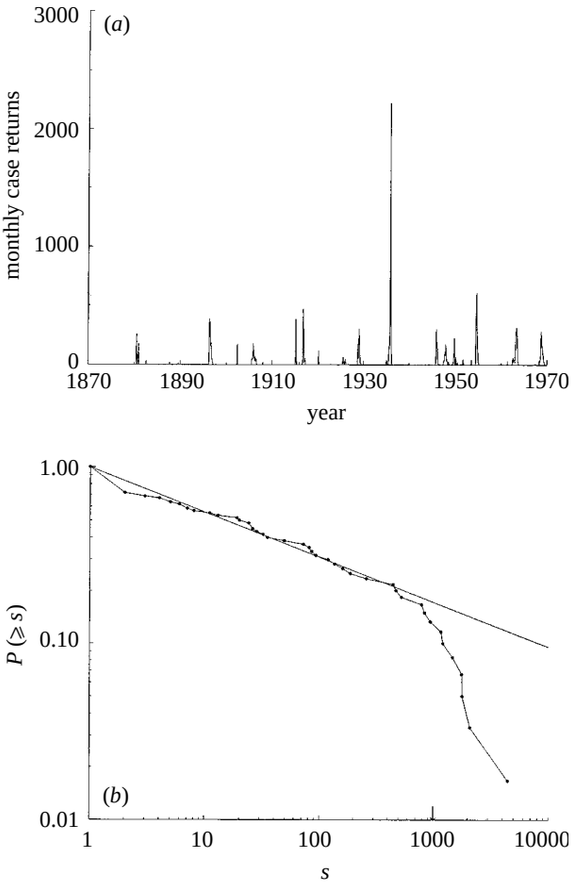

Figure 1. (*a*) The monthly measles cases returns for the Faroe Islands (population *ca.* 25 000); an example of Type III epidemic pattern. (*b*) Epidemic size probability distribution for the Faroe Island measles data (log–log plot). The best-fit line shown is calculated so as only to apply to epidemics of up to 1000 cases. The gradient of the plot gives $-\tau + 2$.

and M_{start} is the first month when cases in an event first appear, and M_{end} is the next month when there are no more cases present. An epidemic outbreak can have a duration of one month up to any integer number of months. An epidemic event has a size, s, where

$$s = \sum_{M_{\text{start}}}^{M_{\text{end}}} C(M), \tag{4}$$

and $C(M)$ is the number of recorded cases of measles in the month M.

The distribution of cluster (i.e. epidemic event) sizes is conventionally denoted by $sn(s)$ and scales as

$$sn(s) \propto s^{-\tau+1}, \tag{5}$$

providing $s < s_{max}$.

This leads to the probability distribution (Grassberger 1993) for observed epidemics

$$P(s) \equiv \text{prob}\{\text{epidemic} \geq s\} = \frac{\sum_{s'=s}^{\infty} s' n(s')}{\sum_{s'=1}^{\infty} s' n(s')} \propto s^{(\tau-2)} \tag{6}$$

Plotting $P(s)$ as a function of s on a log–log plot should give a straight line of gradient $-\tau + 2$.

From the scaling plot in figure 1*b* of the measles epidemic data we see that the measles epidemic events do exhibit scaling dynamics for epidemics up to size *ca.* 1000 and estimate that $\tau = 2.265 \pm 0.014$ (95% confidence interval). A corresponding calculation shows that the epidemic event durations also exhibit scaling too, but we confine our discussion to the size distributions.

(*b*) *Whooping cough*

Whooping cough (pertussis) is a communicable viral disease which causes very distinctive symptoms. It can be easily diagnosed when the characteristic cough is evident, and therefore not confused with other diseases. Infection can occur and atypical symptoms exhibited. The monthly case returns for whooping cough in the Faroe Islands are shown in figure 2*a*. This too is a clear example of a Type III epidemic pattern with the infection present in around 29% of months; 45 distinct epidemics are recorded in just over one hundred years. Calculating the epidemic distribution function, we find from figure 2*b* that $\tau = 2.255 \pm 0.029$ (95% confidence interval).

(*c*) *Mumps*

Finally, the case returns for mumps are shown in figure 3*a*. There are 59 epidemic events with mumps present in 24% of the recorded months; again a good example of Type III dynamics. Mumps, whose aetiological agent is also a virus, is a less contagious disease than either measles or whooping cough, so much closer contact between a susceptible and an infective is required for transmission to occur. Consequently, mumps epidemics tend to be correspondingly smaller than the measles or whooping cough outbreaks.

The epidemic distribution function is shown in figure 3*b*. Again, there appears to be good scaling of the epidemic distribution though the gradient is different. We find that $\tau = 2.447 \pm 0.056$ (95% confidence interval).

We have attempted to fit other forms of distribution to these data, such as exponential (variance equals the mean) or negative binomial (variance is greater than mean), but these do not provide as good a fit as the power-law distribution (Rhodes & Anderson 1996*b*). We also investigated whether power-law distributions in epidemic sizes and durations can arise from conventional SEIR models (Schenzle 1984; Olsen *et al*. 1988; Olsen & Schaffer 1990; Anderson & May 1991; Grenfell 1992; Bolker & Grenfell 1993) which have, hitherto, been used successfully to account for Type I dynamics. Such models are based on the following dynamical equations for the number of susceptibles (S), exposed (E), infective (I) and recovered (R) individuals,

$$dS/dt = \mu\mathcal{N} - \mu S - \beta SI, \tag{7}$$

$$dE/dt = \beta SI - \mu E - \gamma E, \tag{8}$$

$$dI/dt = \gamma E - \mu I - \delta I + v, \tag{9}$$

$$dR/dt = \delta I - \mu R. \tag{10}$$

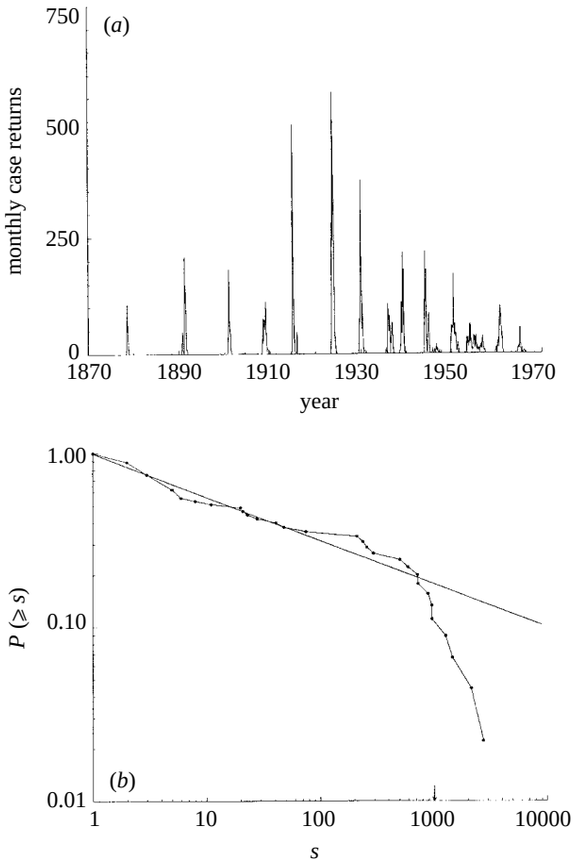

Figure 2. (*a*) The Faroe Island monthly whooping cough case returns for the years 1881–1969 inclusive. (*b*) Epidemic size probability distribution for the Faroe Island whooping cough data (log–log plot). The best-fit line shown is calculated so as only to apply to epidemics of up to 1000 cases.

The total population $S + E + I + R = \mathcal{N}$. The average life span of individuals is given by μ^{-1}, β is the contact rate between susceptibles and infectives (which can be age-structured or seasonal), γ^{-1} is the average incubation period and δ^{-1} is the average infectious period. The term v is a small immigation factor representing the occasional introduction of infectives into the system from outside.

Monte-Carlo simulations using these equations, with and without seasonal contact rates and age-structure, were unable to reproduce the power-law distribution. This is probably due to a breakdown in the mass-action (βSI) assumption that is central to the SEIR formulation because the populations we are dealing with are quite small.

A common feature of the scaling graphs for each of the diseases is a tail-off for the larger epidemics. This could happen because for the largest epidemics the dynamics are fundamentally different from those of the smaller epidemics, leading to a breakdown of scaling. Another possibility is that due to the infrequent occurrence of the very largest epidemics, the time-series we are using is not long enough to sample a representative number of larger epidemics. It is not possible, at present, to distinguish between these two hypotheses.

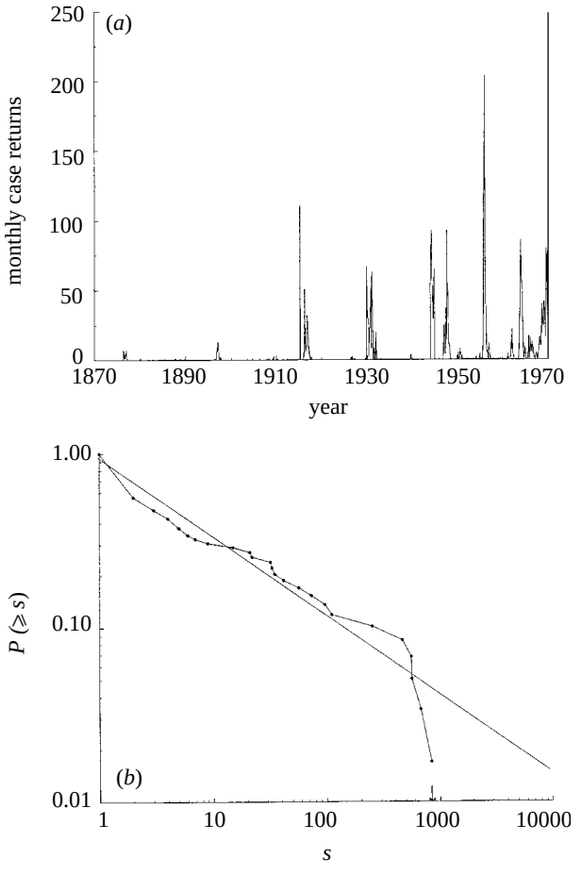

Figure 3. (*a*) The Faroe Island monthly mumps case returns for the years 1881–1969 inclusive. (*b*) Epidemic size probability distribution for the Faroe Island mumps data (log–log plot). The best-fit line shown is calculated so as only to apply to epidemics of up to 850 cases.

4. SCALING EXPONENTS AND DIMENSIONALITY

Above we have shown that for three common communicable diseases in the Faroe Islands there is strong evidence for scaling dynamics in the recorded epidemiological data. As we were motivated by the forest-fire model it is interesting to compare the exponents obtained from the data with the exponents from the model.

In figure 4 we show the modulus of the exponents for the three different diseases, with 95% error bars. Measles and whooping cough have practically identical exponents, whereas the exponent for the less transmissible mumps infection is somewhat larger (i.e. more negative). Also shown are the calculated exponents (with errors) from forest-fire simulations (Clar *et al.* 1994).

We propose that the epidemiological scaling results can be explained by the model simulations in three and five dimensions. Interpreting the space of the lattice as possibly representing social connections and interactions between individuals, rather than corresponding directly to geographical space, it would appear that the effective dimensionality of this space is dependent on the transmissibility of the disease. Specifically, the lower the transmissibility of the

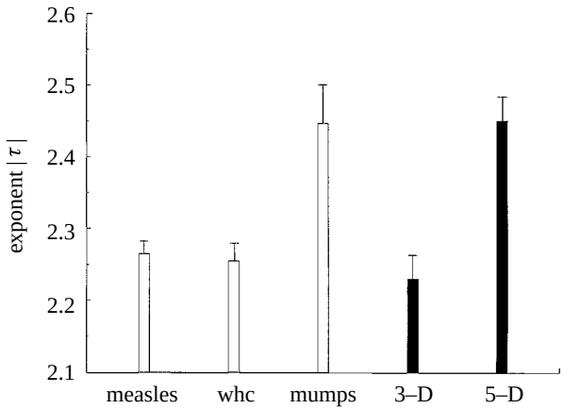

Figure 4. A plot of the absolute values of the scaling exponents $|\tau|$ for the three communicable diseases investigated in the Faroe Island data set (clear bars). Also shown (solid black bars) are the exponents derived from lattice-based simulations in three dimensions and in five dimensions. The error bars give the 95% confidence interval.

disease the higher the dimension of the social interaction space. However, increasing the dimensionality of the lattice lowers the number of available susceptibles that can be infected rather than modelling a less transmissible disease. Hence, it is the effective reproductive rate that is being reduced by this process. Given the good agreement between the model and the data, how this might relate to the specific epidemiology of diseases on the islands remains to be fully understood.

In previous work we suggested that the two-dimensional simulation provides a good explanation for the measles scaling (Rhodes & Anderson 1996*a*,*b*), but it turns out that the three-dimensional simulation provides closer quantitative agreement, a fact not realised when the original analysis was done. Also, we must stress that the above analysis, in terms of lattices of different dimensionality, whilst being a useful method of classifying the different exponents, need not represent the only possible approach to explaining the emergence of scaling in epidemiological systems. Other lattice-based models (which incorporate host movement) also yield scaling exponents for the distribution of fluctuations of epidemic sizes and durations (Rhodes & Anderson 1996*c*). Interestingly, the exponents found in this class of models are, broadly, of similar magnitude to the ones found in the forest-fire models. Work is ongoing to determine whether these models can be used to provide a more general framework in which the specific values of the exponents are dependent on parameters that can be more directly interpreted as being related to the transmissibility of the infection when a susceptible is adjacent to an infective. Clearly, a general epidemiological model that can reproduce the scaling relations (and the associated exponents) which uses a variable parameter that could be more intuitively associated with R_0 would be welcomed.

From these results we can make some general inferences. Whenever scaling dynamics occurs in Type III epidemics the size distribution exponent, τ, will take on a value reflecting the reproductive rate of the disease in that social context. Other diseases in the

same context, will have a higher (less negative) or lower (more negative) gradient depending upon whether they are more or less transmissible. Thus, in a different social context (for example, in a small isolated community on continental mainland) measles may well have a more negative gradient than that observed in the Faroes because its basic reproductive rate is lower. In effect τ can tell us about the *relative* transmissibilities (or basic reproductive rates) of diseases in different situations. Here the situation is quite clear-cut, but performing estimations of transmissibility are usually difficult when highly intermittent Type III dynamics are taking place.

5. DISCUSSION

We have discussed the use of lattice-based models and the application of ideas from a statistical mechanical context to describe the dynamics of epidemics of communicable disease. Our results on a scaling analysis of some real medical case returns for an island population suggests that an interpretation of the epidemiological data in terms of power-law distributions is plausible. It places the dynamics of Type III epidemics in the same class of phenomena as, for example, earthquakes and may well be an example of an SOC phenomenon. Over the range of validity of the scaling relationship the same mechanism that governs small epidemics governs big ones, and the frequency of small epidemics tells us the expected frequency of the big epidemics. Epidemic events can therefore happen on all scales from one case up to the whole island being affected. Interestingly, in 1846, practically the entire population of the Faroes was struck with measles. These results suggest that there is real dynamical structure in data sets which were thought to be largely dominated by stochastic effects. Our conclusions are drawn from a study of three diseases from one of the most accurate and long-running epidemiological data sets known, and we acknowledge that by the criteria usually employed in simulations of critical phenomena the data sets are short. Despite this we believe that our interpretation in terms of scaling is a sound one.

We expect the forest-fire simulation to be able to represent epidemiological dynamics because the model can be interpreted quite naturally as a simple SI model. Near a critical point the specific epidemiological details that distinguish different diseases become irrelevant and the dynamics will be dominated by nearest-neighbour spread in some social interaction space of arbitrary dimension. Attempts to reproduce the observed scaling dynamics failed when a conventional SEIR model was used. This is probably connected with the breakdown of mass-action mixing assumption for such small populations. The analysis proposed in this study is completely general and can be carried out on any data set that might exhibit Type III dynamics, particularly when there is a clear separation of time-scales for production of susceptibles and the arrival of index cases from outside the community. One of the strengths of this approach is that it uses every single individual recorded disease case, rather than ignoring small epidemics and trying to account for only the larger disease outbreaks. In future, we anticipate that a scaling approach will be useful in understanding the occasional outbreaks of communicable disease that occur in otherwise highly vaccinated populations. For instance, in urban centres in the United Kingdom, despite major national initiatives on immunization, pockets of susceptibles to measles infection still build up due to lack of understanding of basic healthcare information, resulting in ignorance of the possibility of vaccination, as well as due to vaccine failure. In this situation the arrival of an index case then usually triggers an epidemic. Current mass-action based models do not work so well in this post-mass-vaccination regime, and the scaling analysis appears to provide a useful insight (Rhodes *et al*. 1997).

Finally, we believe that this study illustrates how ideas developed to understand critical phenomena in the physical sciences can be used to gain insight into the dynamics of real epidemic diseases. It illustrates that biological systems can also exhibit critical phenomena, that the conditions for critical behaviour to occur can arise quite naturally and that simple models which capture the essential interactions can explain the dynamical behaviour of a number of different diseases in a human population.

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REFERENCES

Anderson, R. M. & May, R. M. 1991 *Infectious diseases of humans, dynamics and control*. Oxford University Press.

Bailey, N. J. T. 1965 The simulation of stochastic epidemics in two dimensions. *Proc. 5th Berkeley Symp. on Math. Statist. and Prob*. **4**, 237–257.

Bailey, N. J. T. 1975 *The mathematical theory of infectious diseases and its applications*. Oxford University Press.

Bak, P., Chen, K. & Tang, C. 1990 A forest-fire model and some thoughts on turbulence. *Phys. Lett.* A **147**, 297–300.

Bak, P. & Sneppen, K. 1993 Punctuated equilibrium and criticality in a simple model of evolution. *Phys. Rev. Lett.* **71**, 4083–4086.

Bak, P., Tang, C. & Weisenfeld, K. 1988 Self-organised criticality. *Phys. Rev.* A **38**, 364–374.

Boccara, N. & Cheong, K. 1992 Automata network models for the spread of infectious diseases in a population of moving individuals *J. Phys. A: Math. Gen.* **25**, 2447–2461.

Boccara, N. & Cheong, K. 1993 Critical behaviour of a probabilistic automata network SIS model for the spread of an infectious disease in a population of moving individuals. *J. Phys. A: Math. Gen.* **26**, 3707–3717.

Bolker, B. M. & Grenfell, B. T. 1993 Chaos and biological complexity in measles dynamics. *Proc. R. Soc. Lond.* B **251**, 75–81.

Cardy, J. 1983 Field theoretic formulation of an epidemic process with immunisation. *J. Phys. A: Math. Gen.* **16**, L709–712.

Cardy, J. & Grassberger, P. 1985 Epidemic models and percolation. *J. Phys. A: Math. Gen.* **18**, L267–271.

Chen, K., Bak, P. & Obukhov, S. P. 1991 Self-organised criticality in a crack-propagation model of earthquakes. *Phys. Rev.* A **43**, 620–635.

Christensen, K., Flyvberg, H. & Olami, Z. 1993 Self-organised critical forest-fire model: mean-field theory and simulation results in 1 to 6 dimensions. *Phys. Rev. Lett.* **71**, 2737–2740.

Clar, S., Drossel, B. & Scwabl, F. 1994 Scaling laws and simulation results for the self-organised critical forest-fire model. *Phys. Rev.* E **50**, 1009–1018.

Clar, S., Drossel, B. & Schwabl, F. 1996 Forest fires and other examples of self-organised criticality. *J. Phys.: Condens. Matter* **8**, 6803–6824.

Cliff, A., Haggett, P. & Smallman-Raynor, M. 1993 *Measles. An historical geography of a major human viral disease from global expansion to local retreat 1840–1990.* Oxford: Blackwell.

Cox, J. T. & Durrett, R. 1988 Limit theorems for the spread of epidemics and forest fires. *Stoch. Process. Applics* **30**, 171–191.

De Boer, J., Derrida, B., Flyvberg, H., Jackson, A. D. & Wettig, T. 1994 Simple model of self-organised biological evolution. *Phys. Rev. Lett.* **78**, 906–909.

Drossel, B. & Schwabl, F. 1992 Self-organised critical forest-fire model. *Phys. Rev. Lett.* **69**, 1629–1632.

Drossel, B. & Schwabl, F. 1993 Forest-fire model with immune trees. *Physica* A **199**, 183–197.

Drossel, B. & Schwabl, F. 1994 Formation of space–time structure in a forest-fire model. *Physica* A **204**, 212–229.

Durrett, R. 1995 Spatial epidemic models. In *Epidemic models: their structure and relation to data.* (ed. D. Mollison), pp. 187–201. Publications of the Newton Institute, Cambridge University Press.

Durrett, R. & Cox, J. T. 1988 Limit theorems for the spread of epidemics of forest fires. *Stoch. Proc. Appl.* **30**, 171–191.

Durrett, R. & Levin, S. A. 1994*a* The importance of being discrete (and spatial). *Theor. Popul. Biol.* **46**, 363–394.

Durrett, R. & Levin, S. A. 1994*b* Stochastic spatial models: a user's guide to ecological applications. *Phil. Trans. R. Soc. Lond.* B **343**, 329–350.

Durrett, R. & Neuhauser, C. 1991 Epidemics with recovery in $d = 2$. *Ann. Appl. Prob.* **1**, 189–206.

Flyvberg, H. 1996 Simplest possible self-organised critical system. *Phys. Rev. Lett.* **76**, 940–943.

Frette, V., Christensen, K., Malthe-Sorenssen A., Feder, J., Jossang, T. & Meakin, P. 1996 Avalanche dynamics in a pile of rice. *Nature* **379**, 49–52.

Grassberger, P. 1983 On the critical behaviour of the general epidemic process and dynamic percolation. *Math. Biosc.* **63**, 157–172.

Grassberger, P. 1993 On a self-organised critical forest-fire model. *J. Phys. A: Math. Gen.* **26**, 2081–2089.

Grassberger, P. & Kantz, H. 1991 On a forest-fire model with supposed self-organised criticality. *J. Stat. Phys.* **63**, 685–700.

Grenfell, B. T. 1992 Chance and chaos in measles dynamics. *Jl R. Statist. Soc.* B **54**, 383–398.

Hassell, M. P., Comins, H. N. & May, R. M. 1991 Spatial structure and chaos in insect population dynamics. *Nature* **353**, 255–258.

Held, G. A., Solina, D. H., Keane, D. T., Haag, W. J., Horn, P. M., Grinstein, G. 1990 Experimental study of critical mass fluctuations in an evolving sandpile. *Phys. Rev. Lett.* **65**, 1120–1123.

Henley, C. L. 1993 Statics of a self-organised percolation model. *Phys. Rev. Lett.* **71**, 2741–2744.

Johansen, A. 1994 Spatio-temporal self-organisation in a model of disease spreading. *Physica* D **78**, 186–193.

Johansen, A. 1996 A simple model of recurrent epidemics *J. Theor. Biol.* **178**, 45–51.

Loreto, V., Pietronero, L., Vespignani, A. & Zapperi, S. 1995 Renormalisation-group approach to the critical behaviour of the forest-fire model *Phys. Rev. Lett.* **75**, 465–468.

Mollison, D. 1977 Spatial contact models for ecological and epidemic spread. *Jl R. Statist. Soc.* B **39**, 283–326.

Mollison, D. & Kuulasmaa, K. 1985 Spatial endemic models: theory and simulations. In *The Population dynamics of rabies in wildlife.* (ed. P. J. Bacon), pp. 292–309. New York: Academic Press.

Mossner, W. K., Drossel, B. & Schwabl, F. 1992 Computer simulations of the forest-fire model. *Physica* A **190**, 205–217.

Newman, M. E. J. & Roberts, B. W. 1995 Mass extinction: evolution and the effects of external influences on unfit species. *Proc. R. Soc. Lond.* B **260**, 31–37.

Olami, Z., Feder, H. J. S. & Christensen, K. 1992 Self-organised criticality in a continuous non-conservative cellular automata modelling earthquakes. *Phys. Rev. Lett.* **68**, 1244–1247.

Olsen, L. F., Truty, G. L. & Schaffer, W. M. 1988 Oscillations and chaos in epidemics: a nonlinear dynamic study of six childhood diseases in Copenhagen, Denmark. *Theor. Popul. Biol.* **33**, 344–370.

Olsen, L. F. & Schaffer, W. M. 1990 Chaos versus noisy periodicity: alternative hypotheses for childhood epidemics. *Science* **249**, 499–504.

Rand, D. A., Keeling, M. & Wilson, H. B. 1995 Invasion stability and evolution to criticality in spatially extended, artificial host–pathogen ecologies. *Proc. R. Soc. Lond.* B **259**, 55–63.

Rhodes, C. J. & Anderson, R. M. 1996*a* Power laws governing epidemics in isolated populations. *Nature* **381**, 600–602.

Rhodes, C. J. & Anderson, R. M. 1996*b* A scaling analysis of measles epidemics in a small population. *Phil. Trans. R. Soc. Lond.* B **351**, 1679–1688.

Rhodes, C. J. & Anderson, R. M. 1996*c* Persistence and dynamics in lattice models of epidemic spread. *J. Theor. Biol.* **180**, 125–133.

Rhodes, C. J. & Anderson, R. M. 1996*d* Dynamics in a lattice epidemic model. *Phys. Lett.* A **210**, 183–188.

Rhodes, C. J., Butler, A. R. & Anderson, R. M. 1997 Epidemiology of communicable disease in small populations. *J. Molec. Med.* (In the press).

Roberts, B. W. & Newman, M. E. J. 1996 A model for evolution and extinction. *J. Theor. Biol.* **180**, 39–54.

Rohani, P. & Miramontes, O. 1995 Host–parasitoid metapopulations: the consequences of parasitoid aggregation on spatial dynamics and searching efficiency. *Proc. R. Soc. Lond.* B **260**, 335–342.

Schenzle, D. 1984 An age-structured model of pre and post vaccination measles transmission. *IMA J. Math. Appl. Med. Biol.* **1**, 169–191.

Sneppen, K. 1995 Extremal dynamics and punctuated coevolution. *Physica* A **221** 168–179.

Sneppen, K., Bak, P., Flyvberg, H. & Jensen, M. H. 1995 Evolution as a self-organised critical phenomenon. *Proc. Natl. Acad. Sci. USA* **92**, 5209–5213.

Solé, R. V. & Bascompte, J. 1996 Are critical phenomena relevant to large-scale evolution? *Proc. R. Soc. Lond.* B **263**, 161–168.

Solé, R. V. & Manrubia, S. C. 1995 Are rainforests self-organised in a critical state? *J. Theor. Biol.* **173**, 31–40.

Vespignani, A., Zapperi, S. & Loreto, V. 1996 Renormalisation of non-equilibrium systems with critical stationary states. *Phys. Rev. Lett.* **77**, 4560–4563.

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