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Stochastic dynamics and a power law for measles variability

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Since the discovery of a power law scaling between the mean and variance of natural populations, this phenomenon has been observed for a variety of species. Here, we show that the same form of power law scaling also occurs in measles case reports in England and Wales. Remarkably this power law holds over four orders of magnitude. We consider how the natural experiment of vaccination affects the slope of the power law. By examining simple generic models, we are able to predict the effects of stochasticity and coupling and we propose a new phenomenon associated with the critical community size.

Keywords: power law; measles; vaccination; stochasticity; critical community size

1. INTRODUCTION

The last two decades have seen a major synthesis of ideas from epidemiology and ecology (Anderson & May 1979; May & Anderson 1979; Grenfell & Dobson 1995; Earn *et al.* 1998). Host–parasite interactions have proved to be some of the best systems for the study of ecological dynamics, including predator–prey dynamics and natural enemy interactions in single communities (Dobson 1985; Grenfell 1988, 1992; Anderson & May 1992), questions of persistence (Keeling 1997; Keeling & Grenfell 1997), metapopulation dynamics (Nee & May 1992; Grenfell & Harwood 1997) and nonlinear behaviour (Olsen & Schaffer 1990; Rand & Wilson 1991).

One area of epidemiology which has been relatively unexplored is the basic relationship between abundance and variability. The ecological paradigm here is Taylor's power law (Taylor 1961; Taylor *et al.* 1978; Taylor & Woivod 1980; Anderson *et al.* 1982; Hanski & Tiainen 1989; Boag *et al.* 1992). Taylor *et al.* (1978) proposed a power law relationship between the mean M and variance V of natural populations: $V \propto M^\alpha$. This was originally interpreted by invoking density-dependent behaviour of the organisms, leading to differing levels of aggregation (Taylor *et al.* 1978). However, Anderson *et al.* (1982) pointed out that simple birth–death processes could also produce power law behaviour.

In order to explore variability–abundance relationships, we ideally require a system with a large quantity of spatio-temporal data and plausible models. As with many other theoretical studies in ecology and epidemiology we propose that the dynamics of measles in developed countries is highly suitable for exploring this issue.

- (i) Data availability—in the UK measles is a notifiable disease, which has led to a highly detailed spatio-temporal data set of reported cases. Approximately 60% of all cases are reported and this proportion remains roughly constant, both over time and

between communities. The available data span a large range of urban community sizes, from populations of just over 1000 to London with a population of several million. This range of community sizes means that a large variety of behaviour is observed although all the time-series possess strong biennial components.

- (ii) Models—the majority of compartmental models for measles are of the SEIR type, classifying individuals as susceptible (S), exposed (E), infectious (I) or recovered (R) from the disease. The latest model of this type is the stochastic, pulsed realistic age-structured (PRAS) model (Keeling & Grenfell 1997) which accurately captures the observed qualitative dynamics (Finkenstädt *et al.* 1998) and persistence of infection.

In this paper, we explore the temporal (as opposed to spatial) variability in abundance of measles in communities in England and Wales. In the next section we consider the observed data together with suitable epidemiological models, using different historical levels of vaccination as a natural experiment. In order to interpret the power law of the mean–variance relationship, we propose a simple birth–death model; this allows us to analyse the effects of stochasticity, coupling and community size.

2. THE POWER LAW BEHAVIOUR OF MEASLES

We examine the power law relationship between the temporal mean and variance, looking for changes in the exponent (α) as the level of vaccination varies. This is most easily achieved by measuring the least-squares gradient of $\log(\text{variance})-\log(\text{mean})$ plots; power law behaviour appears as a linear relationship between these two quantities.

(a) Case reports

Figure 1a shows the mean–variance relationship for measles case reports, each point representing the mean

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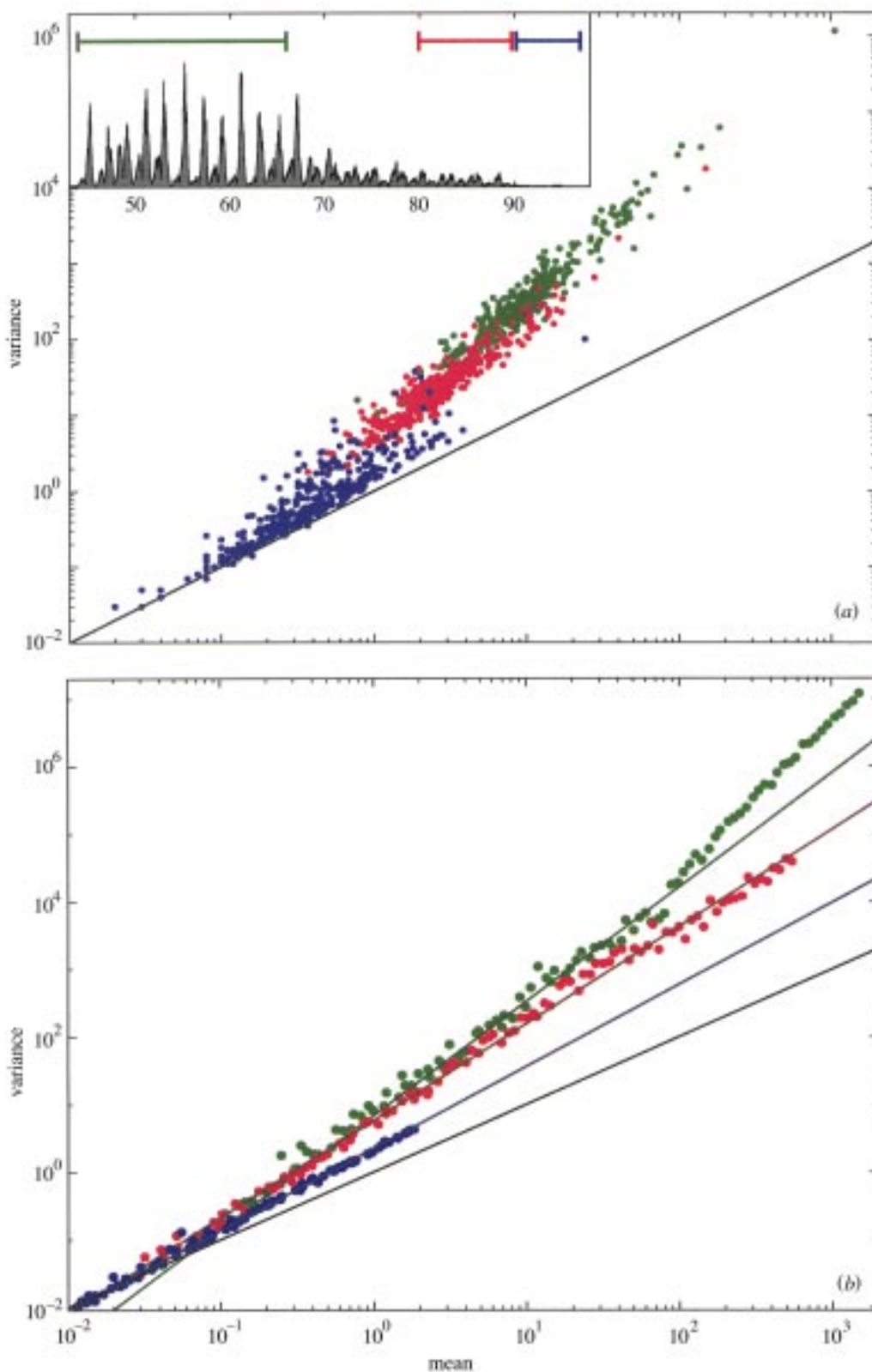


Figure 1. (a) Mean–variance plots for the case reports of measles in 366 communities in England and Wales. The data are subdivided between the pre-vaccination era (1944–1966, green dots), the 80% vaccination era (1980–1990, red dots) and the 90% vaccination era (1990–1997, blue dots); data from 1968–1980, when the vaccination rate was 60%, shows the same power law scaling as the pre-vaccination era. The solid black line is the expected results for Poisson distributions where $V = M$. The inset graph gives the total number of reported cases in England and Wales, with the bars indicating the three vaccination eras. (b) Mean–variance plots from 250 years of the PRAS model (Keeling & Grenfell 1997) for the three regimes of 0, 80 and 90% vaccination. The solid lines show the best power law fit to the England and Wales data—there is clear agreement between the model and observations. For the model results in the pre-vaccination era, there is a change in the power law gradient for large populations ($M > 70$). The black line is the Poisson distribution.

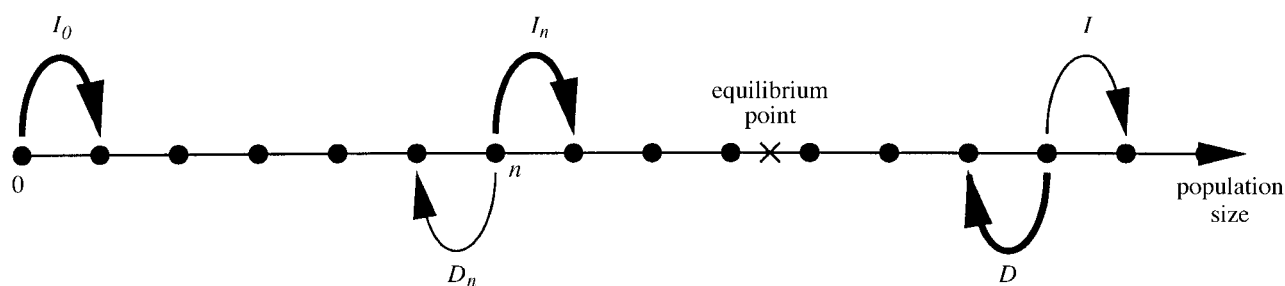


Figure 2. The stochastic dynamics of a population can be expressed as a Markov chain, with the probabilistic rates of increase and decrease being I_n and D_n respectively (see text). Below the equilibrium point of the underlying dynamics we expect I_n to dominate, while above the equilibrium point D_n should be larger; this leads to a stable distribution of population sizes P_n .

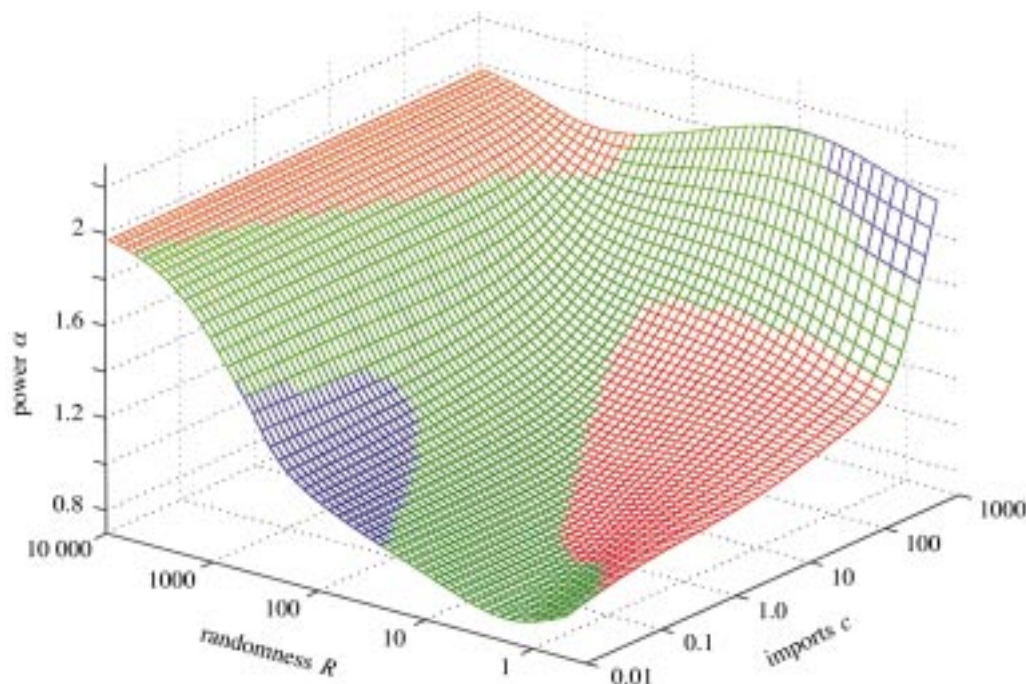


Figure 3. Below the critical point the power law scaling α is, in general, an increasing function of both the randomness R and the import rate c . As the amount of stochasticity increases the power law asymptotes to 2. α was calculated as the gradient of a least-squares fit to the $\log(\text{mean})$ versus $\log(\text{variance})$ relationship, over the values of K when extinctions were common. Notice that power law gradients outside the range 1–2 are associated with extreme import rates. The regions of the graph are colour coded to show how much of the variance is explained by the power law; red corresponds to greater than 99.9%, green to between 99 and 99.9% and blue to less than 99%.

and variance from one community. The data are taken from the *Registrar General's Weekly Reports* and give the number of reported cases of measles each week in 366 communities in England and Wales. The data has been partitioned into four eras approximately corresponding to different levels of vaccinations. If the number of reported cases in community i during week w is C_w^i , then the mean and variance are defined as:

$$M_i = \frac{1}{W} \sum_{w=1}^W C_w^i$$

and

$$V_i = \frac{1}{W} \sum_{w=1}^W (C_w^i - M_i)^2.$$

The data from 1944–1968 show a power law scaling with a gradient of *ca.* 1.7 holding from populations of just over 1000 to large cities. As well as reducing the total number of cases, vaccination also decreases the slope of the power law (figure 1a) bringing the system closer to a Poisson distribution ($V = M$). During 1968–1980, when the vaccination coverage was *ca.* 60%, there is little or no change in the mean–variance relationship from the pre-vaccination case: it is interesting to note that during this period (1944–1980) the critical community size also remained constant (Bolker & Grenfell 1996; Keeling 1997). In later eras, vaccination levels reached as high as 80 and 90% and the associated power law gradient, α , was reduced to 1.5 and 1.2, respectively (figure 1a). Associated with the increase in vaccination is an increase in the residuals about the power law, this may in part be due to heterogeneity in vaccination coverage.

Not all the cases of measles in a given community are recorded. If we assume that reporting a case is an independent random event with probability r , then this will lead to a binomial distribution. The recorded mean and variance (M and V) will then be related to the actual mean and variance (\hat{M} and \hat{V}) as follows:

$$M = r\hat{M}$$

and

$$V = r^2\hat{V} + r(1-r)\hat{M}.$$

However, as the variance is usually much greater than the mean and $r \sim 0.6$ (Finkenstädt & Grenfell 1998) there should be little change in the power law gradient α , due to underreporting.

We note that, for all three vaccination eras, the intercept between a Poisson distribution ($V = M$) and the mean–variance power law (once the data has been corrected for underreporting, i.e. using \hat{M} and \hat{V}) occurs at a population size of one. Therefore, only isolated individuals experience a Poisson-distributed force of infection; the aggregation of individuals in a community causes aggregated patterns of infection.

(b) *A comparison with epidemiological models*

Age-structured models of measles, seasonally forced by school-term structure, have been highly successful (Schenzle 1984; Bolker 1993); in particular, these models have been shown to capture the stochastic dynamics of the disease accurately (Ferguson *et al.* 1996; Keeling & Grenfell 1997; Finkenstädt & Grenfell 1998; Finkenstädt *et al.* 1998). The comparison between models and reported cases relies on the assumption that there are minimal changes in population size or other parameters for the observed data. Small changes in population size or birth rate will lead to a slight increase in the power law slope α (Keeling & Grenfell 1999). However, by partitioning the observed data into biennial cycles we can reduce the amount of social change in each sample; although these data are slightly noisier, they display the same power law gradient.

Figure 1*b* is the mean–variance relationship for the PRAS model corresponding to the observed data. We again witness a decrease in the power law slope with increasing vaccination, as well as an overall decrease in the mean. The solid lines in figure 1*b* are the best fit power laws to the England and Wales data, showing that there is good agreement between the reported cases and model predictions. As with all stochastic disease models external imports of infection are necessary to ensure the persistence of the disease. The number of imports used is that given in Keeling & Grenfell (1999) and, although changing the import rate has a slight multiplicative effect on the variance, it does not noticeably change the power law.

The data from the PRAS model is a far tighter fit to the power law curve than observed in real populations. Even for very short time-series (using just a two-year sample) the PRAS model still shows far less variation. This leads us to conclude that the unexplained residuals in the observed cases are primarily due to external effects such as differences in birth rate or different levels of infection imports in each community.

Although such complex models can reproduce the observed scaling, it is important to consider what can be inferred about the disease dynamics from α , the gradient of the power law.

3. INTERPRETING THE POWER LAW

Anderson *et al.* (1982) conclude, that the power law scalings observed for the majority of ecological time-series generally lie between 1 and 2. This agrees with results from a variety of one- and two-species models (Keeling & Grenfell 1999). When $\alpha = 1$ the population behaves as if it were composed of many independent elements (cf. Keeling *et al.* 1997) or that all events are independent leading to a Poisson distribution. Therefore, large amounts of heterogeneity or weak spatial interaction will reduce α toward unity.

When $\alpha = 2$, the individual nature of the population is unimportant, as the standard deviation scales linearly with the mean. This limit is achieved when the deterministic dynamics swamp the stochastic noise and the system is homogeneous, but can also be produced when the stochastic noise is very large relative to the dynamics. We can see the observed power law as scaling between these two extremes, from local independence to global determinism.

The stochastic SEIR model and other compartmental disease models are more biologically realistic and, therefore, more complex versions of simple birth–death models. However, interpretation of the results from these disease models is complicated, so simpler, more tractable models need to be considered.

(a) *A demographic model*

To understand the origin of power law scaling and the meaning of the power observed, we shall consider a single-species stochastic model. Although the methodology given will hold for any such system, we shall concentrate on the stochastic logistic equation with imports as the simplest model which displays the full range of stochastic behaviour.

Given a population of n individuals, we shall assume that the rates at which the population increases or decreases by 1 are I_n and D_n respectively. The underlying differential equation for the population would generally be taken as

$$\frac{dn}{dt} = I_n - D_n = r \left(c + n - \frac{n^2}{K} \right),$$

where the right-hand side is the logistic equation with imports, K being the carrying capacity and c the import rate. The parameter r acts as a temporal scaling, so without loss of generality we can take $r = 1$. We assume that I and D have the following forms (figure 2):

$$I_n = c + (R + 1)n \quad D_n = Rn + \frac{n^2}{K}.$$

This formulation for stochastic changes to the population gives us a Markov chain—the probability of moving to a new population level is only dependent on the current population level. Notice that both the rates contain a term R_n which cancels; we can therefore use the

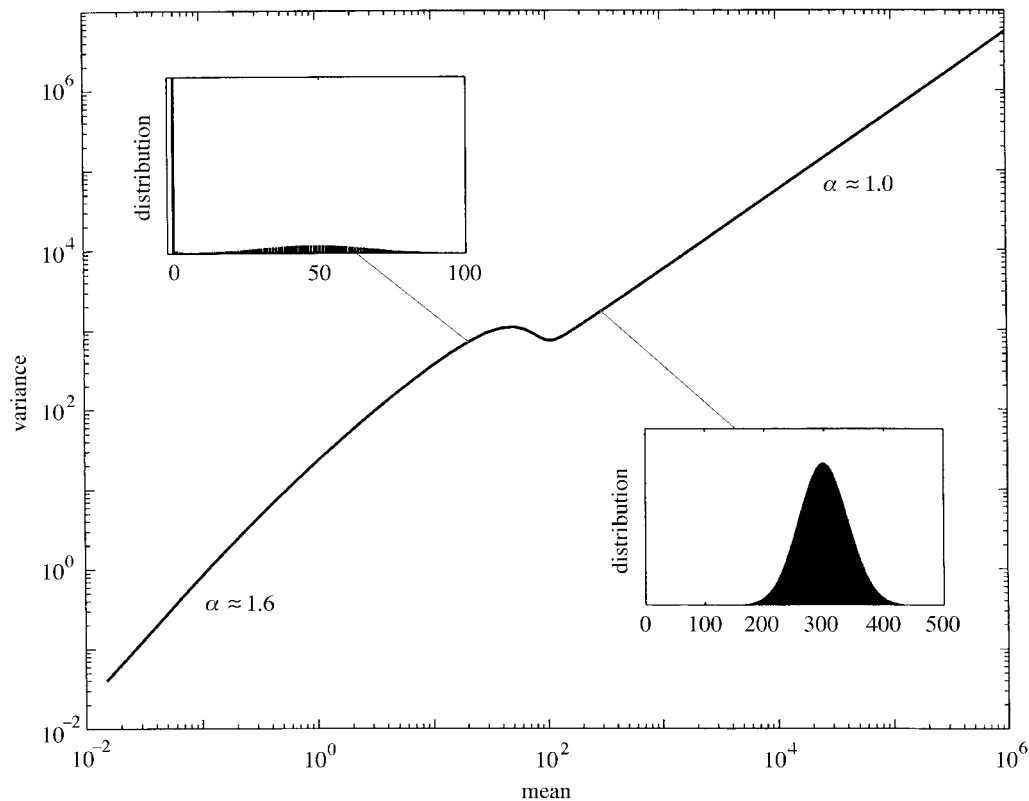


Figure 4. The typical mean–variance behaviour for the logistic equation with imports; $R = 5$ and $c = 0.025$. There is a clear decrease in the variance at the critical point which occurs when the mean is around 70. Below the critical point the gradient is $\alpha \sim 1.6$, whereas above the critical point $\alpha \sim 1.00$. The long-term distribution of population sizes P_n is shown either side of the critical point—there is a change from dominance of zeros to a monotonic distribution.

parameter R to increase the amount of stochasticity in the model without changing its deterministic behaviour.

The probability distribution of population sizes (P_n) can be found in terms of the rates of change:

$$\frac{dP_n}{dt} = I_{n-1}P_{n-1} - (I_n + D_n)P_n + D_{n+1}P_{n+1}.$$

From this infinite set of coupled differential equations we can find an expression for the equilibrium probability distribution of the system:

$$P_n = \prod_{m=1}^n q_m$$

where

$$q_m = \frac{I_{m-1}}{D_m}.$$

From the distribution P_n we can calculate numerically the expected mean and variance of the population. Note that in the absence of imports ($c = 0$) all populations are doomed to permanent extinction and $M = V = 0$.

As the carrying capacity K is varied, mean–variance plots for many one-species systems (with a variety of functional forms) demonstrate two distinct regions of power law scaling as discussed later. A more technical discussion of the behaviour for large populations is given in Keeling & Grenfell (1999), but, in essence, the power law frequently asymptotes to either 1 or 2. We shall initially concentrate on the behaviour for smaller commu-

nities as this most closely matches the observed pattern for measles in England and Wales.

For smaller populations the slope α is found to increase with the randomness R asymptoting to 2 as R becomes large (figure 3). In agreement with results from the PRAS model, we find that, for biologically realistic values, changes in the import rate c are associated with a multiplicative change in the variance but little change in the power law. The decrease in α associated with higher levels of vaccination can therefore be compared to R tending to zero in the logistic model—the system then becomes closer to a Poisson process.

4. THE CRITICAL COMMUNITY SIZE AND A CHANGE IN THE POWER LAW

Figure 4 shows the mean–variance behaviour for the stochastic logistic equation; two distinct regions of power law scaling are observable. When the mean is small, the distribution of population sizes contains many zeros and $\alpha \sim 1.6$, whereas when the mean is large the distribution is closer to a Gaussian and $\alpha \sim 1$ (see inset graphs in figure 4). The critical point, where the distribution changes from having many to few zero states, can be compared to the critical community size for a disease (Bartlett 1957; Keeling 1997) and for many parameter values is associated with a jump in the variance. This jump in variance appears to be due to the distribution changing from bimodal, with many zeros, to unimodal with few zeros. As expected, this critical point increases with the randomness R , but decreases with the import

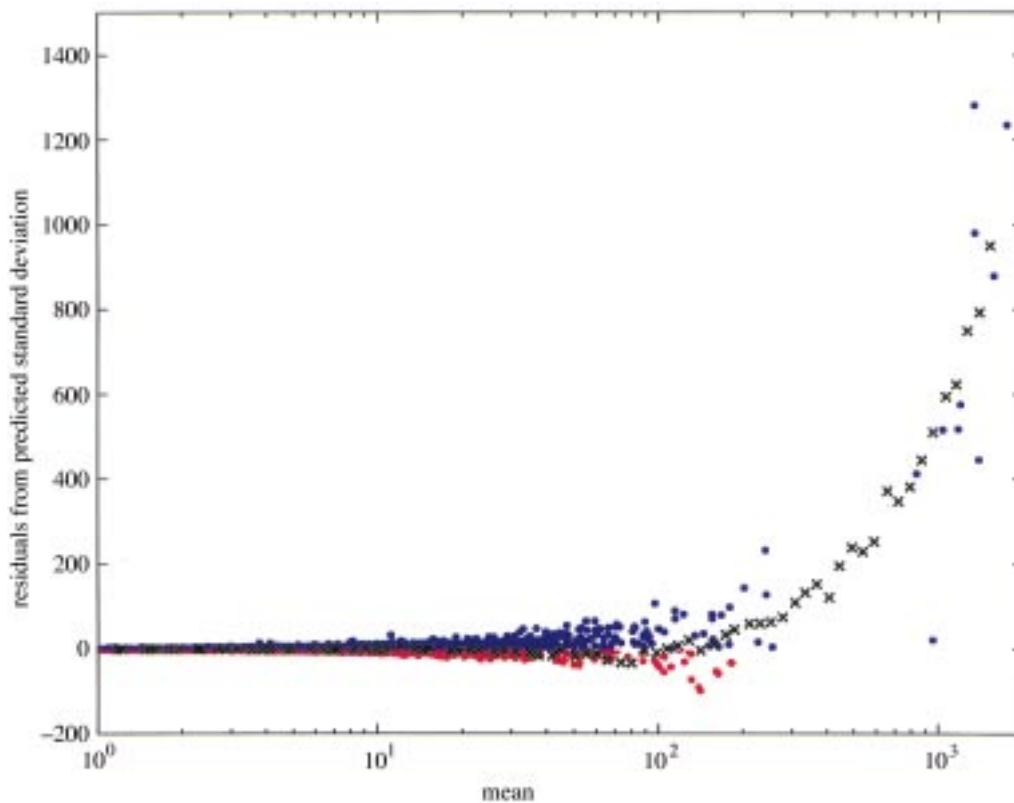


Figure 5. For each biennial epidemic cycle from 1944 to 1965, the dots represent the residuals from the standard deviation predicted by the power law for 845 communities in England and Wales. The dots are colour coded red or blue depending on whether the variance is less than or greater than predicted. By using biennial samples, rather than aggregating over the entire period, more data points are provided and effects due to changes in the birth rate should be reduced. Black crosses are for results from the PRAS model; this displays a clear trend for larger variances than predicted at large means.

rate c . As most communities suffer frequent fade-outs of measles, the power law behaviour below the critical point should most closely match that from the reported cases.

This change in power law is also visible in the pre-vaccination PRAS model (figure 1*b*) where, above the critical point of *ca.* 200 000, the power law scaling changes to $\alpha \sim 2$. Once again the critical point is associated with a transition from many to few zeros and a jump in the variance. The power of 2 scaling seen for large populations is due to the increasing dominance of the deterministic biennial dynamics—in this region stochasticity and the individual nature of the population have little effect.

Closer examination of the data from the pre-vaccination era (figure 5) shows the residuals from the power law scaling of $\alpha \sim 1.7$, while the change in gradient is obvious for the model results, there are too few populations above the critical point for this to be clear in the England and Wales data. However, we would argue that unless a power law of 2 eventually holds, then in large populations the annual or biennial nature of the epidemics is lost. When $\alpha < 2$, the relative magnitude of the non-stationary dynamics, as measured by the standard deviation to mean ratio ($\sqrt{V/M}$), decays with population size. In the absence of seasonal forcing, when the deterministic dynamics have a fixed point attractor, we find that about the critical community size the power law is 1, confirming our belief that it is the biennial dynamics that generate the power of 2.

5. DISCUSSION

Measles data from England and Wales has been shown to be consistent with Taylor's power law and this relationship is observed to hold over a huge range of population sizes. The natural experiment of vaccination leads to a decrease in the power law exponent with the distribution of cases becoming closer to Poisson. Epidemiological models are in close agreement with this behaviour, but show less variability.

The use of simple demographic models has shown that, in general, the power law α is an increasing function of the amount of stochasticity in the system, although power laws close to 2 can also be associated with the dominance of large-scale deterministic behaviour. As the level of vaccination increases, so the power law α decreases from *ca.* 1.7, when there is little or no vaccination, to *ca.* 1.2 when *ca.* 90% of the population are vaccinated. As the level of vaccination approaches the eradication threshold, we would expect the number of cases to behave like uncorrelated white noise and so $\alpha \rightarrow 1$. This decrease in α may be further enhanced by an increase in the level and scale of heterogeneities (Bolker & Grenfell 1996). Hence and somewhat surprisingly, there is more stochasticity (cf. large R -values) associated with the more regular dynamics of the disease before vaccination than the noisy behaviour at high vaccination levels.

The clear change in power law behaviour, for both the single species model and the PRAS simulation, gives us a

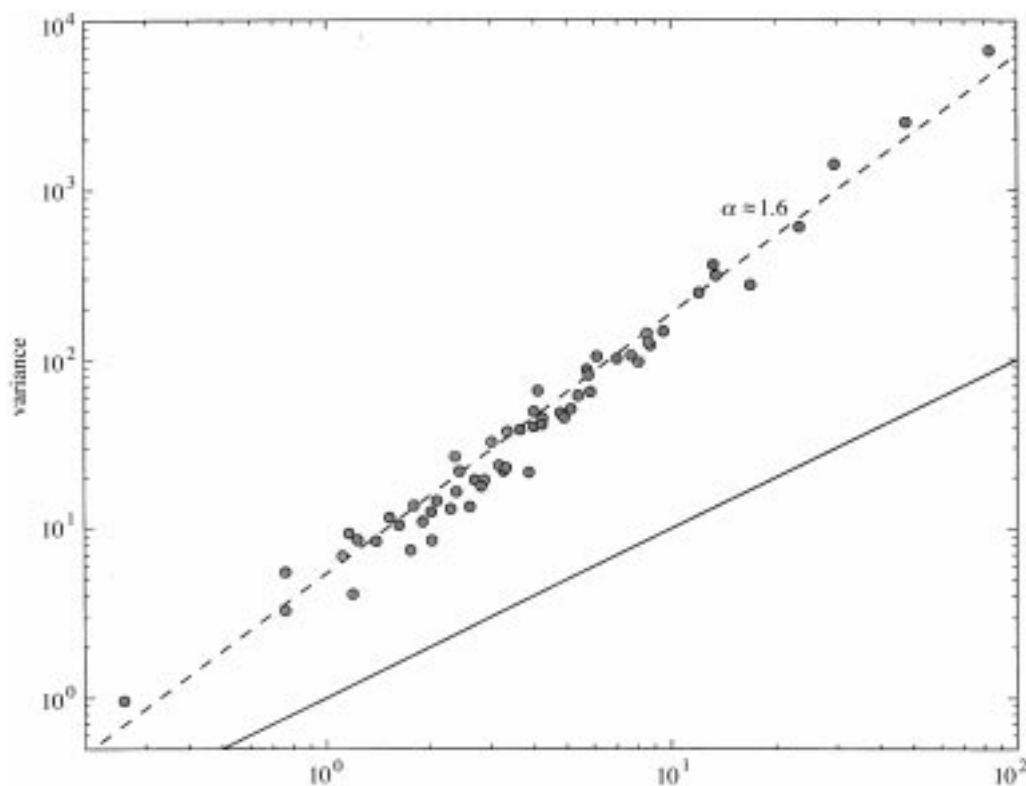


Figure 6. The mean–variance relationship for whooping cough, demonstrating a gradient of $\alpha \sim 1.56$. The data are weekly case reports for 60 towns and cities in England and Wales from 1946 to 1974. The solid line shows the Poisson distribution ($V = M$) for comparison.

new method of pinpointing the critical community size. Unfortunately there are too few data points around the critical point to determine its precise value, but, in principle, this gives us a more robust definition of the critical community size.

Obviously the data for measles are more complicated than simple demographic models, for example there is no fixed carrying capacity for the number of infectious cases; instead it is the level of susceptibles that acts to regulate their level (Finkenstädt *et al.* 1998). Other difficulties are the existence of biennial cycles in the measles data and the fact that the amount of stochasticity will vary around this cycle. Despite this, we believe that the analytical logistic equation allows us to interpret the more complex behaviour of measles.

Such clear power law relationships are not exclusive to measles dynamics; figure 6 shows the mean–variance behaviour of whooping cough for 60 cities in England and Wales. Again a clear power law scaling is observed, with $\alpha \sim 1.6$. Therefore, despite a far more complex natural history than measles, a more intermittent vaccination campaign and a lower rate of reporting (Rohani *et al.* 1998) whooping cough still conforms to the power law behaviour associated with the simple models.

We believe the analytical results we have discussed here are generic for many ecological systems. Similar mean–variance relationships are also obtained for a variety of functional responses in the one-species model, as well as for more complex two-species simulations. We therefore predict that two regions of power law scaling separated by a phase transition should be observed in many stochastic systems where a large range of means are observed.

This work further strengthens the analogies between ecological and epidemiological systems and highlights how epidemiological dynamics are often ideal for testing ecological theory.

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