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Persistence, chaos and synchrony in ecology and epidemiology

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The decline of species in natural habitats concerns ecologists, who view extinction as a danger and conservation of biological diversity as a goal. In contrast, the proliferation of 'undesirable' species is the principal concern of epidemiologists, who view persistence as a problem and eradication as an achievement. While ecologists and epidemiologists have essentially opposite goals, the mathematical structure of the population dynamics that they study is very similar. We briefly review the similarities and differences between these two fields, emphasizing recent work in both areas on the effects of spatial synchrony and dynamical chaos. We hope to stimulate further cross-fertilization of ideas between the disciplines.

Keywords: metapopulation, extinction, dispersal, eradication, conservation, biological control

1. INTRODUCTION

What enables some species to persist while others become extinct? This question has shaped the history of research on population dynamics and remains a central issue for ecologists and epidemiologists.

In a recent issue of this journal, Heino *et al*. (1997) stress the important point that synchrony between subpopulations is a fundamental element in the process of species extinction. However, there is no mention of the related research in epidemiology, some of which has also been published in this journal. This prompts us to review some of the relevant history, and to highlight the very fruitful interaction that is possible between ecologists and epidemiologists working on population dynamics.

2. PERSISTENCE AND CHAOS IN ECOLOGY

A substantial body of theoretical work has explored the relationship between dispersal and population persistence (e.g. see references in Hanski & Gilpin (1997)). The principal conclusion of these theoretical investigations is that migration can facilitate the persistence of otherwise non-persistent systems: asynchrony between patches fuels a 'rescue effect' (Brown & Kodric-Brown 1977), whereby regions that have suffered extinction are recolonized by dispersing individuals from surviving subpopulations (Nicholson & Bailey 1935; Levins 1969; Adler 1993).

A related issue, which is at the centre of a continuing debate, is the potentially important role of chaotic dynamics in population ecology (e.g. Hastings *et al*. 1993; Stone 1993; Rohani & Earn 1997). Until recently, a popular view has been that chaos will be selected against, because the 'boom and bust' dynamics usually associated with it increase the probability of population extinction (Berryman & Millstein 1989). However, Allen *et al*. (1993) surprised the community by showing that chaos can in fact *prevent* global population extinctions, if there are several distinct subpopulations that are weakly coupled by migration and subject to locally varying external noise. The idea is that chaotic population dynamics within individual patches will indeed cause more frequent extinction events *locally*, but will also lead to asynchrony between different patches, and hence to the rescue effect described above. Ruxton (1994) showed further that extrinsic noise is not required; migration alone can prevent global extinctions if the individual subpopulations are chaotic in isolation. When weakly coupled, all patches can show simpler (cyclical or stable) dynamics, reducing the probability of global extinction, since different patches tend to be out of phase with each other (Kaneko 1990). Note that in these simple models, it seems essential that unlinked subpopulations are chaotic; otherwise, migration will synchronize populations (Holt & McPeek 1996) and increase the probability of global extinction. Most recently, Heino *et al*. (1997) have expanded on this work by introducing measures that can be used to quantify the importance of spatial synchrony in extinction events.

3. PERSISTENCE IN EPIDEMIOLOGY

Epidemiologists are interested in persistence from a different perspective, since they wish to *cause* the extinction of the (parasitic) animals that they study. Because of their public health importance—and the existence of an extensive medical infrastructure in developed countries for many decades—population dynamics of infectious diseases tend to be much better documented than any of the standard ecological examples. Large data sets of case reports of infectious diseases are available for the United States (e.g. Cliff *et al*. 1997) and for England and Wales (e.g. Grenfell & Harwood 1997). In Britain, for example, the Registrar General's Weekly Returns list notified cases of scarlet fever, whooping cough, diphtheria and measles in individual towns and cities in each week

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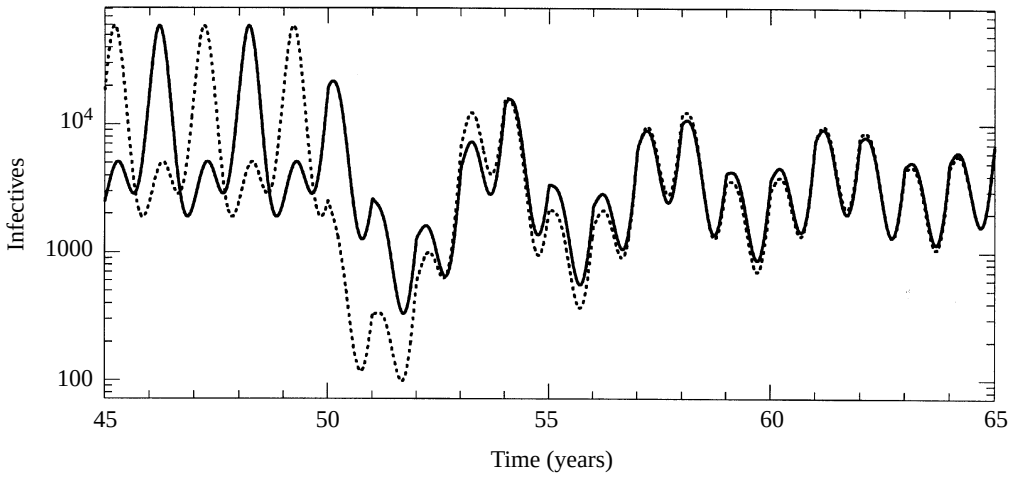

Figure 1. Synchronization by pulse vaccination in a simple epidemic model (a two-patch SEIR model). Solid and dotted curves show the number of infectives in each patch. The initial conditions yield a biennial epidemic cycle in which the two patches remain out of phase in the absense of vaccination. Immunization is initiated in year 50, after which 20% of the susceptible population is vaccinated on the first day of each year. Epidemic peaks, and the troughs between them, rapidly synchronize. The details of the model are as follows. The numbers of individuals who are susceptible, exposed or infective in the jth patch are denoted S_j, E_j and I_j, respectively. The total population in each patch is fixed at $N_1 = N_2 = 5 \times 10^7$. The mean lifetime of an individual is taken to be 50 years, so the (constant) per-capita birth rate is $\mu = 0.02\,\mathrm{yr}^{-1}$. The mean latent and infectious periods of the disease are $\sigma^{-1} = 8$ days and $\gamma^{-1} = 5$ days, respectively. The mean contact rate is $b_0 = 1250\,\mathrm{yr}^{-1}$, which yields a basic reproductive ratio of $R_0 \simeq b_0\,\gamma^{-1} \simeq 17$, similar to measles. The force of infection in the jth patch is $\lambda_j = \beta(t)[I_j + 0.001\,\Sigma_{k\neq j} I_k]/N_j$, where the seasonally forced contact rate is $\beta(t) = b_0[1 + b_1\cos(2\pi t)]$. The amplitude of seasonal variation in contact rate, relative to the mean, is $b_1 = 0.15$. The dynamical equations are $dS_j/dt = \mu N_j - (\lambda_j + \mu)S_j$, $dE_j/dt = \lambda_j S_j - (\sigma + \mu)E_j$, and $dI_j/dt = \sigma E_j - (\gamma + \mu)I_j$. The initial conditions were $S_1 = 0.05\,N_1$, $E_1 = I_1 = 0.0001\,N_1$, $S_2 = 0.07\,N_2$ and $E_2 = I_2 = 0.001\,N_2$.

since 4 November 1939. Weekly case reports for scarlet fever and diphtheria go back further to 7 January 1922. Deaths resulting from smallpox, measles, scarlet fever, diphtheria, whooping cough, typhus, enteric (typhoid and paratyphoid) fever and diarrhoea have been recorded weekly since 1897.

Recent work, using these data, has repeatedly emphasized that the best example of an ecological metapopulation with recorded dynamics over many generations is measles (Grenfell *et al.* 1995*b*; Lloyd & May 1996; Hess 1996; Grenfell & Harwood 1997). These studies build on the seminal work of Bartlett (1957, 1960). He identified a population threshold—the critical community size—above which measles persisted endemically. Below the threshold, measles was likely to suffer stochastic extinction in the troughs between epidemics; in metapopulation terms, Bartlett essentially identified a criterion for separating 'core' and 'satellite' patches (Grenfell & Harwood 1997). Incidence of measles is very widespread; before a mass vaccination programme was initiated in the late 1960s, the vast majority of people (worldwide) would have been infected by the age of ten. The data are especially good because measles has a particularly simple natural history of infection and is easy to diagnose unambiguously (e.g. Hamann 1994).

The existence of these data sets, the insights that have accumulated from analysing them and the large body of theory that has been developed to understand them (Anderson & May 1991), need to be brought to the attention of ecologists. We suggest that the ecological examples cited by Heino *et al.* (1997) do not demonstrate the importance of spatial synchrony nearly as convincingly as measles and other infectious diseases. Ecologists first developed the notion of the rescue effect, but epidemiology may be its most fertile testbed. This presents a wonderful opportunity for productive interchange between the two fields.

Detailed analysis of measles data reveals several broad patterns. In the absense of vaccination, parasite population densities in different towns and cities in many regions change roughly synchronously, and fade-outs (extinctions) are observed only in small places (Bolker & Grenfell 1996; Levin *et al.* 1997; Grenfell & Harwood 1997). Vaccinating at very high levels will reduce the reproductive ratio of infectives below unity and eliminate the infection (Anderson & May 1991); with vaccination below this level, large cities can fade out occasionally, but as the total infective density is reduced, the correlation of infective densities in different centres decreases (Bolker & Grenfell 1996). This may occur because reducing the total number of infections decreases the epidemic coupling between different regions.

Thus, paradoxically, continuous vaccination beyond a certain level causes decorrelation and hence *promotes* global persistence of the infection. An alternative immunization strategy that is gaining popularity involves periodically pulsed mass vaccination across age cohorts (e.g. Agur *et al.* 1993; Ramsay *et al.* 1994; Nokes & Swinton 1995, 1997; Stone *et al.* 1998). The original motivation for this policy was economic, but there may be a further benefit, which recent ecological theory highlights in our minds: simultaneous vaccination pulses in all subpopulations may synchronize local epidemics and thereby increase the probability of global fade-out

(Grenfell & Harwood 1997). As far as we are aware, this effect has not been demonstrated before, so we show explicitly in figure 1 that simultaneous pulse vaccination can synchronize epidemics in two coupled populations, potentially counteracting decorrelation caused by continuous vaccination. Of course, the overall effects of pulse vaccination may not be as straightforward as figure 1 suggests (we are currently investigating this problem in greater detail).

From our present point of view, the history of epidemics and immunization programmes can be interpreted as an extraordinary long-term empirical study of the role of spatial synchrony in ecological metapopulation dynamics, including extensive experimental manipulations (via vaccination). Only when the study animal is a pest can these sorts of experiments be carried out, but the knowledge accrued is valuable for other ecological research, and should be borne in mind when planning conservation strategies for endangered species, or biological control programmes.

4. CHAOS IN EPIDEMIOLOGY

Extensive analyses have also suggested that measles is a very good candidate for chaos, though there is as yet no unequivocal evidence (Hastings *et al*. 1993; Engbert & Drepper 1994; Grenfell *et al*. 1995*a*). Grenfell *et al*. (1995*b*) investigated the effects of locally chaotic dynamics on global persistence in the standard epidemiological model (the SEIR model, e.g., Anderson & May 1991). In contrast to the conclusions of Allen *et al*. (1993) and Ruxton (1994), they found that chaos is associated with *synchronous* local epidemics, increasing the probability of global parasite extinction. This occurs in epidemic models because chaos typically appears only at high levels of seasonal forcing (Glendinning & Perry 1997), which also tend to synchronize epidemics in local subpopulations.

5. CONCLUDING REMARKS

The basic elements in ecological and epidemological modelling are very similar because infectious disease dynamics have much in common with predator–prey relationships. The correspondence is especially dramatic for violently epidemic infections with short infectious periods, such as measles; however, the analogy with predators and prey is relevant to most infectious diseases (Anderson & May 1979; May & Anderson 1979). There are some parallels with host–parasitoid systems with non-overlapping generations, but the epidemiological models correspond most closely to predator–prey interactions with overlapping generations: susceptibles play the role of prey, infectives are the predators, spread of infection corresponds to dispersal of predators, and vaccination is loosely akin to refugia for the prey.

Despite these strong parallels, there are obvious biological differences between typical predator–prey interactions and infectious diseases, and these differences identify some potentially fruitful avenues of research. For example, diseases such as measles do not currently constitute the principal source of mortality for their host populations, unlike many pest species subject to biological control. How does this difference influence predator persistence patterns and the correlation of predator densities in distinct regions? Could the creation of prey refugia facilitate the persistence of pests by decorrelating local subpopulations? Another issue is seasonality, which plays a crucial role in synchronizing epidemics. How important are seasonal changes for predator–prey population dynamics?

These questions further illustrate the potential for cross-fertilization between the two disciplines. While research continues on the effects of synchrony and chaos, ecologists should pay attention to parallel developments by epidemiologists, and vice versa. There has also been relevant parallel research in theoretical physics on coupled map lattices (e.g. Kaneko 1990, 1994) and coupled oscillators (e.g. Ashwin & Swift 1992; Ashwin *et al*. 1993).

Synchrony appears to be undesirable for ecologists (hindering conservation efforts) but very advantageous for epidemiologists (increasing the probability of eradication). Since chaos is associated with asynchronous local extinctions of endangered species, but with synchronous epidemics of disease, chaotic dynamics appear to be desirable in both ecology and epidemiology. This conclusion is surprising, given what seems to us to be a widespread consensus that the existence of dynamical chaos is probably biologically irrelevant. Quite the contrary, detecting chaos in a biological population may have deep practical importance.

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