Ecology Letters, (1999) **2** : 167–177

REPORT

Resonance effects and outbreaks in ecological time series

Albert Blarer and Michael Doebeli
Zoology Institute, University of Basel, Rheinsprung 9, CH-4051 Basel, Switzerland. E-mail: blarera@ubaclu.unibas.ch

Abstract

Organismal response to environmental variability is an important aspect of ecological processes. We propose new mechanisms whereby environmental variability can cause cyclic population outbreaks due to the nonlinearity of the organismal response. We consider stage-structured populations that respond to variable environments with variable diapause or dormancy, and in which cyclic changes of the environment induce a resonance-like boost in the population size. If there is also a stochastic component of variation in the environment, the population outbreaks are magnified by the phenomenon of ''stochastic resonance''. The results show that large population fluctuations may not be due to extrinsic or intrinsic factors alone, but to a nonlinear interaction between the external environment and internal population processes. Indeed, in the presence of such nonlinearities even very small environmental fluctuations can cause massive fluctuations in population size. Our theoretical results may help to explain periodic population cycles and outbreak dynamics found in many infectious diseases and pest species. We also discuss the evolution of the response parameters that regulate diapause or dormancy and promote the outbreak dynamics in variable environments.

Keywords

Diapause, dormancy, nonlinear population dynamics, outbreaks, seasonality, stage-structured model, stochastic environments, stochastic resonance.

Ecology Letters (1999) 2 : 167–177

INTRODUCTION

Population outbreaks of pestiferous organisms and infectious diseases have, for various reasons, always received a great deal of attention. However, it is only recently that a rigorous theory for outbreak dynamics has begun to emerge (Berryman 1987). The aim of such a general outbreak theory is to classify different types of outbreaks and, most importantly, to investigate their underlying ecological mechanisms.

In this paper we offer new explanations for population outbreaks. The basis for these explanations is the interaction of organisms with their variable environments. Almost all environments exhibit two major forms of variability: periodic and erratic changes. Long-term climate cycles, such as the ongoing El Niño phenomenon, often cause periodic environmental changes spanning several years. Annual seasonality is another example of periodic and fairly predictable environmental changes. On a shorter time scale, less predictable weather changes dominate the environmental variability. Short-term day-to-day fluctuations in temperature, precipitation or humidity, exhibit irregular patterns that are best described in terms of stochastic fluctuations (Clifford & McClatchley 1996).

In general, organisms display a huge repertoire of responses to cope with periodic as well as erratic environments. This repertoire extends from purely physiological responses to specific behavioural patterns, such as dispersal and migration (Dingle & Hegmann 1982; Tauber *et al.* 1986; Dingle 1996). In this study we will focus on a very common organismal response to variable environments: diapause. Since Cohen's seminal work (Cohen 1966), diapause, or dormancy, has become a paradigm for an optimal life history strategy in variable environments. In short, if environmental conditions become unfavourable many organisms are able to enter a dormant stage in order to continue the life cycle when conditions improve.

Diapause is very common in insects (Brown & Hodek 1983), including those forest insects that cause severe defoliation damage during their outbreaks (see Berryman

©1999 Blackwell Science Ltd/CNRS

1988; Watt *et al.* 1990; Hunter 1995). Many plants endure hostile periods as dormant seeds (Lang 1996; Simpson 1990), among them many weed species. Among the microbial organisms that are important to public health, many bacterial species are able to survive adverse conditions as spores (Brock 1994). Others, such as the important group of arthropod-borne viruses, have extremely persistent life stages to survive adverse conditions (Reeves 1974; Monath 1993).

From an ecological and an evolutionary perspective, diapause or dormancy are usually interpreted as risk-reducing or bet-hedging strategies that maximize some measure of fitness (see Yoshimura & Clark 1993). In a series of papers by Tuljapurkar and Istock (Istock 1981; Tuljapurkar 1990; Tuljapurkar & Istock 1993) it was shown that diapause is adaptive in variable environments. Here we shall adopt an ecological rather than evolutionary view towards organismal responses and stress the following point: diapause, or other organismal responses to variable environments, not only affect the fitness of individuals but may also have a major influence on the population dynamics. Using a simple stage-structured population model and diapause as a response, we will show that nonlinear organismal responses to environmental variability can have drastic effects on population dynamics. In particular, we show that nonlinear organismal responses may cause the sporadic bursts in population size typical of the outbreak dynamics encountered in important infectious diseases and many pest species.

THE MODEL

We start by considering two major types of environmental variation: (i) periodic or seasonal changes, and (ii) irregular or stochastic fluctuations. With

$$E_t = \mu + A\sin\left(\frac{2\pi}{T}t\right) + D\xi(t) \qquad (1)$$

we may embody an environmental factor E_t, such as an ambient temperature or rainfall at time t, which cycles periodically with amplitude A and period T around a mean value μ. In addition, fluctuations of the environmental states E_t may have a stochastic component $D\xi(t)$, where $\xi(t)$ is drawn from a Gaussian distribution with mean $<\xi> = 0$ and variance $\mathrm{Var}(\xi) = 1$, and where the parameter D quantifies the magnitude of the stochastic fluctuations.

Following Tuljapurkar (1990), we next consider a population that is composed of two stages: immature and adult individuals. The environmental states given by eqn 1 are assumed to affect the immature stages that respond to variable values of E_t with variable diapause or dormancy. The dynamics of the numbers of immatures I_t and adults N_t is described in discrete time by

$$\begin{bmatrix} I_{t+1} \\ N_{t+1} \end{bmatrix} = \begin{bmatrix} f(E_t) & M(N_t) \\ s(1-f(E_t)) & 0 \end{bmatrix} \begin{bmatrix} I_t \\ N_t \end{bmatrix} \qquad (2)$$

Here, $f(E_t)$ denotes the fraction of immatures that delay their maturation and enter diapause or dormancy, depending on the environmental state E_t at time t. The pool of immatures at time $t+1$ then consists of two groups: a diapausing fraction $f(E_t)I_t$, and newborns produced by N_t adults at a reproductive rate $M(N_t)$. Adults are assumed to die after one reproductive season, whereas the immature individuals that do not enter diapause mature and survive with probability s to become adults. Therefore $s[1-f(E_t)]I_t$ is the number of adults at time $t+1$.

The fecundity M in eqn 2 is assumed to depend on N_t, i.e. on the number of reproducing adults at time t. More precisely, we assume that fecundity is given by

$$M(N_t) = \frac{\lambda}{1 + aN_t}. \qquad (3)$$

Here λ represents the maximal growth rate that is attained in the absence of any density effects. As the density N_t increases the fecundity $M(N_t)$ decreases according to a function of Beverton-Holt type (e.g. Yodzis 1989, p. 52ff). The parameter a scales the carrying capacities of adults and immatures. The assumption of density-dependence is not made in the original model (Tuljapurkar 1990) and is incorporated here in order to obtain realistic ecological dynamics. It is well known that density-dependence in discrete time systems affects the population dynamics and can give rise to various types of population dynamics, including limit cycles and chaos (May 1981; Hastings *et al.* 1993). However, the type of density-dependence used in this study does not induce such complex population dynamics, because it describes strict contest competition (Bellows 1981). The results below are not sensitive to the particular choice of function used to describe density dependence. For example, very similar results are obtained with stable Ricker systems. Preliminary investigations not reported here indicate that with complex dynamics resulting, e.g. from scramble competition, the resonance effects reported below are still present, but would interfere with intrinsic population fluctuations resulting from strong density dependence.

The key ingredient of our model is the immature's response to variable environmental states, i.e. the probability of diapausing $f(E_t)$. We use the following sigmoid relationship for this function:

$$f(E_t) = \delta\left(1 - \frac{1}{1 + \exp[c(P - E_t)]}\right). \qquad (4)$$

©1999 Blackwell Science Ltd/CNRS

A typical form of eqn 4 is shown in Fig. 1. For illustrative purposes we may think of E_t as representing the ambient temperature. Low temperatures then yield large fractions of diapausers in Fig. 1, whereas for increasing temperatures the diapausing fraction falls off in a nonlinear fashion. This type of response is regularly observed in insect diapause, which may depend on temperature as well as on the photoperiod (see, for example, Tauber *et al.* 1986). In general, however, eqn 4 may represent any type of organismal response that "switches" in a nonlinear way over some range of environmental states.

The shape and position of the response function are determined by the two parameters c and P in eqn 4. The parameter c determines the slope in the transitional part of the response function. Increasing values of c yield a more abrupt transition. Thus, c may reflect the sensitivity with which the pool of immatures responds to a changing environment. For example, eqn 4 turns into a step function as $c \to \infty$; diapause then represents an all-or-none response depending on whether the environmental states E_t are above or below the "threshold-value" P. The parameter P denotes the inflection point of the response curve. In the following discussion we will refer to P as the "position" of the response curve, because a change of its value causes the response curve to shift along the environmental axis, without changing its shape. The response described so far is multiplied by a parameter δ whose value is chosen $\leqslant 1$; δ represents the maximal fraction of diapausers.

Eqns 3 and 4 determine the dynamical system (2), which we studied by numerical simulations. As we will see, the position of the response curve P, as well as the stochastic term $D\xi(t)$ in the environmental state E_t, have major effects on the dynamics of system (2).

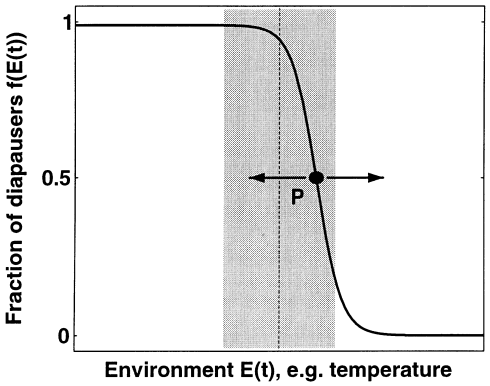

Figure 1 The diapause response. The probability of entering diapause varies as a function of the state of the environment. The grey area represents a realized range of environmental variation, e.g. of the ambient temperature. With respect to such a fixed environmental range the response curve can be shifted along the environmental axis by changing the values of P in eqn 4, which represents the inflection point of the response function. The shape, i.e. the slope at the inflection point P, is determined by the parameter c in eqn 5. The larger the values of c, the more abrupt is the transition between diapausing and nondiapausing fractions.

RESULTS

Periodic environments without noise

From Fig. 1 it is obvious that the position P of the response function f with reference to a fixed range of environmental fluctuations (the grey area in Fig. 1) is crucial in determining the population structure of immatures and adults. Consider the following extreme positions of the response function: Shifted to the far left, corresponding to $P << \mu$, we obtain a population in which almost all individuals are direct developers, irrespective of the environmental state (grey area), and diapausing stages will be encountered only rarely. At the other extreme, if $P >> \mu$, large diapause fractions are the rule and direct development will be rare. In contrast, we expect large variations in the dormancy response for intermediate positions, i.e. when P lies within the typical range of environmental variation. In this case even small fluctuations in the environmental state may cause large changes in the diapause fractions.

We first consider the deterministic case of cycling environments. By setting $D = 0$ in eqn 1 we obtain periodic cycles of the environment within the fixed range $\mu \pm A$. Figure 2, for which we chose $\mu = 2$ and $A = 0.05$, shows the population dynamics of adults as a function of the response's position P for increasing environmental periods $T = 3$, 7 and 11.

For a constant fraction of diapausers f it can be shown analytically that the population resides in a stable equilibrium state. Such constant fractions f occur in our model for the two most extreme positions of P: for $P << \mu$, i.e. for a life history with almost no diapause, the fraction $f \to 0$, whereas at the other extreme, with $P >> \mu$, $f \to \delta$. The corresponding equilibrium state for the adults is straightforward to calculate from eqn 2:

$$N^{\star} = \frac{\lambda s - 1}{a}, \qquad (5)$$

where a is the scaling parameter in eqn 3, λ is the maximal growth rate and s is the survival probability of diapausing juveniles. This equilibrium state can be seen in Fig. 2 for the two most extreme positions of P.

More interesting population dynamics occur when the position P of the response function is shifted towards the range of environmental variation. Then the population exhibits larger and larger cycles that are in phase with the environmental variation and reach a maximum at positions

©1999 Blackwell Science Ltd/CNRS

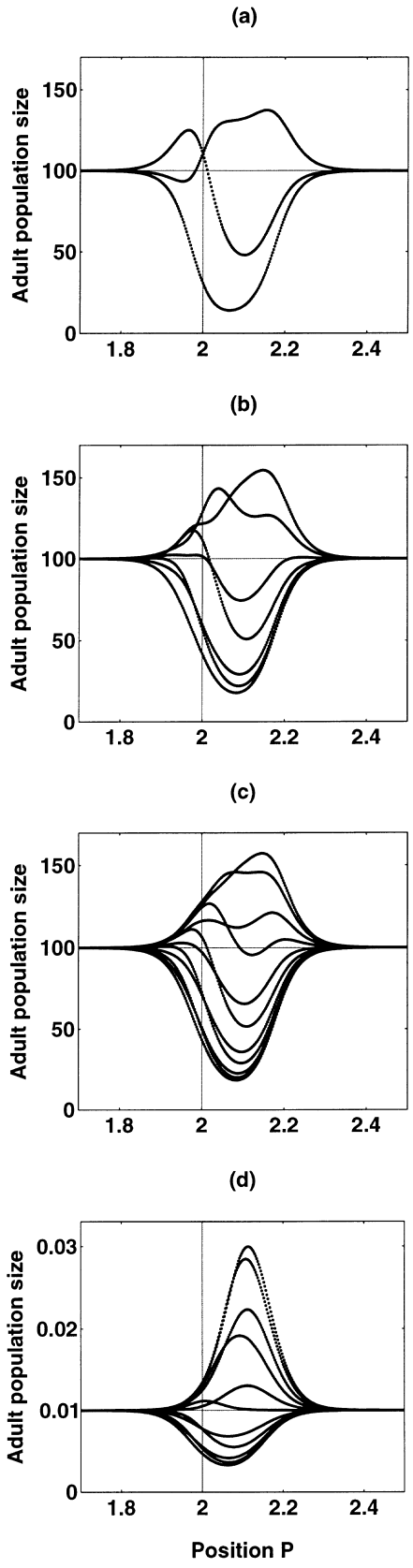

Figure 2 (Left) The dynamics of the adult population are shown as a function of the position P of the immatures' response function. In (a)–(c) the environment cycles deterministically with different periods $T = 3$, 7 and 11, respectively. The adult population size at the most extreme positions of P corresponds to the equilibrium state (see text). However, for intermediate positions of P the population dynamics cycles in step with the environmental cycles, i.e. population size alternates between three different states if $T = 3$, between seven states if $T = 7$, and so forth. (The graphs should be interpreted as analagous to a bifurcation diagram.) Positions P near the environmental mean ($\mu = 2.0$) yield the largest population fluctuations, despite the small amplitude ($A = 0.05$) of the environmental variation. This resonance effect is a consequence of the nonlinear diapause response. Note that the magnitude of the fluctuations in the resonance region increases as the environmental period T becomes larger. In (d) the same environmental settings are used as in (c), except that a smaller survival rate s reduces the adult carrying capacity (see text). Nevertheless, the resonance at intermediate positions P persists. The parameter values are $D = 0$, $c = 30.0$, $\delta = 0.99$, $\lambda = 2.0$ $a = 0.001$, $s = 0.55$ in (a)–(c) and $s = 0.500005$ in (d).

P that are slightly above the environmental mean μ. It is important to realize that the fluctuations in population size are very large compared with the environmental fluctuations. More precisely, if we compare the coefficients of variation (i.e. standard deviation divided by mean) of the population time series and the environmental time series, then for intermediate positions of P the population's coefficient can exceed the environmental coefficient by roughly three orders of magnitude. Thus, even very small environmental variation can induce large fluctuations in population size due to the nonlinearity of the organismal response: the nonlinearity leads to an amplification of environmental variation and therefore induces a resonance effect in the population dynamics.

It should be mentioned at this point that such amplification of environmental variation can also be observed in linear models, e.g. in periodically perturbed Lotka–Volterra systems (Nisbet & Gurney 1982). In fact, there is quite a large literature investigating periodically driven systems, as exemplified by the forced pendulum (e.g. Drazin 1992, ch. 7). The effects of adding stochastic environmental fluctuations on top of periodic forces have also been investigated (e.g. Nisbet & Gurney 1982). While stochastic effects may sometimes lead to rather complicated phenomena (e.g. Rand & Wilson 1991), the traditional notion still is that the magnitude of the effects of stochastic noise is proportional to the noise intensity (Nisbet & Gurney 1982). Here we show that this need not necessarily be the case, and that very small environmental fluctuations can lead to very large population outbreaks even though large environmental fluctuations will not.

©1999 Blackwell Science Ltd/CNRS

This occurs when the nonlinearity of the organismal response leads to stochastic resonance (see below).

In the system with deterministic (periodic) environmental fluctuations, population resonance at intermediate values of P can be seen for all environmental periods and becomes more prominent if we increase the length of the periods (Fig. 2a–c). The size of the resonance region and the precise dynamics in this region depend on the shape of the response function. In all panels of Fig. 2 we used the same slope for the response function. Simulations show that larger values of c, i.e. steeper responses, narrow the resonance region, but increase the magnitude of the fluctuations. The opposite pattern is observed for smaller c, and resonance completely disappears if $c \to 0$, which corresponds to a constant diapause response independent of the environmental states.

Interestingly, the population boosts persist if the carrying capacity of the adults is lowered. From eqn 5 it is easy to see that we need $s > 1/\lambda$ in order to obtain a persistent population, and as s approaches the marginal value $1/\lambda$ the carrying capacity of adults shrinks. However, the resonance-like boosts persist as is shown in Fig. 2(d), for which we chose the same environmental settings as in Fig. 2(c), but a smaller survival rate s for the dormant stage. Not only is the resonance phenomenon still present, but the ratio between the maximum population size of the boost and the carrying capacity is much larger in Fig. 2(d) as compared to Fig. 2(a)–(c).

Adding environmental noise

So far we have described a purely deterministic model. We now investigate how noisy variation of the environment interacts with the nonlinear organismal response and whether it induces similar magnification effects in the population size. The classic view of noise in (ecological) systems is that it simply blurs otherwise deterministic population dynamics. This view holds for most population dynamics of our model, in particular those outside the resonance regions shown in Fig. 2. However, within the resonance region, and in particular for the dynamics that appear around the maximal boost of population size, even small amounts of noise can produce very drastic effects.

Figure 3 shows four different time series that are obtained by adding different intensities of noise to the same deterministic population dynamics. The purely deterministic case is shown in Fig. 3(a), and corresponds to a time series in the deterministic resonance region (see Fig. 2c). Adding very small amounts of noise leads to the traditional picture by perturbating slightly the otherwise deterministic cycles (Fig. 3b). However, further increasing the noise intensity changes the population dynamics drastically (Fig. 3c): at more or less regular intervals,

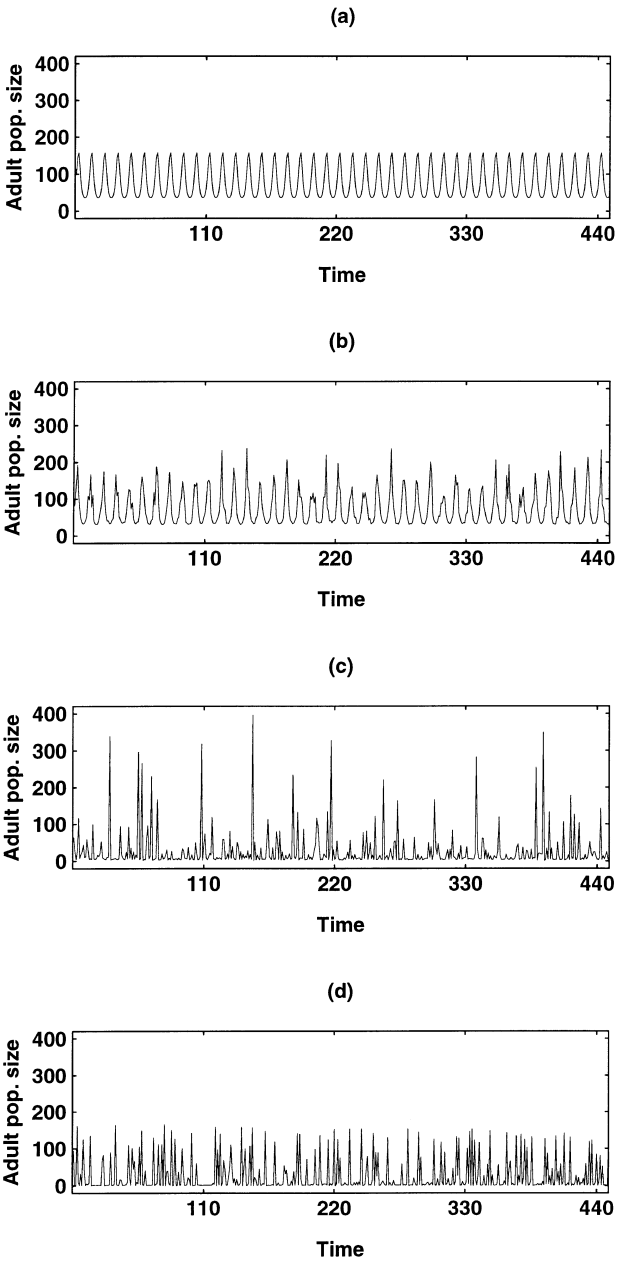

Figure 3 Different intensities of environmental noise change the population dynamics. In (a) the deterministic case with $D = 0$ in eqn 1 is shown. Small noise intensities (b), i.e. small values of D, cause only a small perturbation of the deterministic cycles. For intermediate noise levels, stochasticity causes large, regular outbreaks, as shown in (c). This outbreak dynamics is due to stochastic resonance (SR). Characteristically for SR, the coherence and the amplifying effects vanish for large noise intensities, as shown in (d). Here outbreaks are much less regular and not exceedant anymore. Noise levels used for the panels (a)–(d) are $D = 0.0$, 0.01, 0.075 and 0.15, respectively. In all parts we set $\mu = 2.0$, $A = 0.05$, $T = 11$, $P = 2.15$, $c = 30.0$, $\lambda = 2.0$, $a = 0.001$ and $s = 0.55$.

©1999 Blackwell Science Ltd/CNRS

which correspond to the underlying environmental periodicity, the adult population exhibits large, pulse-like outbreaks (or "pulse gradient outbreaks" using the classification of Berryman 1987). The mean population size, as compared with the deterministic case, has decreased in this noisy environment, and the change in population size during an outbreak pulse therefore becomes even more prominent. The intuitive explanation for the periodicity of the outbreaks in Fig. 3(c) is as follows: the position P used for all simulations in Fig. 3 implies that dormancy is rather common in the population. Thus, only environmental states that clearly exceed the value of P will release large numbers of adults from the pool of dormants and produce an outbreak. Such exceedant environmental states in turn occur stochastically, but preferentially when the deterministic cycles of the environment, though very small, attain their maximal values $\mu + A$. This explains the periodicity of the outbreaks. Before we turn to an explanation for the exceedant population sizes during these outbreaks, we first note what happens when the level of noise is further increased (Fig. 3d): the population displays more, but much smaller outbreaks at much less regular intervals. The overall picture we draw from this sequence of time series is that the (temporal) coherence and the magnitude of the population's outbreaks is maximized at intermediate noise levels.

The outbreak behaviour at intermediate noise levels can be described using the concept of stochastic resonance, a phenomenon that is widely known in physics and explains the two observations of noise-induced magnification and of the increase in the coherence of outbreaks at intermediate noise levels. Stochastic resonance (SR) has been studied and established in a variety of experimental systems and models, and in different fields of science (for a review see Wiesenfeld & Moss 1995). In essence, SR describes a nonlinear and cooperative effect of noise by which an underlying (weak) periodic signal is strongly amplified. The basic ingredients necessary for stochastic resonance are (i) a periodic signal, (ii) noise and (iii) a threshold function. It is obvious that the former ingredients correspond to the environmental variation in our model. With reference to the threshold function we already mentioned in the model section that the organismal response defined by eqn 4 turns into such a threshold (or step) function as the parameter $c \to \infty$. However, a more general response function with $c < \infty$, but with a still sufficiently steep (i.e. nonlinear) transition, often suffices to induce the phenomenon of SR (see Bezrukov & Vodyanoy 1997). Thus, our ecological model contains the basic components necessary for stochastic resonance, and we now show that this phenomenon does indeed occur in our model.

Stochastic resonance

To infer stochastic resonance as a causal mechanism and to quantify the exceedant population peaks at intermediate noise levels we have to show two properties. First, that outbreaks exhibit a periodicity that is related to the underlying, periodic "signal", i.e. the environmental cycles. Second, we have to show that the magnitude of outbreaks undergoes a noise-induced amplification that varies with increasing noise levels in a characteristic way: the amplification should be large for intermediate noise levels, and it should be small for very small as well as for large noise levels. This is in contrast to the traditional notion that the effects of noise are proportional to the noise intensity. The starting point for the following analysis are the population time series shown in Fig. 3. Since we are interested in quantifying the outbreaks in the time series, we will first extend our phenomenological description of outbreaks by the following procedure: we define an outbreak as a rise in population size within two consecutive time steps that exceeds a certain ratio θ. For example, a value of $\theta = 0.05$, which we will use here, implies that an outbreak event occurs at time t if the population size at time t exceeds the population size at time $t-1$ by a factor that is larger than 20. (The exact value of θ is not critical for the results, i.e. we obtain qualitatively the same results if outbreaks are defined as, say, a 10-or a 50-fold increase in population size.) Using this procedure we now filter the original time series to extract outbreak events. Let $\hat{N}_t$ represent the population size of the filtered time series at time t. Then

$$\hat{N}_t = \begin{cases} N_t, \text{ if } \frac{N_{t-1}}{N_t} < \theta \\ 0 \ , \text{ if } \frac{N_{t-1}}{N_t} \geq \theta \end{cases} \qquad (6)$$

Using the filtered time series $\{\hat{N}_1, \hat{N}_2, \ldots, \hat{N}_t\}$ we can now ask questions about the periodicity and magnitude of outbreaks. First we ask, what are the waiting times between two successive outbreaks? For low noise levels (corresponding to Fig. 3b) there are no outbreaks at all (applying to our outbreak definition above), and the waiting times are therefore infinite. At noise levels where outbreaks first appear (noise intensity $D \approx 0.04$), the intervals between outbreaks are scattered over a large range, although a slight regularity of outbreak events might be already discernible. For intermediate noise levels (corresponding to Fig. 3c) the distribution of waiting intervals is shown in Fig. 4(a): the successive peaks of this histogram clearly correspond to the underlying environmental periodicity (here $T = 11$) and integer multiples of it. The regularity of this oscillating pattern implies a strong temporal coherence of the outbreak events. It is important to note that the deterministic part, i.e. the weak

©1999 Blackwell Science Ltd/CNRS

periodic fluctuation of the environment, is responsible for this temporal coherence. Without this weak periodic forcing outbreaks of similar size might occur, although their temporal regularity vanishes. As we further increase the level of noise the temporal coherence is destroyed and the waiting times exhibit a distribution as shown in Fig. 4(b). Therefore, as noise levels become large the noise-induced amplification vanishes.

The shape of these histograms and in particular the exponential decrease of the individual maxima of Fig. 4(a) are a first hint towards SR (Zhou & Moss 1990), but the shape alone is not a sufficient criterion to infer SR (Wiesenfeld & Moss 1995). We need further information on the magnitudes of the outbreaks. The classical way to do this (and to infer SR more generally) is to calculate the so-called signal-to-noise ratio (SNR) for a given time series and for different noise intensities. The SNR can be calculated from the power spectrum of a time series using, for instance, the Fast Fourier Transform algorithm (Press *et al.* 1990; Walker 1996). Figure 4(c) shows a typical power spectrum for the filtered population time series, shown in Fig. 3(c). We recognize a distinct peak at the frequency $1/T \approx 0.091$ (with $T = 11$) that corresponds to the regularly recurrent outbreaks. Information about the magnitude is contained in the height of this spectral peak. The SNR is then calculated from the height of the spectral peak and the noise level just adjacent to the spectral peak, as indicated in Fig. 4(c). When plotted against different noise intensities D the SNR yields a unimodal shape, as shown in Fig. 4(d), with a maximum at intermediate noise levels $D \approx 0.08$. The unimodal shape of this SNR-curve is the signature of stochastic resonance, and it is interpreted as follows: the noise of different intensities supports the spectral peak with different amounts of power. The SNR-curve then shows that intermediate noise levels supply the peak with maximal power. This in turn reflects the maximal magnitudes and the large coherence of outbreaks in the time series at intermediate environmental noise levels. We also tested an alternative approach to infer SR, using the histograms of Fig. 4 directly. Here, the number of intervals, i.e. the height of the histogram at the fundamental period T, is plotted against different noise intensities (see Zhou & Moss 1990). The result is the same, a unimodal curve with a maximum at intermediate noise levels.

Evolutionary considerations

In our model, outbreak dynamics emerge from the interplay between the extrinsic environmental variation and the nonlinear organismal response to this variation. From an evolutionary point of view a crucial question is, how does the response function evolve in a given environment? Will selection favour response types that yield large population fluctuations or even outbreaks? This question can be answered using evolutionary invasion scenarios (Dieckmann 1997; Geritz *et al.* 1998). For this the position of the response function P is considered as a phenotypic trait, subject to the forces of natural selection. The starting point for an invasion scenario is a resident population with a given phenotype. Then, a mutant phenotype is introduced into the resident population, and its long-term growth rate is calculated using the mutant's response function to environmental cues for determining its diapause probability, and using the resident's density for determining the mutant's reproductive output (since the mutant is rare its own density is negligible). If the mutant's response function is such that the mutant can cope better with the variable conditions of the environment, then the mutant's long-term growth rate is larger than 1, hence its frequency in the population increases. This scenario is called a successful invasion. By classifying successful and unsuccessful invasions for different combinations of mutant and resident phenotypes, we gain a systematic overview over the evolutionary favoured response types. Note, however, that successful invasions do not necessarily imply an exclusive persistence of the mutants, i.e. the resident is not necessarily displaced by a better coping rare mutant.

If the invasion scenario is applied to our diapause model several different outcomes are possible, representing a range of selection regimes from disruptive to stabilizing selection. There are, however, two general trends: first, selective forces are always very weak, i.e. invasion of mutants and displacement of residents, if it occurs, typically take a long time. This confirms the results of Tuljapurkar (1990) and Tuljapurkar & Istock (1993), who find weak selection in their similar but density-independent models. Second, phenotypes that yield a large mean population size are evolutionarily favoured. Therefore, one can use the mean population size as a correlate of the selective forces, with which we can summarize the evolutionary outcomes for the phenotype P as follows (Fig. 5): for most ecological settings the resonance dynamics (shown in Fig. 2 for the deterministic cases) yield a distinct depression of the mean population size (solid lines in Fig. 5), and the environmental stochasticity may magnify this depression (dashed lines in Fig. 5). Since phenotypes leading to higher mean population size are generally favoured by selection, this scenario corresponds to disruptive selection on P, as indicated by the arrow in Fig. 5(a). However, simulating full systems of competing phenotypes (instead of just long-term growth rates of rare mutants) reveals that phenotypes leading to smaller mean population sizes (but larger fluctuations!) may sometimes coexist with phenotypes inducing larger mean population sizes. This can be

©1999 Blackwell Science Ltd/CNRS

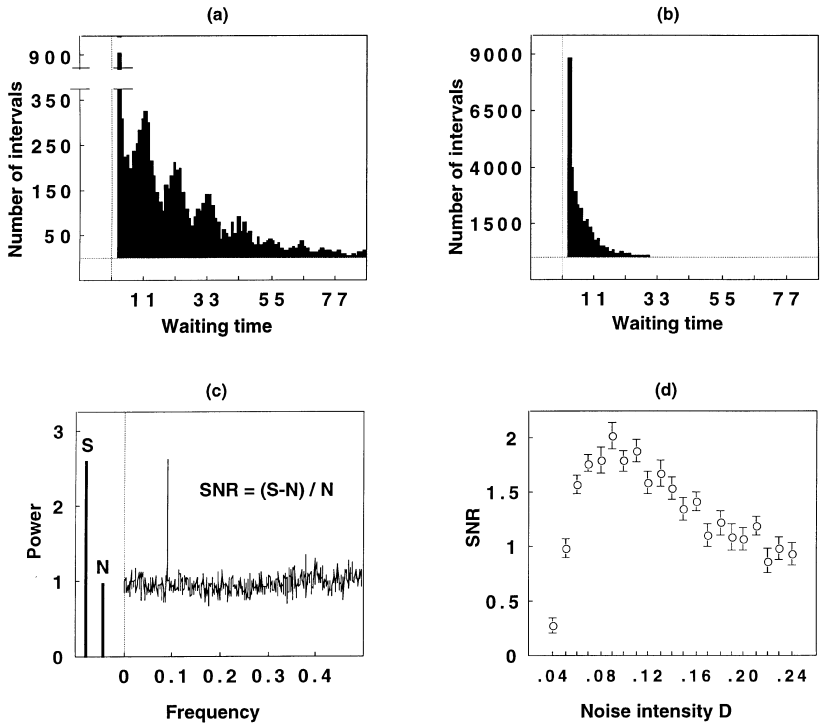

Figure 4 The distribution of waiting times between two successive outbreaks. For intermediate noise levels (a, time series of Fig. 3c) the temporal coherence of waiting times becomes maximal: at regular intervals, which correspond to the environmental period $T = 11$ and integer multiples of it, outbreak events occur more often. The coherent pattern is destroyed for larger noise levels (b, time series of Fig. 3d), where the sequence of outbreaks is temporarily much more dense. There are no predominant waiting times. The parameter settings are those used in Fig. 3(c, d), with $D = 0.075$ in (a) and $D = 0.15$ in (b). Part (c) shows a typical power spectrum of the filtered time series of an outbreak dynamics. The distinct peak embedded in a noisy background represents the regular outbreaks. The signal-to-noise ratio (SNR) is obtained from the height of the spectral peak S and the noise level N near the spectral peak, as indicated in (c). Calculating the SNR for different noise levels D yields a unimodal curve (d). The shape of the SNR curve implies not only an increase of the coherence but also an increase of the outbreak's magnitude at intermediate noise levels. The unimodal shape of the SNR-curve is the signature of stochastic resonance. The power spectrum and individual SNRs represent the average of 50 replicates; error bars in (d) correspond to one standard error.

explained by the weakness of the selective forces that act on a very local scale in phenotype space and allow for the coexistence of sufficiently different phenotypes. Thus, all things considered, the disruptive selection regime yields a heterogenous mixture of coexistent phenotypes, including the sporadic appearance of phenotypes exhibiting outbreak dynamics.

On the other hand, particular settings of the diapause model can also yield stabilizing selection. For example, in the system with a low carrying capacity shown in Fig. 2(d), the mean population size as a function of the phenotype P shows a distinct maximum in the resonance region (solid line in Fig. 5b), which becomes even more pronounced when environmental cycles are noisy (dashed line in Fig. 5b). Again, phenotypes that yield the largest mean population size are evolutionarily favoured, and hence selection is stabilizing for phenotypes centred around the peak region of Fig. 5(b). These selected phenotypes display a resonance dynamics and have the potential for stochastic resonance and outbreaks. In other words, natural selection may favour demographic parameters that lead to outbreak dynamics.

DISCUSSION

Variable diapause and dormancy are paradigms of strategies that organisms use to cope with variable environments. We considered two major types of environmental variation in this study: periodic cycles and stochastic fluctuations. The juvenile stage of our model organism is assumed to respond to variable environments with variable diapause. In a first part we have shown that even weak deterministic cycles of the environment can induce large fluctuations in population size. In our model this amplification of an environmental signal is caused by (i) a sufficient nonlinearity of the organismal response, and (ii) a

©1999 Blackwell Science Ltd/CNRS

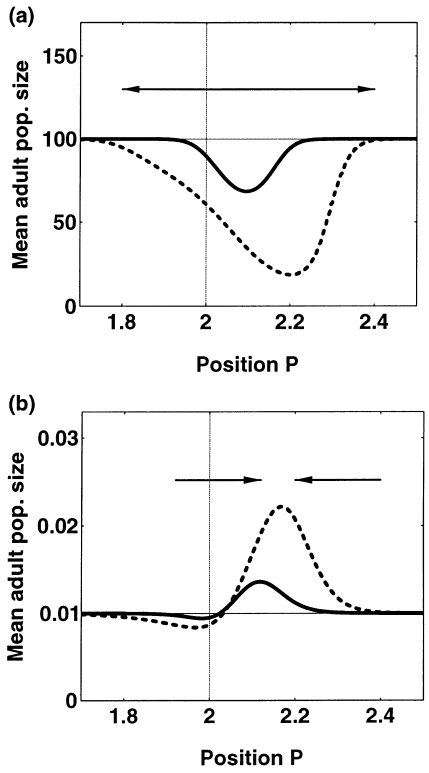

Figure 5 Invasion analysis yields two major selection regimes: disruptive and stabilizing selection. These are reflected in the mean population size. In (a) the mean population size is shown for various position P of the response function for the parameter values of Fig. 2. Solid lines represent the deterministic case with $\mu = 2.0$, $A = 0.05$, $T = 11$ and $D = 0$. Dashed lines represent the noisy environment with $D = 0.075$. Selection is disruptive as indicated by the drop in mean density for intermediate values of P. For (b) the parameter values of Fig. 2(d) were used. Here the mean population sizes show a maximum for intermediate values of P. Accordingly, selection is stabilizing. Note that the maximum becomes more pronounced in the stochastic environment.

particular position of the response function with respect to the range of environmental variability. If, in addition, the environment has a stochastic component, then for particular noise intensities the adult population dynamics can exhibit large and regular outbreaks. The regularity of outbreaks is explained by the periodic forcing of the environmental cycles. Even though the magnitude of these environmental cycles might be very small, the periodic forcing is essential for the temporal coherence of outbreak events. As in the deterministic case, the nonlinearity and the position of the response function are critical for inducing this outbreak behaviour. Because the largest amplification occurs for intermediate noise levels, we have interpreted the outbreaks as stochastic resonance.

Stochastic resonance (SR) describes a nonlinear, noise-induced effect, whereby the coherence and the magnitude of an underlying periodic signal becomes greatly magnified. An excellent review on this phenomenon is given by Wiesenfeld & Moss (1995), and many technical aspects of SR and recent research results are reviewed by Gammaitoni *et al.* (1998). SR has a very interdisiplinary appearance: it was first introduced in a palaeoclimatic model to account for the periodic reoccurrence of the Earth's ice ages (Benzi *et al.* 1982; Nicolis 1982). Although most results come from various disciplines within physics, the concept of SR has also been studied in neurophysiology, where it has been found in the mechanosensory system of crayfishes. Here, the SR-system acts as a neurosensorial amplifier, able to detect very weak periodic signals that lie below the detection threshold of the sensory neurones (Douglass *et al.* 1993).

In ecology, however, the formulation of stochastic resonance is new. Only very recently Huppert & Stone (1998) used SR to explain the periodic mass bleaching events in coral reefs. Mass bleaching occurs every $\sim$ 3–4 years in step with the El Niño phenomenon, which is known to induce periodic changes in the sea surface temperature. Random environmental fluctuations then have the potential to reinforce these temperature oscillations and to create hot spot zones where the water temperature exceeds a critical threshold, with fatal consequences for the coral reefs (see also Stone *et al.* 1998).

Outbreak dynamics of pest species or infectious diseases have to our knowledge never been investigated within the framework of SR. Even though the idea is not new that extrinsic environmental factors and stochastic effects may actively trigger pest outbreaks or epidemics (e.g. London & Yorke 1975; Schwartz 1985; Martinat 1987), the common view is that extrinsic environmental variation is responsible for the synchronization of population fluctuations [often referred to as the "Moran effect", see, for example, Gurney *et al.* (1991, 1994) for synchronizing effects of diapause], but is not the cause of large fluctuations in population size, except if the environmental fluctuations themselves are large [see, for example, Myers (1998), Grenfell *et al.* (1998)]. Stochastic resonance as proposed by our model implies both elements: synchronization is obtained through the increase in coherence, and outbreaks are triggered by the nonlinear interaction between environment and organismal response. In particular, our results show that even very small environmental variation can induce very large fluctuations in population size. Part of the biased view towards environmental variation may be due to the purely correlative (and therefore noncausal) comparison between ecological and environmental time series. The following example may illustrate this point further and emphasize at the same time the possible significance of SR in ecology: many infectious diseases and pest species in forestry or agriculture are well-known for their cyclic reoccurrence and periodic outbreaks

©1999 Blackwell Science Ltd/CNRS

(e.g. Anderson & May 1980). In some cases the periodic occurrence correlates well with particular environmental events. Monath (1993), for instance, mentions the St. Louis encephalitis, an infectious disease caused by an arthropod-born virus. The appearance of encephalitis epidemics in the Southern United States follows a $\sim$ 10–11-year cycle and correlates well with the nadir of sun spot activity. The causal relationships between sun spot activity and disease dynamics remain obscure. However, sun spot cycles are known to cause a series of slight, periodic changes in the Earth's surface temperature, and the $\sim$ 10–11-year cycle is among the most prominent cycles (Schove 1983). The point is that weak environmental changes, even barely distinguishable from their noisy background (as is the case in the temperature changes induced by sun spot activity) could be detected by a nonlinear response of living systems and result in resonant population dynamics, as proposed in this paper. In the encephalitis example such a nonlinear response is not necessarily confined to the virus itself, but may occur in the life history of the vector insects or even in a nonlinear transmission rate that depends on temperature (e.g. Watts *et al.* 1987). There are other examples where sunspot activity has been associated with outbreak dynamics, especially in forest insect outbreaks (Myers 1998). Still other examples associate the course of epidemics with the periodic changes of El Niño: Bouma & Dye (1997) found that in the year following an El Nino event in the Eastern Tropical Pacific, malaria mortality and morbidity increases on average by 37% in Venezuela. However, the (causal) connection between outbreaks and weak environmental changes in all these examples remains speculation. Here, stochastic resonance offers a possible mechanism that explains the correlation between population outbreaks and faint environmental events.

To further test such ideas, time series analysis of "real" data is necessary. Such an analysis of outbreaks would include a comparison of population time series from different geographical locations that differ in their environmental variation. The comparison of such time series may provide more information on the interactions between environment and organismal response, and their effects on the population dynamics [see, for example, the approaches of Istock (1981) and Grenfell *et al.* (1998)].

The time series analysis described in Fig. 4 focuses on the variability of environments and the dynamics of population but excludes the variation of the organismal response. This variation may be critical for the explanation of outbreak behaviour, because only particular responses yield outbreak dynamics. Our evolutionary considerations suggest a broad variability for the diapause response, which corresponds well with the variability found in natural populations. In natural populations considerable amounts of genetic variability and phenotypic plasticity have been found in the diapause response (Tauber *et al.* 1986).

An approach that focuses more on the organismal response than on the dynamic aspects of outbreaks has been recently put forward by Hunter (1995), who compared life-history traits and ecological traits of outbreak and non-outbreak forest Lepidoptera. Since outbreak and non-outbreak species often share the same environment, outbreak species must differ in life-history or ecological traits, and in fact, Hunter (1995) found such significant differences in traits. In this paper we have argued that outbreak dynamics result from the nonlinear interaction between environment and organismal response, and that differences in the response may determine whether a species does or does not exhibit outbreak dynamics. Following this idea we propose that traits that can be associated with outbreak behaviour should be tested for their environmental determination and in particular for their effects on the population dynamics. Moreover, an evolutionary approach may give further insights. As we have seen, a change of one single trait, here the survival rate s, was sufficient to change the evolutionary outcome (Fig. 5), which in turn determines whether a population exhibits outbreak dynamics. Thus, using an approach that combines population dynamics, environmental dynamics and the evolution of life histories seems to be a promising way to gain a better understanding of outbreak dynamics.

ACKNOWLEDGEMENTS

We thank Lewi Stone for discussions. This research was supported by the Schweizerische Nationalfonds (Grant 3100–43042.95 to M.D.) and the Roche Research Foundation.

REFERENCES

Anderson, R.M. & May, R.M. (1980). Infectious diseases and population cycles of forest insects. *Sci.*, 210, 658–661.

Bellows, T.S. (1981). The descriptive properties of some models for density dependence. *J. Anim. Ecol.*, 50, 139–156.

Benzi, R., Parisi, G., Sutera, A. & Vulpiam, A. (1982). Stochastic resonance in climatic change. *Tellus*, 34, 10–16.

Berryman, A.A. (1987). The theory and classification of outbreaks. *Insect Outbreaks*, eds P. Barbosa & J.C. Schultz. London: Academic Press, pp. 3–30.

Berryman, A.A. (1988). *Dynamics of Forest Insect Populations: Patterns, Causes, Implications*. New York: Plenum Press.

Bezrukov, S.M. & Vodyanoy, I. (1997). Stochastic resonance in non-dynamical systems without response thresholds. *Nature*, 385, 319–321.

Bouma, M.J. & Dye, C. (1997). Cycles of Malaria associated with El Nino, in Venezuela. *J. Am. Med. Assoc.*, 278, 1772–1774.

Brock, T.D. (1994). *Biology of Microorganisms*. London: Prentice Hall.

©1999 Blackwell Science Ltd/CNRS

Brown, V.K. & Hodek, I. (1983). *Diapause and Life Cycle Strategies in Insects*. Boston: The Hague.

Clifford, N.J. & McClatchey, J. (1996). Identifying the time-scales of environmental change: the instrumental record. *Time-scales and Environmental Change*, eds T.S. Driver & G.P. Chapman. London: Routledge, pp. 88–107.

Cohen, D. (1966). Optimizing reproduction in a randomly varying environment. *J. Theoret. Biol.*, 12, 119–129.

Dieckmann, U. (1997). Can adaptive dynamics invade? *Trends Ecol. Evol.*, 12, 128–131.

Dingle, H. (1996). *Migration–the Biology of Life on the Move*. London: Oxford University Press.

Dingle, H. & Hegmann, J.P. (1982). *Evolution and Genetics of Life Histories*. New York: Springer.

Douglass, J.K., Wilkens, L., Pantazelou, E. & Moss, F. (1993). Noise enhancement of information transfer in crayfish mechanoreceptors by stochastic resonance. *Nature*, 365, 337–340.

Drazin, P.G. (1992). *Nonlinear Systems*. Cambridge: Cambridge University Press.

Gammaitoni, L., Hänggi, P., Jung, P. & Marchesoni, F. (1998). Stochastic resonance. *Rev. Modern Physics*, 70, 223–287.

Geritz, S.A.H., Kisch, É., Meszéna, G. & Metz, J.A.J. (1998). Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.*, 12, 35–57.

Grenfell, B.T., Wison, K., Finkeristädt, B.F., Coulson, T.N., Murray, S., Albon, S.D., Pemberton, J.M., Clutton-Brock, T.H. & Crawley, M.J. (1998). Noise and determinism in synchronized sheep dynamics. *Nature*, 394, 674–677.

Gurney, W.S.C., Crowley, P.H. & Nisbet, R.M. (1991). Locking life-cycles on to seasons: circlemap models of population dynamics and local adaptation. *J. Mathemat. Biol.*, 30, 251–279.

Gurney, W.S.C., Crowley, P.H. & Nisbet, R.M. (1994). Stage-specific quiescence as a mechanism for synchronizing life cycles to seasons. *Theoret. Pop. Biol.*, 46, 319–343.

Hastings, A., Hem, C.L., Ellner, S., Turchin, P. & Godfray, H.C. (1993). Chaos in ecology: is mother nature a strange attractor? *Annu. Rev. Ecol. Syst.*, 24, 1–33.

Hunter, A.F. (1995). Ecology, life history and phylogeny of outbreak and nonoutbreak species. In *Population Dynamics. New Approaches and Synthesis*, eds Cappuccino, N. & Price P.W. San Diego: Academic Press, pp. 41–64.

Huppert, A. & Stone, L. (1998). Chaos in the Pacific's coral reef bleaching cycle. *Am. Naturalist*, 152, 447–459.

Istock, C.A. (1981). Natural selection and life history variation: theory plus lessons. *Insect Life History Patterns: Habitat and Geographic Variation*, eds R.F. Denno & H. Dingle. New York: Springer, pp. 113–127.

Lang, G.A., ed. (1996). *Plant Dormancy*. Cambridge: Cambridge University Press.

London, W., & Yorke, J. (1975). Recurrent outbreaks of measles, chickenpox, and mumps. 1. Seasonal variation in contact rates. *Am. J. Epidem.* 98, 453–468.

Martinat, P.J. (1987). The role of climatic variation and weather in forest insect outbreaks. *Insect Outbreaks*, eds P. Barbosa & J.C. Schultz. London: Academic Press, pp. 3–30.

May, R.M. (1981). Models for single populations. *Theoretical Ecology*, ed. R.M. May. Oxford: Blackwell, pp. 5–29.

Monath, T.P. (1993). Arthropod-borne viruses. In *Emerging Viruses*, ed. S.S. Morse. New York: Oxford University Press, pp. 138–148.

Myers, J.H. (1998). Synchrony in outbreaks of forest lepidoptera: a possible example of the Moran Effect. *Ecol.*, 79, 1111–1117.

Nicolis, C. (1982). Stochastic aspects of climatic transitions–response to a periodic forcing. *Tellus*, 34, 1–9.

Nisbet, R.M. & Gurney, W.S.C. (1982). *Modelling Fluctuating Populations*. Chichester: John Wiley.

Press, W.H., Flannery, B.P., Teukolsky, S.A. & Vetterling, W.T. (1990). *Numerical Recipes in C–the Art of Scientific Computing*. Cambridge: Cambridge University Press.

Rand, D.A. & Wilson, H.B. (1991). Chaotic stochasticity–a ubiquitous source of unpredictability in epidemics. *Proc. Royal Soc. London B*, 246, 179–184.

Reeves, W.C. (1974). Overwintering of arboviruses. *Prog. Med. Virol.*, 17, 193–220.

Schove, D.J. (1983). *Sunspot Cycles*. Stroudsburg, Pennsylvania: Hudchinson Ross Publishing.

Schwartz, I.B. (1985). Multiple stable recurrent outbreaks and predictability in seasonally forced nonlinear epidemic models. *J. Mathemat. Biol.*, 21, 347–361.

Simpson, G.M. (1990). *Seed Dormancy in Grasses*. Oxon: CAB International.

Stone, L., Saparin, P.I., Huppert, A. & Price, C. (1998). El Nino chaos: The role of noise and stochastic resonance on the ENSO cycle. *Geophys. Res. Lett*, 25, 175–178.

Tauber, M.J., Tauber, C.A. & Masaki, S. (1986). *Seasonal Adaptations of Insects*. New York: Oxford University Press.

Tuljapurkar, S. (1990). Delayed reproduction and fitness in variable environments. *Proc. Natl. Acad. Sci. USA.*, 87, 1139–1143.

Tuljapurkar, S. & Istock, C.A. (1993). Environmental uncertainty and variable diapause. *Theoret Population Biol.*, 43, 251–280.

Walker, J.S. (1996). *Fast Fourier Transforms*, 2nd edn. Boca Raton: CRC Press.

Watt, A.D., Leather, S.R., Hunter, M.D. & Kidd, N.A.C. (1990). *Population Dynamics of Forest Insects*. Andover: Intercept.

Watts, D.M., Burke, D.S., Harrison, B.A., Whitmire, R.E. & Nisalak, A. (1987). Effect of temperature on the vector efficiency of Aedes aegypti for Dengue 2 virus. *Am. J. Trop. Med. Hygiene*, 36, 143–152.

Wiesenfeld, K. & Moss, F. (1995). Stochastic resonance and the benefits of noise: from ice ages to crayfish and SQUIDs. *Nature*, 373, 33–36.

Yodzis, P. (1989). Introduction to *Theoretical Ecology*. New York: Harper & Row.

Yoshimura, J. & Clark, C.W., eds (1993). *Adaptation in Stochastic Environments*. Berlin: Springer.

Zhou, T. & Moss, F. (1990). Analog simulations of stochastic resonance. *Phys. Rev. A*, 41, 4255–4264.

BIOSKETCH

Albert Blarer is a PhD student at the Zoology Institute, University of Basel. His main research interests are population dynamics in general, with special focus on the effects of noise on nonlinear dynamics. His broad interests are in evolutionary ecology and life history theory.

Editor, A. Sasaki
Manuscript received 21 December 1998
First decision made 27 January 1999
Manuscript accepted 19 February 1999

©1999 Blackwell Science Ltd/CNRS