Predator-Prey Cycles from Resonant Amplification of Demographic Stochasticity

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We present the simplest individual level model of predator-prey dynamics and show, via direct calculation, that it exhibits cycling behavior. The deterministic analogue of our model, recovered when the number of individuals is infinitely large, is the Volterra system (with density-dependent prey reproduction) which is well known to fail to predict cycles. This difference in behavior can be traced to a resonant amplification of demographic fluctuations which disappears only when the number of individuals is strictly infinite. Our results indicate that additional biological mechanisms, such as predator satiation, may not be necessary to explain observed predator-prey cycles in real (finite) populations.

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Predator-prey cycles are one of the most striking phenomena observed in population biology, and as such, inspire intense discussion among ecologists [1]. Cycles are also seen in a wide variety of other ''host-natural enemy'' systems such as host-pathogen [2] systems; one of the most well known examples is measles, epidemics of which have been studied for many years [3]. In this Letter, we will be concerned with modeling the phenomenon of cycles, and will focus on predator-prey systems, for concreteness, but our main results will have direct applicability to other host-natural enemy systems, since they can be modeled in a similar way, often using identical equations. We also believe that, since the phenomenon we describe is quite generic in certain classes of stochastic systems, it should be found outside population dynamics. It seems that the precise mechanism underlying the existence of the cycles has not so far been elucidated because it involves the analysis of stochastic systems with a large, but finite, number of constituents, and also because it involves concepts such as resonance, which are more familiar to physicists than biologists.

Among the numerous hypotheses put forward to explain cycles, perhaps the simplest is that cycles arise directly from predator-prey interactions. Within this conceptual framework, theoretical modeling of cycles has traditionally been developed using deterministic population-level models (PLMs). Such discussions begin with Volterra-like equations, which are coupled differential equations for the predator and prey densities. Equations of this type encapsulate the simplest processes of predator and prey mortality, prey reproduction and competition, and predation. Surprisingly these models do not predict stable cycles: additional biological mechanisms, such as predator satiation, need to be included within the framework of differential equations to give cycles [4]. It seems puzzling that cycles, which are so easy to understand intuitively, can only be described mathematically in models which include these more subtle mechanisms. In order to probe this issue, we shall take a different approach here and describe the predator-prey system using an individual level model (ILM). The individuals, which are either predators or prey, are acted upon by simple stochastic processes of mortality, reproduction, and predation. We are able to derive an exact description of this model when the number of individuals is large and finite. We find that the predator and prey numbers undergo large cycles, just as one would expect intuitively. The cycles, which arise from a novel resonance effect, disappear only when the number of individuals is taken to be strictly infinite, that is, when the PLM is recovered. From a statistical physics viewpoint, we would term the PLM a mean field theory of the underlying ''microscopic'' ILM which includes statistical fluctuations.

Predator-prey cycles observed in nature will have a stochastic component—this will affect both their amplitude and phase. Therefore, care must be taken in averaging over replicates. A direct average of the population densities from different replicates will result in a constant average density since, in the absence of an external ''forcing,'' there is a lack of synchrony between the cycles from different replicates. This fact is crucial when modeling predator-prey cycles. A given PLM is written in terms of a population density, which can be thought of as the result of an average of the population numbers from a large number of ILM replicates. If a given ILM shows oscillatory behavior, such cycles will be lost in the modeling transition to a PLM. Thus, it is necessary to study quantities, such as the autocorrelation function and power spectrum arising from an ensemble of ILMs, in order to determine the presence and properties of predator-prey cycles.

The specific ILM we study in this Letter is a nonspatial stochastic model. At a given time, a realization of the ILM consists of n individuals of species A (the predators) and m individuals of species B (the prey). Since we are interested in what is essentially the simplest model of predator-prey interactions, we include only birth processes $BE \xrightarrow{b} BB$,

death processes $A \xrightarrow{d_1} E$, $B \xrightarrow{d_2} E$, and predator-prey interactions $AB \xrightarrow{p_1} AA$, $AB \xrightarrow{p_2} AE$. Here (b, d_1, d_2, p_1, p_2) are rate constants. The symbol E corresponds to what would be available sites in a spatial model. In this nonspatial model, the E's are $(N - n - m)$ passive constituents of the system, which are required for prey reproduction, and which result in intraspecific prey competition. Note, the overall number of A, B, and E constituents is fixed to be N. The dynamics of the model can either be numerically simulated or studied analytically using the formalism of master equations [5,6]. Simulations have been performed using two different algorithms: the first consists of making small increments in time (in our case $\delta t = 0.05$) and within each increment choosing constituents at random and implementing the rules given above; the second follows Gillespie's exact algorithm [7] in which one of the processes is enacted according to its relative statistical weight, and time is incremented by an amount drawn from the appropriate exponential distribution. We have found excellent agreement between the results from both algorithms. Gillespie's algorithm is superior in that it is exact and highly efficient. In constructing the master equation, the transition rates $T(n', m'|n, m)$ from the state (n, m) to the state (n', m') are given by

$$\begin{aligned} T(n-1, m|n, m) &= d_1 n, \\ T(n, m+1|n, m) &= 2b\frac{m}{N}(N-n-m), \\ T(n, m-1|n, m) &= 2p_2\frac{nm}{N} + d_2 m, \\ T(n+1, m-1|n, m) &= 2p_1\frac{nm}{N}, \end{aligned} \tag{1}$$

where the b and p_i have been scaled by a factor of $(N - 1)$ and the d_i by a factor of N. We have already given an extensive discussion of this approach elsewhere in the context of competition models [8], and we refer the reader to this paper for a fuller discussion of the formalism.

The master equation for the probability that the system consists of n predators and m prey at time t, $P(n, m, t)$, is

$$\begin{aligned} \frac{dP(n,m,t)}{dt} &= (\mathcal{E}_x - 1)[T(n-1, m|n, m)P(n, m, t)] \\ &\quad + (\mathcal{E}_y^{-1} - 1)[T(n, m+1|n, m)P(n, m, t)] \\ &\quad + (\mathcal{E}_y - 1)[T(n, m-1|n, m)P(n, m, t)] \\ &\quad + (\mathcal{E}_x^{-1}\mathcal{E}_y - 1)[T(n+1, m-1|n, m)P(n, m, t)], \end{aligned} \tag{2}$$

where the step operators $\mathcal{E}$ are defined by their actions on functions of n and m by $\mathcal{E}_x^{\pm 1} f(n, m, t) = f(n \pm 1, m, t)$ and $\mathcal{E}_y^{\pm 1} f(n, m, t) = f(n, m \pm 1, t)$.

The mean field limit of this ILM may be obtained by multiplying (2) by n and m in turn, and subsequently summing over all allowed values of m and n. This gives equations for the mean values $f_1 = \langle n \rangle / N$ and $f_2 = \langle m \rangle / N$ in the limit $N \to \infty$ if we ignore terms which are $1/N$ down on others and make the replacements $\langle m^2 \rangle \to \langle m \rangle^2$ and $\langle mn \rangle \to \langle m \rangle \langle n \rangle$. This mean field theory, or PLM, takes the form

$$\begin{aligned} \frac{df_1}{dt} &= n(f_2) f_1 - \mu f_1 \\ \frac{df_2}{dt} &= r f_2 \left(1 - \frac{f_2}{K}\right) - g(f_2) f_1. \end{aligned} \tag{3}$$

The Eqs. (3) are frequently referred to as the Volterra equations, to distinguish them from the Lotka-Volterra equations which have no term in f_2/K [5]. The constants μ, r, and K are simply functions of the rate constants:

$$\mu = d_1, \qquad r = 2b - d_2, \qquad K = 1 - \frac{d_2}{2b}, \tag{4}$$

and the linear numerical and functional responses are given by $n(f_2) = 2p_1 f_2$ and $g(f_2) = 2(p_1 + p_2 + b) f_2$.

As is well known [5], the analysis of this model shows a complete absence of cycles. There is a single fixed point for which the predators and prey have nonzero population sizes. Denoting these stationary values by $f_1^{(s)}$ and $f_2^{(s)}$, then in terms of the original rate constants they are given by:

$$f_1^{(s)} = \frac{(2bp_1 - bd_1 - p_1 d_2)}{2p_1(p_1 + p_2 + b)}, \qquad f_2^{(s)} = \frac{d_1}{2p_1}. \tag{5}$$

The stability of this fixed point may be studied by performing linear stability analysis. This results in a stability

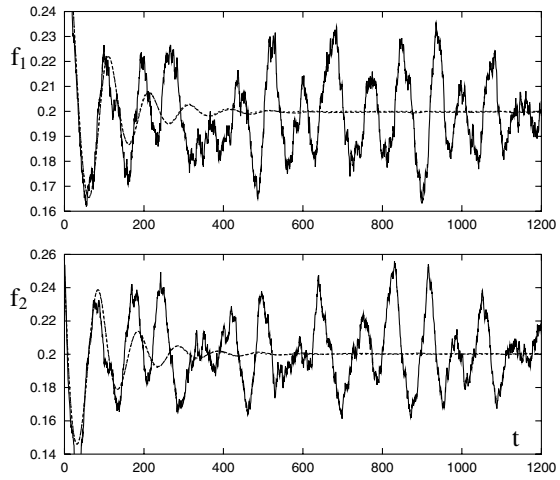

FIG. 1. Predator and prey densities as a function of time. The upper panel shows the predator density f_1 for $N = 3200$. The dashed line is calculated from numerical integration of the mean field Volterra equations (3). The dotted line is the average of the predator density time series from 10 000 replicates generated from the ILM and is almost indistinguishable from the mean field solution. The solid line is the predator density time series for a single typical replicate. The lower panel is the equivalent plot for the prey density f_2. Parameter values are $b = 0.1$, $d_1 = 0.1$, $d_2 = 0.0$, $p_1 = 0.25$, and $p_2 = 0.05$.

matrix which has entries given by $a_{11} = 0$, $a_{12} = 2p_1 f_1^{(s)}$, $a_{21} = -2(p_1 + p_2 + b)f_2^{(s)}$, and $a_{22} = -2bf_2^{(s)}$. We have expressed the entries in terms of the fixed point values, since these are manifestly positive, and it is easy to see that the eigenvalues of the matrix both have a negative real part, implying that the fixed point is stable. While there is no limit cycle in the Volterra system (3), a limit cycle does exist in the Lotka-Volterra equations (obtained by taking $K \to \infty$), but it is neutrally stable due to a conserved quantity in the model. This unrealistic behavior disappears with the introduction of a finite carrying capacity, K, in (3), but, as mentioned above, leads to a complete absence of cycling behavior. Only with the introduction of other mechanisms, such as predator satiation where the functional response $g(f_2)$ has a more complex functional form which does not grow linearly for all f_2, are stable limit cycles observed [4]. We will show below that cycles can be found in the ILM, but only when N is finite; the $N \to \infty$ limit which was taken in order to derive the PLM, eliminates the cycles present in the original ILM.

We first note that the ensemble averaged population density of the ILM, determined from numerical simulations, agrees beautifully with the solution of this deterministic model (Fig. 1, dashed and dotted lines, respectively) showing a decaying oscillatory transient followed by a constant steady-state density, typical of a Volterra system. In marked contrast, individual realizations of the ILM show large persistent cycles (Fig. 1, solid line). The amplitude of these oscillations is much larger than the naïve estimate based on the law of large numbers. In fact, the oscillations are of order $(1/\sqrt{N})$ as would be expected, but amplified by a very large factor due to a noise-induced resonance effect, as explained below.

This cycling behavior can be investigated analytically by extracting an ''effective theory'' valid for large N, through applying a standard method to the master equations, due to van Kampen [6]. Essentially the method involves the replacements $n/N = f_1 + x/\sqrt{N}$ and $m/N = f_2 + y/\sqrt{N}$ in the transition probabilities that appear in the master equation. By changing from a description based on the (discrete) variables n and m to one based on the (continuous) variables x and y, terms of different orders in $1/N$ can be identified in the master equation: the leading order terms give rise to a deterministic set of equations and the next-to-leading order terms give rise to a linear Fokker-Planck equation. The leading order set of equations (mean field theory) are the PLM (3), which we have already obtained by a more direct method.

At next-to-leading order, rather than write down the Fokker-Planck equation, it is simpler to give the set of Langevin equations to which it is equivalent [6]. They take the form

$$\dot{x} = a_{11}x + a_{12}y + \eta_1(t) \quad \dot{y} = a_{21}x + a_{22}y + \eta_2(t). \quad (6)$$

These are a pair of differential equations which describe the stochastic behavior of the ILM at large N: $x(t)$ and $y(t)$ are stochastic corrections to the deterministic behavior of the predator and prey densities, respectively, at large but finite N. The constants, a_{ij}, appearing in Eq. (6), are exactly the entries of the stability matrix found from a linear stability analysis about the nontrivial fixed point of Eq. (3). The noise covariance matrix b_{ij}, which is responsible for generating the large-scale oscillations, cannot be determined from Eq. (3) and is derived from the master equation using the van Kampen expansion. Since the noise is white, $b_{ij} = \langle \tilde{\eta}_i(\omega)\tilde{\eta}_j(-\omega)\rangle$ is independent of the frequency ω. The explicit expressions for these constants are $b_{11} = 2d_1 f_1^{(s)}$, $b_{12} = b_{21} = -d_1 f_1^{(s)}$, and $b_{22} = 2d_1(1 + p_2/p_1)f_1^{(s)} + 2d_2 f_2^{(s)}$. It is not the average behavior of replicates that interests us, but rather, measures which characterize the oscillations. Examining x and y as functions of frequency allows us to determine the nature of the oscillations.

To search for oscillations in noisy data, one of the most useful diagnostic tools is the power spectrum $P(\omega) = \langle|\tilde{x}(\omega)|^2\rangle$, where $\tilde{x}(\omega)$ is the Fourier transform of $x(t)$. Taking the Fourier transform of Eq. (6), solving for $\tilde{x}(\omega)$, and averaging its squared modulus, we find

$$P(\omega) = \frac{\alpha + \beta\omega^2}{[(\omega^2 - \Omega_0^2)^2 + \Gamma^2\omega^2]}, \quad (7)$$

where α and β are functions of the ILM rates: $\alpha = b_{11}a_{22}^2 + 2b_{12}a_{12}|a_{22}| + b_{22}a_{12}^2$ and $\beta = b_{11}$. The constants in the denominator have the especially simple forms $\Omega_0^2 = a_{12}|a_{21}|$ and $\Gamma = |a_{22}|$. The spectrum predicted by Eq. (7) gives the solid line shown in Fig. 2. The agreement with the spectrum obtained from simulation of the ILM (noisy line) is excellent. Note, the naïve $O(1/\sqrt{N})$ estimate

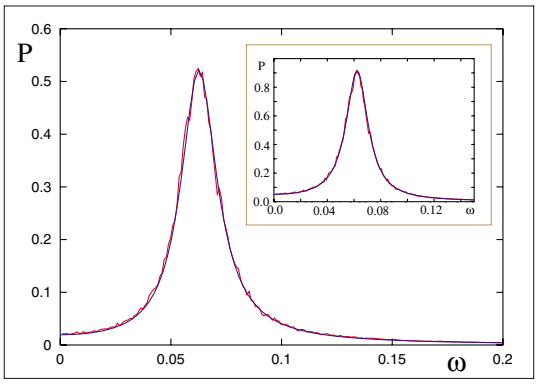

FIG. 2 (color online). A plot of the power spectrum $P(\omega)$ for the predator time series as a function of frequency ω. The slightly noisy line corresponds to P calculated from 500 replicate runs of the ILM. The smooth line is the prediction from our theory, namely, Eq. (7). The parameter values are the same as those described in the caption to Fig. 1. The inset shows the analogous power spectra (data and theory) for the prey time series.

of the size of stochastic fluctuations corresponds to the zero frequency value of $P(\omega)$. Figure 2 clearly illustrates the very large amplification of these fluctuations due to the resonance effect.

The spectrum given above is reminiscent of that for a simple mechanical system—namely a linear damped harmonic oscillator, with natural frequency Ω_0 and driven at frequency ω. In a mechanical oscillator the driving frequency must be tuned to achieve resonance. In the stochastic predator-prey model described here no tuning is necessary. The system is driven by white noise, as shown in Eq. (6), which covers all frequencies—thus the resonant frequency of the system is excited without tuning. We stress that the noise which drives the system is *not external* but arises from the demographic stochasticity contained in the individual processes which define the model; there is no environmental stochasticity in our model. Furthermore, the resonance phenomenon we report here is not related to ''stochastic resonance.''

The damping term, represented by the constant Γ, limits the amplitude of the oscillations. Predator-prey systems for which Γ happens to be small will be at risk of extinction through resonant oscillations, despite having large population sizes. The resonant oscillation occurs in the regime $2a_{12}|a_{21}| > a_{22}^2$, where the resonant frequency $\omega_0 = \sqrt{\Omega_0^2 - \Gamma^2/2}$ is real. A similar analysis can be carried out to obtain the spectrum for the prey time series. It again has the form Eq. (7), but now with $\alpha = b_{11}a_{21}^2$ and $\beta = b_{22}$. The positions of the peaks for these two power spectra are only weakly dependent on the α's and β's, and so they are almost coincident.

Predator-prey systems (and related host-pathogen systems) have been studied theoretically for decades. Most of the previous studies have focused on the role of environmental stochasticity, the relevance of nonlinear interactions or of spatial effects, to explain the mechanism of cycling [5,9]. Some authors have discussed the role that demographic stochasticity may have on cycles [5,10], and physicists have investigated the effects of internal noise [11]. Most of this discussion has been qualitative; the nearest to our own discussion was a prescient analysis by Bartlett nearly 50 years ago [10], in which he postulated equations similar to (6). However, he did not note the existence of a resonance, and so proposed cycles with an amplitude which were not enhanced by this effect and which were therefore of limited biological interest.

The idea that external perturbations with a dominant frequency can entrain the predator-prey dynamics in a cyclic nature is fairly intuitive; the phenomenon we discuss here is more fundamental and less intuitive. The noise in our system is internal—purely a result of the demographic stochasticity inherent in discrete birth, death, and predation events. These excitations are typically of limited interest in a large population of N individuals, since they give rise to small $O(1/\sqrt{N})$ fluctuations about the mean population densities. Such is the case, for example, in competition models [8]. The predator-prey system, and related ones such as epidemic models, are exceptional in that the equations describing linear fluctuations about the steady-state are susceptible to resonant amplification in the vicinity of an internal frequency Ω, which is a property of the population itself. The internal noise, in exciting all frequencies, automatically resonates the system giving rise to large oscillations in the population densities. We expect that this resonance mechanism will occur in other stochastic systems in which the mean field theory shows damped oscillations, for instance, biochemical reactions in microscopic systems [12].

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