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PREY ADAPTATION AS A CAUSE OF PREDATOR-PREY CYCLES

PETER A. ABRAMS[1] AND HIROYUKI MATSUDA[2]
[1]*Department of Zoology, University of Maryland, College Park, Maryland 20742*
[2]*Population Dynamics of Marine Organisms, Ocean Research Institute, University of Tokyo, 1-15-1 Minamidai, Nakano-ku, Tokyo 164, Japan*

Abstract.—We analyze simple models of predator-prey systems in which there is adaptive change in a trait of the prey that determines the rate at which it is captured by searching predators. Two models of adaptive change are explored: (1) change within a single reproducing prey population that has genetic variation for vulnerability to capture by the predator; and (2) direct competition between two independently reproducing prey populations that differ in their vulnerability. When an individual predator's consumption increases at a decreasing rate with prey availability, prey adaptation via either of these mechanisms may produce sustained cycles in both species' population densities and in the prey's mean trait value. Sufficiently rapid adaptive change (e.g., behavioral adaptation or evolution of traits with a large additive genetic variance), or sufficiently low predator birth and death rates will produce sustained cycles or chaos, even when the predator-prey dynamics with fixed prey capture rates would have been stable. Adaptive dynamics can also stabilize a system that would exhibit limit cycles if traits were fixed at their equilibrium values. When evolution fails to stabilize inherently unstable population interactions, selection decreases the prey's escape ability, which further destabilizes population dynamics. When the predator has a linear functional response, evolution of prey vulnerability always promotes stability. The relevance of these results to observed predator-prey cycles is discussed.

Key words.—Antipredator traits, chaos, cycles, predation, predator-prey dynamics, runaway evolution, stability.

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Traditionally, evolution has been thought to affect the stability of predator-prey systems by moving the values of population dynamic parameters into or out of regions where the population dynamics causes cycles (Rosenzweig 1973; Rosenzweig and Schaffer 1978; Schaffer and Rosenzweig 1978; Oksanen 1992). Prey evolution has usually been thought to enhance stability (Rosenzweig 1973). Behavioral change by prey has also been shown to be stabilizing in the context of at least one model (Ives and Dobson 1987). These results stand in contrast to recent models of adaptive change in continuous traits of either predators (Abrams 1992) or both predator and prey species (Saloniemi 1993; Dieckmann et al. 1995; Abrams and Matsuda 1997). These more recent models show that either predator evolution or predator-prey coevolution can cause population cycles. The present article builds upon a model introduced by Matsuda and Abrams (1994a), and shows that adaptive change in capture-related traits of the prey can also generate cycles. The presence of a saturating functional response in the predator is essential for the mechanism described here. We also show that prey adaptation can result in a runaway decrease in the prey's escape ability when the population interaction is responsible for the cycles. These results argue against the generalization that prey adaptation stabilizes predator-prey systems.

HOW PREY EVOLUTION CAN GENERATE INSTABILITY: QUALITATIVE ARGUMENTS

We consider adaptive change of prey traits that affect the rate at which they are caught by predators. We refer to this as evolution of prey vulnerability, although the model and the mechanism it illustrates also apply to other types of adaptive change. Evolution in prey species has generally been thought to be stabilizing in the context of single prey-single predator systems. The logic behind this idea is as follows. When predators are very abundant, selection favors decreased vulnerability in the prey, which seemingly should reduce predator abundance. Conversely, when predators are rare, the prey should evolve greater vulnerability, assuming there is a pleiotropic cost to reduced vulnerability. These responses tend to restore predator abundance. More generally, prey evolution is thought to move the predator isocline to the right (i.e., toward greater prey densities; Rosenzweig 1973), which is also often stabilizing. These same arguments suggest that other adaptive processes, such as behavior, will have similar stabilizing effects (Ives and Dobson 1987). There are certainly some situations in which evolution or another adaptive process in the prey stabilizes predator-prey systems (as is shown below). However, the intuitive arguments for stabilization neglect the increase in predator's level of satiation that accompanies increased prey vulnerability. The greater predator satiation causes the prey to experience a lowered risk of predation, which favors still further decreases in vulnerability. This positive-feedback process should be capable of generating instability.

In the following section, we use a series of simple models to show how these considerations can lead to evolutionary destabilization of predator-prey systems when the prey's vulnerability to capture evolves in response to both predation pressure and pleiotropic effects on other components of prey fitness. The destabilizing force in all of the models is provided by the decelerating rate of increase of the predator's prey capture rate (its functional response) as prey availability (vulnerability $\times$ abundance) rises. The models investigate what conditions are required for this destabilizing force to generate sustained cycles or chaos in population dynamics.

The following analysis considers two classes of models. In the first, adaptation occurs via genetic change in a quantitative trait in the prey that determines its vulnerability. We derive conditions for local stability of the equilibria of this system for a model with unspecified functional components. We then numerically investigate the dynamics of unstable versions of a particular example. The second case analyzed

is a specific model in which adaptation occurs via competition between two independently reproducing prey types. It is shown that the dynamics of this system are very similar to those of the genetic model. The discussion considers the relationship between these and previous models of the evolution or adaptation of prey traits. We also discuss the applicability of these ideas to observed predator-prey cycles.

Models and Stability Analyses

All of the models analyzed here are systems of ordinary differential equations describing predator and prey population dynamics, with population densities P and N, respectively. The prey population is assumed to have variation in a trait that determines vulnerability to capture by predators. Vulnerability is measured by the parameter C, the per-prey rate at which an unsatiated predator individual captures prey with the trait value corresponding to C. The two families of models considered below differ in their assumptions about the form of the variability in C within the prey population. In models of the first type, we assume a distribution of prey phenotypes with a generally fixed shape, whose mean value, C^*, changes at a rate proportional to the derivative of individual fitness with respect to the trait value. (The trait distribution is, however, assumed to become narrower as the mean trait value approaches lower and upper limits.) In the second group of models, there are two prey phenotypes that reproduce independently and have fixed, but different C-values.

In both types of model, decreases in vulnerability are assumed to decrease prey fitness in the absence of predation. Thus, a prey individual's per capita growth rate, $r(C)$ is an increasing function of vulnerability. In the first set of models, the prey population's mean growth rate is assumed to be approximately $r(C^*)$, the growth rate of an individual with the average trait value, C^*. In the second set of models, the prey type with the larger C has a larger r. In the continuous trait models, $r(C)$ is assumed to be linear or decelerating ($d^2r/dC^2 \equiv r'' \leq 0$). This is reasonable because reproductive rate cannot continue to increase indefinitely with increasing vulnerability. In both models, the per capita growth rate of the prey is reduced by density-dependent interactions; this reduction is described by an increasing function of total prey population, $f(N)$. The realized capture rate by a searching predator is reduced because of handling time or satiation; the proportional reduction is a decreasing function, $g(C^*N)$, of the predators' capture rate while searching. (In the model with two prey phenotypes, $C^*N = C_1N_1 + C_2N_2$.) The satiation function g approaches one as C^*N approaches zero, and the functional response, $C^*Ng(C^*N)$, is assumed to increase with C^*N. In the limiting case of no satiation (i.e., a linear predator functional response) $g = 1$. The predator's per capita growth rate is an increasing function, denoted b, of its functional response. In both models, the prey's individual fitness is given by $w(C, C^*) = r(C) - f(N) - CPg(C^*N)$. The system is completed by describing the rate of change of mean vulnerability, which is implicitly determined by the population dynamics of the two types in the second set of models.

Case I: Evolutionary Models

The trait dynamic described below is based on an approximation for quantitative traits influenced by many loci having small additive effects. Prey fitness is frequency dependent because predator satiation depends on the mean vulnerability of the prey population. Because of this frequency dependence, the dynamics of the capture rate parameter is described using an approximation to Lande's (1976) quantitative genetic recursions developed in Iwasa et al. (1991), Taper and Case (1992), and Abrams et al. (1993a). Under this approach, the mean trait value is assumed to change at a rate proportional to the individual fitness gradient; if $w(C, C^*)$ is the per capita growth rate of an individual with trait C in a population with mean trait C^*, dC^*/dt is proportional to $\partial w/\partial C$ evaluated at $C = C^*$. This approximation often requires the assumption that the phenotypic variance of the trait is relatively small, but it is valid for large variances provided that the fitness function is a linear or quadratic function of the individual's trait value (Abrams et al. 1993a). This gradient dynamics model can also describe adaptation via development or behavior (Abrams et al. 1993b), and a modified fitness function can be used to model large phenotypic variances or highly nonlinear fitness functions (Abrams et al. 1993a). The same dynamic equation arises from models in which variation is generated by mutation (Vincent et al. 1993; Dieckmann et al. 1995), and has simply been assumed by other authors (Rosenzweig et al. 1987; Getz 1993). The rate of change of C^* is also proportional to the additive genetic variance, which is described here by the function $v(C^*)$. In most of our simulations this function is assumed to be approximately constant, except when C^* approaches minimum or maximum values, in which case v approaches zero. The approximate dynamics of populations and mean trait value are:

$$dP/dt = Pb[C^*Ng(C^*N)], \tag{1a}$$

$$dN/dt = N[r(C^*) - f(N) - C^*Pg(C^*N)], \tag{1b}$$

and

$$dC/dt = v(C^*)[dr/dC|_{C=C^*} - Pg(C^*N)], \tag{1c}$$

where the quantities in brackets in eqations (1b,c) are respectively, $w(C, C^*)$ and $\partial w(C, C^*)/\partial C$, both evaluated at $C = C^*$. In most of the following simulations, we assume there exists a minimum possible C, but no maximum. More details on the derivation and meaning of equation (1c) are given in Abrams et al. (1993a,b), Matsuda and Abrams (1994a,b), and Abrams and Matsuda (1997).

The Appendix describes the local stability analysis of equations (1a–c). Here, we are interested in two questions. (1) Can adaptative change of the capture rate parameter C^* stabilize a predator-prey system that would exhibit limit cycles in the absence of adaptive change? (2) Can adaptative change of the capture rate parameter C^* destabilize a predator-prey system that would be stable in the absence of adaptive change? If the answer to question (1) is negative or (2) is affirmative, we must determine what types of population dynamics result. We can answer both questions using the results presented in the Appendix. The local stability of the nonevolutionary predator-prey system is determined by the quantity, $f' + C^{*2}Pg'$. Positive values imply local stability, negative

values imply instability. Thus a satiating functional response ($g' < 0$) is necessary for population dynamic instability. The evolutionary system without population dynamics has a stable equilibrium at an intermediate vulnerability if and only if $g'NP - r'' > 0$ at that equilibrium. Given that $r'' \leq 0$, it is clear that the evolutionary dynamics in isolation must be stable when there is no predator satiation ($g = 1$; $g' = 0$). On the other hand, a sufficiently high degree of satiation will destabilize the trait dynamics. Expressions (A1a,b) in the Appendix show that a negative r'' sufficiently large in magnitude will result local stability of the entire system (eqs. [1a–c]); this shows that trait dynamics can stabilize population dynamics. Inequality (A1a) also shows that a sufficiently large genetic variance will cause unstable trait dynamics to destabilize the entire system.

Three aspects of this general stability analysis should be noted. First, predator satiation is necessary for instability when population dynamics are stable. This may be verified by substituting $g' = 0$ in the stability conditions (A3) in the Appendix. This result implies that, when the predator's functional response is linear, evolutionary change cannot cause cycles. This same result establishes a second conclusion: that cycles cannot be attributed to the time lag that results from the noninstantaneous nature of prey evolution. If there is no predator satiation ($g' = 0$), our assumption that $r'' \leq 0$ guarantees that the equilibrium of equations (1a–c) will always be locally stable, regardless of the speed of the evolutionary response, which is scaled by v. Finally, the instability of the equilibrium does not result from disruptive selection on the prey's vulnerability. Disruptive selection on vulnerability has been shown to be capable of generating cycles in the context of coevolutionary models (Abrams and Matsuda 1997). However, the assumption $r'' \leq 0$ guarantees that selection in the current model is either stabilizing or neutral at the equilibrium point. The instability is generated by the saturation of the predator's functional response ($g' < 0$), which can make the evolutionary equilibrium convergence unstable, even though it maximizes individual fitness (Christiansen 1991; Abrams et al. 1993b). Unfortunately, the local stability analysis of this general model provides limited insight into the effects of particular environmental parameters on stability and global dynamics. These can be clarified by a simulation analysis of a specific model.

Our analysis concentrates on the algebraically simplest model that can be constructed under the basic assumptions outlined above. The prey has logistic growth in the absence of the predator ($f[N] = kN$, where k is a constant). The predator has a disk equation (Holling 1959) functional response ($g = 1/[1 + hC^*N]$), where h is handling time). The predator also has a linear numerical response, so $b(C^*Ng) = BC^*N/(1 + hC^*N) - d$. A prey individual's maximum growth rate increases linearly as its capture rate constant, C, increases, $r(C) = R + qC$. Because $r'' = 0$, trait dynamics in isolation from population dynamics are always unstable; this form of $r(C)$ is best regarded as an approximation to a model with a negative quadratic term (see eq. [4]). The variance function is $v(C^*) = V_0\text{Exp}(-s/[C^* - s])$, were s is a small constant (10^{-3} in most of the following simulations), and V_0 is the approximate genetic variance when C^* is significantly greater than s. This function prevents the consumption rate from becoming less than s. The form of $v(C^*)$ does not change the local stability of the equilibrium (Abrams et al. 1993b), but can have a major effect on the nature of cycles. The fitness of a prey individual with trait value C in a population with mean trait value C^* is a linear function of C, that is, $w(C, C^*) = R + qC - kN - CP/(1 + hC^*N)$. The linearity of this function means that the individual fitness gradient ($\partial w/\partial C$) is identical for all phenotypes in the population, and dynamics near equilibrium do not depend on any assumptions about the distribution of phenotypes. These assumptions result in the following set of equations:

$$dP/dt = P[BC^*N/(1 + hC^*N) - d] \quad (2a)$$
$$dN/dt = N[R + qC^* - kN - C^*P/(1 + hC^*N)] \quad (2b)$$
$$dC^*/dt = V_0\text{Exp}[-s/(C^* - s)][q - P/(1 + hC^*N)]. \quad (2c)$$

At the single equilibrium point with positive values of all variables, the population densities and the mean trait value are:

$$P_e = Bq/(B - dh) \quad (3a)$$
$$N_e = R/k \quad (3b)$$
$$C^*_e = kd/[R(B - dh)]. \quad (3c)$$

It is interesting to note that the predator's equilibrium density is independent of two of the growth parameters of the prey (R, k), while the prey's equilibrium density is independent of the population growth parameters of the predator. If there were no adaptation, prey density would be independent of R and k, but would depend on predator growth parameters. Similarly, predator density would be affected by all of the prey growth parameters. The model given by equations (2a–c) assumes that the prey's maximum growth rate can continue to increase indefinitely as its vulnerability increases. This relationship must break down at sufficiently high vulnerabilities, in which case either: (1) $r(C)$ can be assumed to be unimodal or to have an upper asymptote; or (2) an upper limit to C can be imposed by assuming that the adaptive rate function, $v(C^*)$, approaches zero as the capture rate approaches that maximum. Some simulations with assumption (1) are discussed below.

The full conditions for local stability are given by inequalities (A3) in the Appendix. The equilibrium specified by equations (3a–c) can always be destabilized by sufficiently large changes of any of the following types: (1) increases in the adaptation rate parameter V_0; (2) proportional decreases in predator vital rates, B and d; (3) decreases in the prey's density dependence, k; (4) decreases in the prey's per capita growth parameter R; or (5) increases in the cost of lower vulnerability, q. It is appropriate to attribute sustained nonequilibrium dynamics of the entire system to adaptive trait dynamics if a system consisting of equations (2a,b) with C^* fixed at its equilibrium value is stable, but the system with equation (2c) added is unstable. Equation (A4) in the Appendix gives the condition for the population dynamic system with C^* fixed at its adaptive equilibrium value to have a locally stable equilibrium point. Of the five destabilizing parameters mentioned above, the rate of adaptation, V_0, and predator vital rates (B and d) do not affect population dynamic stability. When a set of parameters yields instability in the model with adaptively changing traits, but stability with fixed traits, the population fluctuations can be said to be driven by

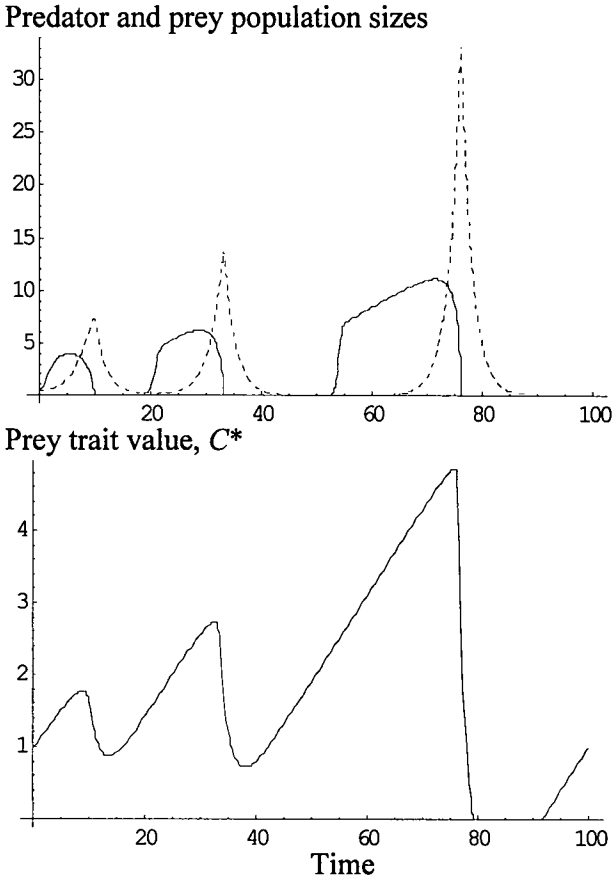

FIG. 1. Unstable population dynamics of equations (2a–c) when the cycles are driven by population dynamics. The top plot shows predator (dashed line) and prey (solid line) densities, and the bottom plot shows the mean prey trait value, C^*. This system exhibits divergent oscillations with a monotonically increasing minimum and maximum values of all three variables. The parameter values are: $h = 1$, $B = 1$, $q = 1.2$, $d = 0.5$, $V_0 = 0.1$, $R = 0.5$, $k = 0.5$, $s = 0.001$.

the adaptive dynamics; sufficient increases in V_0 or decreases in B and d will always destabilize a system with nonzero handling time and population dynamic stability. Because $r(C)$ is linear, trait dynamics can never stabilize a system with unstable population dynamics in this example.

The remainder of this section describes the three qualitatively distinct types of dynamics that were observed in an extensive set of simulations based on the local stability analysis.

1. Runaway Evolution of Increasing C for All Initial Conditions*

This outcome occurs when the equilibrium point given by equations (3a–c) is locally unstable, and the cycles are driven by population dynamics. Simulations of a large number of such systems have shown that the cycles associated with such an equilibrium are of ever-increasing period and amplitude and are associated with an oscillatory increase in the mean vulnerability, C^*. Figure 1 is a typical example of such cycles, which imply extinction of the predator or of both species. The reason for the expanding oscillations can be seen

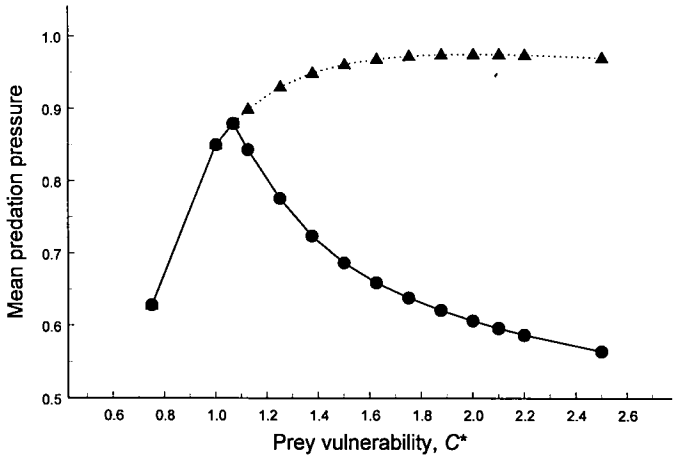

FIG. 2. Mean predation risk per unit change in vulnerability, plotted against the mean vulnerability of the prey population, C^*, for a model consisting of equations (2a,b). The parameter values are: $h = 1$, $B = 1$, $q = 0.85$, $d = 0.5$, $R = 0.5$, $k = 0.5$. The dotted line with triangular points gives the risk per unit vulnerability predicted by equilibrium densities, while the solid line with circular points gives the actual risk per unit vulnerability. The system becomes unstable when $C^* > 1.0664$, which is just above the evolutionary equilibrium value of $C^* = 1$. When $C^* > 1.067$, any increase in mean vulnerability decreases the fitness cost to the individual of further increases in vulnerability.

by considering how population cycles affect the mean predation pressure experienced by a prey individual. Mean predation pressure is given by the quantity, $P/(1 + C^*hN)$, averaged over the period of a cycle. As Figure 2 shows, this average is less than the equilibrium value, and the average decreases as C^* increases, once the equilibrium point becomes unstable. A larger C^* in a cycling system increases the fraction of time that predators are either rare or saturated with food. The decrease in predation pressure with increasing C^* shown in Figure 2 appears to be occur whenever the predator-prey system would be unstable with C^* fixed at the equilibrium value (eq. [3c]). Because the mean risk represents the cost of a unit increase in vulnerability, these results imply that the cost of greater vulnerability decreases as vulnerability increases. The increase in growth rate due to a unit increase in vulnerability is always q. If vulnerability evolves, the net result is a positive feedback for ever-increasing C^* and ever-increasing cycle amplitude. This runaway process could be stopped if the additional growth rate due to a unit increase in C became smaller as C increased; that is, if $r'' < 0$. Constraints that impose a maximum C^* could also halt the increase. A final mechanism for stopping this runaway process is depletion of the prey's resources, which is also likely to reduce the benefit of greater vulnerability (this will be considered in a future article).

It should be noted that the runaway is also the only outcome observed in some cases where population dynamics would be stable if C^* were fixed at the value given by equation (3c). If the equilibrium C^*-value is sufficiently close to the threshold C^* at which population dynamic cycles occur (eq. [A5] in the Appendix), and if the adaptive rate parameter V_0 is large enough to destabilize the equilibrium point, then a runaway may occur for all initial conditions. This is true of

a system with the same parameters as in Figure 1, except that q is reduced from 1.2 to 0.85, provided V_0 is large enough to destabilize the equilibrium. In this example, the equilibrium C^* is only 6.7% less than the minimum value at which population dynamic cycles occur. If the system starts near its equilibrium, cycles are initially driven by evolutionary instability, but this eventually results in C^* becoming large enough that the population dynamic cycles drive a runaway process.

2. *A Stable Equilibrium or Runaway Evolution of Increasing C^**

If a locally stable equilibrium exists, it may or may not be globally stable. If a predator-prey system with C^* fixed at its equilibrium value is stable, the equilibrium point of the trait-population system is always locally stable when the rate of adaptation, V_0, is sufficiently small. However, if the mean vulnerability, C^*, becomes sufficiently large, either because it is initially large or because predator density remains low for a sufficiently long time, then population dynamic cycles will occur and drive a runaway process. In practice, such a runaway may require that the initial C^* be unrealistically large. The size of the basin of attraction of a stable equilibrium is a function of all of the parameters.

3. *A Bounded Nonpoint Attractor or Runaway Evolution of Increasing C^**

This pair of dynamic patterns is possible when the equilibrium C^* (eq. [3c]) is sufficiently smaller than the minimum C^* that generates population dynamic cycles (eq. [A5]). In these cases, the attractor may be a limit cycle or a chaotic attractor, with more complex cycles or chaos being generated by more extreme values of some of the stability-determining parameters; for example, V_0. However, the attractor is not always globally stable, and a runaway evolutionary increase in C^* occurs for some initial conditions, such as a large initial value of C^*.

Figure 3 shows dynamic behaviors for two different adaptive rate parameters, V_0, for a system characterized by dynamic outcome (3). Sufficiently slow adaptation, V_0 (less than approximately 0.03148 in this example) results in a stable system with an oscillatory approach to equilibrium. Increasing the rate of adaptation often leads to a series of bifurcations, leading first to simple limit cycles, as shown in Figure 3A, and then to more complex limit cycles and finally to chaotic dynamics, shown in Figure 3B. The transition between limit cycles and chaos is strongly influenced by the genetic variance function $v(C^*)$; if this function incorporates a minimum C-value (s in eq. [2c]) substantially greater than zero, chaos is less likely to occur. Runaway evolution of greater vulnerability is prevented because the dynamic response of the predator population acts to prevent continued increase (or decrease) of prey vulnerability. However, sufficiently rapid adaptation (large V_0) or a sufficiently large initial vulnerability can result in the expanding cycles and oscillatory increase in C^* shown in Figure 1.

The three dynamic outcomes described above can also occur when equations (2a–c) are modified to make them more realistic. However, a runaway process that continues until prey or predator go extinct can be prevented by two types of modification; incorporation of a maximum vulnerability, or a nonlinear growth function, $r(C)$. A maximum C could be incorporated into the model by assuming that $v(C^*)$ approaches zero as C^* approaches its maximum. In this case, parameters that would otherwise produce a runaway result in large amplitude cycles in which C^* never exceeds its maximum value. In an unstable system that has a bounded attractor, an upper limit to C^* reduces the range of parameters producing chaotic dynamics. The model with two prey phe-

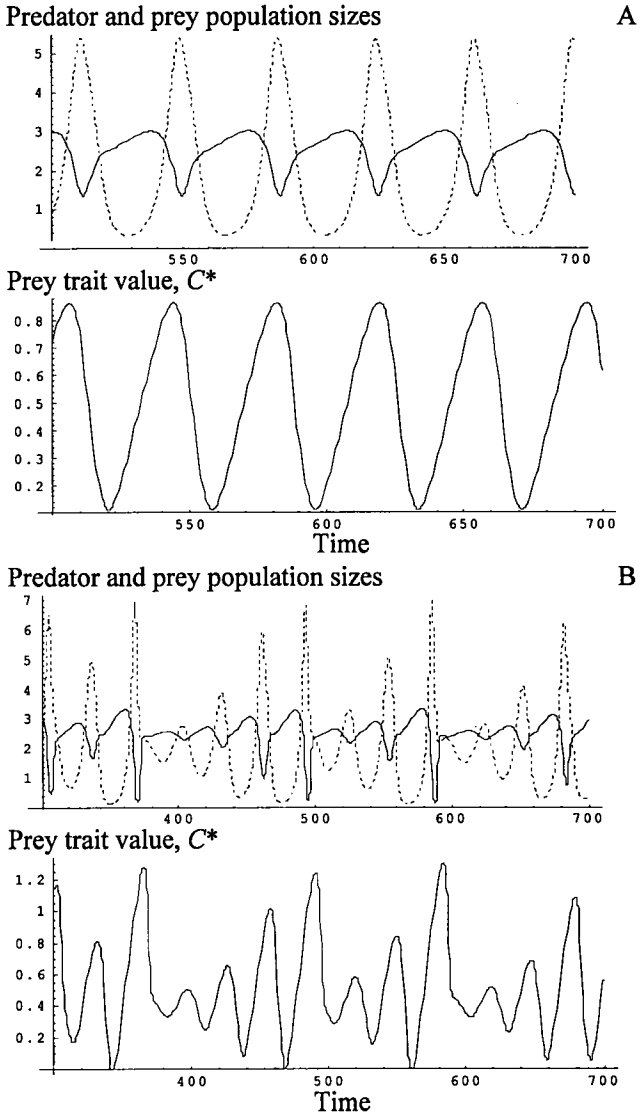

FIG. 3. Population and trait dynamics of equations (2a–c) for two different adaptive rates in a situation where the population dynamics alone would lead to a stable system. In each part of the figure, the top plot shows population densities (prey density is the solid line and predator density is the dashed line). The bottom plot shows trait values. In A ($V_0 = 0.05$), there are limit cycles, whereas B ($V_0 = 0.075$) exhibits what appears to be chaotic fluctuations or limit cycles with a very long period. The common parameter values are: $h = 1$, $B = 1$, $q = 0.8$, $d = 0.5$, $R = 2.5$, $k = 1$, $s = 0.001$. Very large values of V_0 (e.g., $V_0 = 0.5$), which are not shown, lead to ever expanding cycles as in Figure 1, whereas small enough values ($V_0 < 0.03148$) make the equilibrium locally stable.

notypes presented below is an example of the dynamics that result from having one particular type of limits on C^*.

The linear increase in prey per capita growth rate with vulnerability cannot be realistic at very high or very low vulnerabilities. To achieve complete immunity from predation ($C = 0$), a prey individual would probably have to pay a very high price in other components of fitness. Limitations on the predator must set a maximum C^*. Furthermore, the rate of increase in the prey's growth function, $r(C)$, is likely to become very slow as C approaches this maximum. These two features can be incorporated into the growth rate function $r(C)$ as follows:

$$r(C) = q_1C - q_2C^2 - q_3/C, \tag{4}$$

where the q_i are constants, and q_3 is assumed to be close to zero, and much smaller than q_1 and q_2. Because of these assumptions, the maximum C is approximately $q_1/(2q_2)$, and $d^2r/dC^2 < 0$ at the equilibrium point. Local stability for the equilibria of this model must usually be determined numerically. Otherwise, the system can be analyzed in approximately the same way as equations (2a–c). Because equation (4) reduces to q_1C as q_2 and q_3 approach zero, all of the dynamic behaviors discussed above are also possible in this case, except that a positive value of q_2 prevents indefinite increase in C. As was noted in the general model, a large negative r'' is stabilizing. At the equilibrium point $r'' \approx -2q_2$, so large values of q_2 usually result in a stable system. Simulation results suggest that a significant positive value of q_2 also greatly reduces the probability that an unstable system will have chaotic dynamics.

Case 2: Adaptation via Change in the Frequency of Two Phenotypes

Our purpose in examining this second case is to assess the generality of the phenomenon of cycles driven by adaptive change in prey vulnerability. The situation considered here assumes two independently reproducing prey clones (or species) that differ in their values of C but are identical in all other characteristics. This allows adaptation of the total prey community by shifts in the relative abundances of the two types. The fixed values of C in the two types lead to upper and lower limits to the mean capture rate (C^*) of the prey community. The maximum rate of change in C^* itself changes as the relative frequencies of the two types change. In the general case that is analogous to equations (1a–c), the dynamics are described as follows:

$$dP/dt = Pb[(C_1N_1 + C_2N_2)g(C_1N_1 + C_2N_2)] \tag{5a}$$

$$dN_1/dt = N_1[r(C_1) - f(N_1 + N_2) - C_1Pg(C_1N_1 + C_2N_2)] \tag{5b}$$

$$dN_2/dt = N_2[r(C_2) - f(N_1 + N_2) - C_2Pg(C_1N_1 + C_2N_2)]. \tag{5c}$$

Because we are primarily interested in how the mechanism of prey adaptation affects stability, we will only consider the analogue of the specific model, equations (2a–c):

$$dP/dt = P[B(C_1N_1 + C_2N_2)/(1 + hC_1N_1 + hC_2N_2) - d] \tag{6a}$$

$$dN_1/dt = N_1[R + qC_1 - k(N_1 + N_2) - C_1P/(1 + hC_1N_1 + hC_2N_2)] \tag{6b}$$

$$dN_2/dt = N_2[R + qC_2 - k(N_1 + N_2) - C_2P/(1 + hC_1N_1 + hC_2N_2)]. \tag{6c}$$

If the two prey equations (6b,c) are summed, the resulting equation for the dynamics of the total prey population is identical in form to equation (2b). Equations (6b,c) imply that the dynamics of the average vulnerability (C^*, where $C^* = C_1N_1/[N_1 + N_2] + C_2N_2/[N_1 + N_2]$) is given by:

$$dC^*/dt = \{N_1N_2(C_1 - C_2)^2/(N_1 + N_2)^2\} \times [q - P/(1 + hC^*N)]. \tag{7}$$

The quantity in braces is equal to the variance in C in the total prey population, while the term in square brackets is the fitness gradient. Equations (6a), (7), and the sum of (6b) and (6c) give a dynamic system that is equivalent to equations (2a–c) except for the form of $v(C^*)$. Thus, the conditions for local stability of equations (6a–c) are equivalent to those for equations (2a–c) when expressed as a function of the equilibrium $v(C^*)$. In the two-prey-type system, the variance of C within the entire prey community is depleted and the rate of change in the mean vulnerability becomes very small when either type becomes rare relative to the other.

Figure 4 shows some simulation results for the case when C_1 and C_2 are located symmetrically with respect to the equilibrium value for equations (2a–c); that is, $C_1 = (1 + \delta)kd/(R[B - dh])$, and $C_2 = (1 - \delta)kd/(R[B - dh])$. The symmetry assumption simplifies the analysis because it means that the equilibrium predator density is identical to that given by equation (3a), and the equilibrium density of each prey is one-half that given by equation (3b). The mean vulnerability at equilibrium is given by equation (3c). The variance in C at equilibrium is given by $\delta^2(kd)^2/(R[B - dh])^2$. Equations (6a–c) have the same local stability conditions as equations (2a–c) when $V_0 = \delta^2(kd)^2/(R[B - dh])^2$. The dynamics of unstable versions of equations (6a–c) are often limit cycles with negative correlations in the abundances of the two prey types, as illustrated in Figure 4A. If C_1 (the larger C) is sufficiently large to generate population dynamic cycles, prey type 2 can be forced to extinction, as shown in Figure 4B. The system shown in Figure 4B also has a locally stable equilibrium point at which $N_1 = 0.05$ and $N_2 = 0.9$, which can be reached if the initial N_1 is sufficiently small.

Discussion

The results presented here show that several adaptive processes operating on the vulnerability of prey to predators are capable of generating population cycles in predator-prey systems. These cycles are accompanied by cycles in the mean vulnerability. A runaway process is also possible, in which mean prey vulnerability and cycle amplitude both increase until constraints stop the increase in vulnerability, or until one or both species go extinct. A previous analysis of a similar system with constant predator numbers (Matsuda and Abrams 1994a) showed that a runaway decrease in prey numbers and vulnerability could also occur. Both the earlier re-

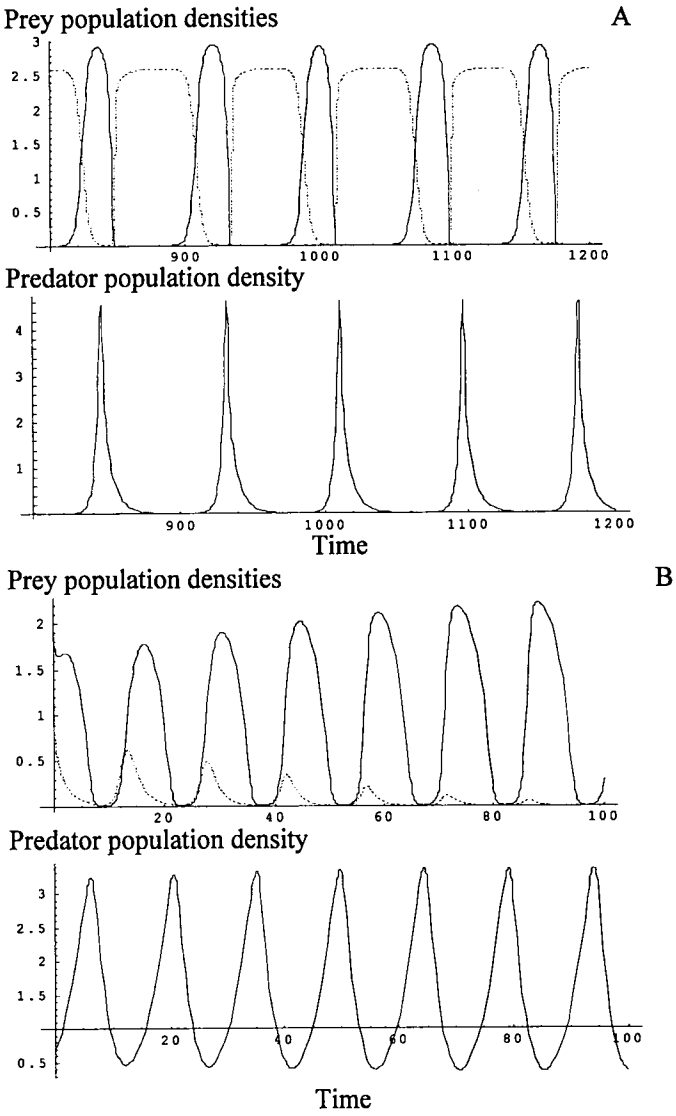

FIG. 4. The dynamics of the two-prey model (eqs. [6a–c]) for two different sets of parameters. In A, the system would be stable if there were a single prey whose vulnerability, C, was equal to either of C_1 and C_2. In B, the system would be unstable if prey 1 alone was present (and stable with prey 2 alone). In both cases, the two vulnerabilities are located symmetrically above and below the ESS value of equation (3c). In both parts of the figure the top plot shows the two prey populations (prey 1 is the solid line and prey 2 is dashed), and the bottom plot shows the predator density. In A, the parameter values are: $R = 2.556$, $d = 0.5$, $B = h = k = 1$, $q = 0.2222$, $C_2 = 0.2$, $C_1 = 2$; in B, the parameter values are: $R = 0.95$; $k = 1$; $d = 0.5$; $q = 0.775$; $B = h = 1$; $C_2 = 1$; $C_1 = 2$.

sults and the present ones are dependent on predators that have saturating functional responses (i.e., per capita capture rate of a predator individual decreases as prey availability increases). Available evidence suggests that such responses are the norm in predator-prey systems (Hassell 1978). Predator-prey coevolution (or coadaptation) is also capable of generating cycles (Dieckmann et al. 1995; Abrams and Matsuda 1997).

These examples of instability caused by adaptation contrast with the intuitive arguments presented in the introduction, and with previous models of prey adaptation by Ives and Dobson (1987), who considered predators with type-2 functional responses, but assumed that the antipredator behavior did not affect the satiation component of the response (our g function). This makes their model comparable to those with a linear predator functional response, where adaptive change of prey vulnerability is stabilizing.

The fact that cycles or runaways are possible in a simple model does not mean that they are likely in natural systems. Systems can be stabilized if there is a sufficient decrease in the advantage of greater vulnerability as the mean vulnerability increases (i.e., $r'' < 0$). It is possible that stability may also result from interactions of the predator or prey with other species. Alternatively, it is possible that adaptive adjustment of predator hunting behavior or evolutionary responses of the predator could stabilize cycling systems or prevent a runaway of vulnerabilities. The latter two possibilities need to be confirmed by mathematical analyses. In single prey–single predator systems, the mechanism for instability proposed here does not require unrealistically rapid rates of evolution. If the population dynamics are close to neutral stability, condition (A1a) shows that even very slow change (small V_0) can lead to instability.

Given the simplicity of the models analyzed here, it is difficult to assess the biological plausibility of different ranges of parameter values. Genetic trade-offs between growth and vulnerability have not been measured. However, the general analysis shows that sufficient predator satiation (a large, negative g') will destabilize the trait equilibrium, and this will always lead to instability if the rate of adaptation is high enough or density dependence in prey growth is weak enough (f' small). We hope that this theoretical analysis will motivate the necessary measurements of these quantities.

There have been many attempts to explain observed cycles in natural populations using predator-prey theory (see review in Royama 1992). A problem with most of these exercises is that the models seem to display much greater changes in dynamics with changes in parameters than do the natural cycles. The period of the hare-lynx cycles and microtine rodent cycles show remarkably little geographic or temporal variation (Hanski et al. 1991; Royama 1992). In contrast, the period (and amplitude) of simple, ordinary differential equation models of predator-prey systems generally change dramatically with changes in population dynamic parameters (Gilpin 1975; Yodzis and Innis 1992), and small changes in the parameters can result in such extreme amplitude cycles that extinction is assured. The models considered here can generate cycles without producing extremely low densities, and the period and amplitude of the cycles are often relatively insensitive to changes in population dynamic parameters. There are, of course, other potential explanations for well-behaved cycles. For example, Ruxton et al. (1992) have noted that at least some cycles that are produced by developmental time lags are also insensitive to many environmental parameters, and Abrams and Walters (1996) have shown the same to be true of models with certain types of prey refuges.

The preconditions for instability in our models are the presence of saturation in the predator's functional response, and dynamic adaptation by the prey to different levels of predation. Both are common features of natural predator-prey

systems. Both features have received some study in the snowshoe hare–lynx (plus other predators) system in the Canadian arctic (Hik 1994). Hik (1994, 1995) showed that hares do have facultative antipredator behaviors; they confine their activity to areas with high cover (closer to spruce trees) when predators are abundant. This reduces their intake of high quality food and their reproductive rate. Antipredator behaviors and/or the presence of predators caused induced chronically high levels of free cortisol (Boonstra et al., in press), which could generate a maternal effect. There is suggestive evidence that the offspring of hares that exhibit high levels of such antipredator behavior are typically smaller and spend more time in areas with high cover than do the offspring of mothers that do not experience high predation (Hik 1994). Such maternal effects are likely to produce relatively rapid dynamics in the hare's vulnerability that can be modeled by a relatively large rate of adaptive change (V_0) in our models. Most avian and mammalian predators are characterized by strongly saturating functional responses (Yodzis and Innis 1992), so the basic prerequisites for trait-driven cycles are met in the hare-lynx system. At present, we do not know enough to estimate most of the current model's parameters for this system. Indeed, it would be desirable to construct a model that incorporates more of the known biology of this system to better understand the mechanisms responsible for cycling. However, the relative lack of variation in cycle period, the presence of a type-2 predator functional response, and the presence of costly antipredator behaviors are all suggestive of the mechanism described here.

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Appendix

Local stability analyses of systems with prey evolution.

The Jacobian matrix that determines the local stability of equations (1a–c) in the text is:

$$A = \begin{pmatrix} \frac{d\dot{P}}{dP} & \frac{d\dot{P}}{dN} & \frac{d\dot{P}}{dC^*} \\ \frac{d\dot{N}}{dP} & \frac{d\dot{N}}{dN} & \frac{d\dot{N}}{dC^*} \\ \frac{d\dot{C}^*}{dP} & \frac{d\dot{C}^*}{dN} & \frac{d\dot{C}^*}{dC^*} \end{pmatrix}$$

$$= \begin{pmatrix} 0 & C^*P(g + C^*Ng')b' & C^*P(g + C^*Ng')b' \\ -C^*Ng & -N(f' + C^{*2}Pg') & -C^*N^2Pg' \\ -vg & -vC^*Pg' & v(r'' - NPg') \end{pmatrix},$$

where dots denote time derivatives. The signs of the derivatives in the above matrix that follow from basic biological considerations are: $b' > 0$, $g' < 0$, $g + C^*Ng' > 0$, $r'' \leq 0$, $f' > 0$.

The Routh-Hurwitz criteria for local stability of the system follow from this matrix, and are:

$$Q_1 = N(f' + C^{*2}g'P) + v(g'NP - r'') > 0, \quad \text{(A1a)}$$

$$Q_2 = N[b'gP(v + C^{*2})(g + C^*Ng') + vf'g'NP - vr''(f' + C^{*2}g'P)] > 0, \quad \text{(A1b)}$$

$$Q_3 = vb'gNP(g + C^*Ng')(f'N - C^{*2}r'') > 0. \quad \text{(A1c)}$$

$$Q_1Q_2 > Q_3. \quad \text{(A1d)}$$

Criterion (A1c) must be satisfied for all biologically reasonable forms of the functions. Criterion (A1d) is violated when either (A1a) or (A1b) are close to being violated. Thus, the factors producing instability are those that lead to small or negative values of Q_1 or Q_2. Inequality (A1a) must be satisfied if each of its two terms is positive, that is, if trait dynamics in isolation are locally stable ($r'' - NPg' < 0$), and population dynamics with traits fixed traits are locally stable ($f' + C^*Pg' > 0$). Inequality (A1b) must be satisfied if the predator numerical response (b') is sufficiently large relative to the adaptive rate, v. Condition (A1b) may be violated when the predator numerical response is slow relative to evolutionary dynamics, provided that the curvature of the prey growth function, r'', is sufficiently close to zero. Although lack of stability of the trait dynamics can destabilize the entire system, stability of the trait dynamics by itself is not sufficient to guarantee stability of the entire dynamic system. Neutral trait dynamic stability (i.e., $g'NP - r'' = 0$) reduces conditions (A1a–d) to:

$$(f' + C^{*2}g'P)[b'g(v - vN + C^{*2})(g + C^*Ng') - vC^{*2}g'^2PN] > 0. \quad \text{(A2)}$$

The first factor ($f' + C^{*2}g'P$) is positive if the population dynamic subsystem is stable. However, the second factor can be negative when the slope of the predator's numerical response (b') is sufficiently shallow. Thus, there are some conditions under which the population dynamic system without trait dynamics would be stable, the trait dynamics in isolation would be neutrally stable, but the entire system is unstable. Condition (A1a) shows that, when the trait dynamic equation in isolation has an unstable equilibrium point, larger rates of adaptive change (larger v) tend to make the entire system unstable. If the trait dynamics alone are sufficiently stabilizing ($NPg' - r''$ sufficiently large and positive), the entire system can be stable, even when the population dynamic system in the absence of trait dynamics is unstable. Note that if the predator's functional response is linear ($g' = 0$), all four stability conditions (A1a–d) must be satisfied.

The stability analysis of Example 1 in the text follows from the general treatment given above. The corresponding Routh-Hurwitz conditions are:

$$Q_1 = R - [(V_0hqR)(B - dh)]/(Bk) - (d^2hkq)/[BR(B - dh)] > 0 \quad \text{(A3a)}$$

$$Q_2 = [q/(BkR)][d^2k^2 + V_0R^2(B - dh)^2 - V_0hR^3(B - dh)] > 0 \quad \text{(A3b)}$$

$$Q_3 = V_0(B - dh)^2qR^2/(Bk) > 0 \quad \text{(A3c)}$$

$$Q_1Q_2 - Q_3 > 0 \quad \text{(A3d)}$$

Note that condition (A3a) is violated if R approaches zero, if k approaches zero, or if q becomes very large. Condition (A3b) is violated if R is very large. Proportional increases in B and d do not affect equilibrium densities, but they increase the predator's numerical response; such changes therefore increase Q_2, but do not change Q_1. Thus, if R is large (condition [A3b] violated), an increase in predator numerical response rate (larger B, d) can stabilize the system.

Corresponding stability conditions for the case where C^* is fixed at its equilibrium value are given in Yodzis and Innis (1992) and Abrams and Roth (1994). Substituting the equilibrium values given by text equations (2a–c) yields the following criterion for the predator-prey system to be stable when C^* is fixed at its equilibrium value of $kd/(R[B - dh])$:

$$R^2 > d^2hkq/(B^2 - Bdh) \quad \text{(A4)}$$

Another value of C^* that is important for the behavior of this model is the minimum C^*-value that would produce population dynamic cycles when there is no adaptive change, but when C^* is not necessarily at its adaptive equilibrium. This is found by a straight-forward local stability analysis of equations (2a,b) and is:

$$C^*_m = -\frac{R}{2q} + \frac{1}{q}\sqrt{\frac{4kq(B + dh) + hR^2(B - dh)}{4h(B - dh)}}. \quad \text{(A5)}$$