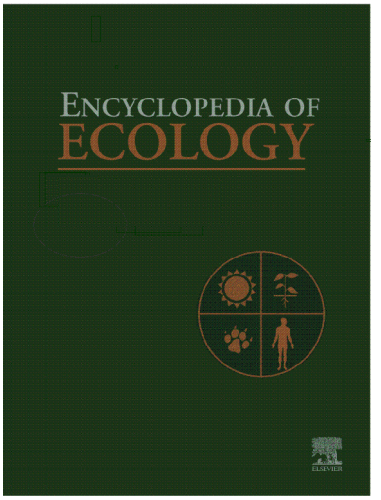

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behavior. Thus, the dramatic decline of the tiger is not completely described by comparing the estimated 100 000 specimens still living in 1900 with the estimated 7000 of 1990 and the 2000 likely surviving at the beginning of this century. Due to the conspicuous racial differences within the species, additional concern is provided by the estimate that no more than 400 Siberian tigers survive in the wild, while three other subspecies (the Bali tiger, the Caspian tiger, and the Javan tiger) already went extinct between the 1940s and the 1980s.

Many studies have been performed on the effects that removing a top predator from an ecosystem may cause on other species. Often, but not always, further species loss has been reported, that is, a series of cascading extinctions in the food web, where the top predator originally performed a stabilizing role. Relationships between food web complexity and effect of top predator removal have been suggested, but the point is still mooted.

See also: Coevolution.

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Prey–Predator Models

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The Lotka–Volterra Model

Why did a complete closure of fishery during World War I cause an increase in predatory fish and a decrease in prey fish in the Adriatic Sea? This was the question that led Vito Volterra to formulate a mathematical conceptualization of prey–predator population dynamics. In his endeavor to explain mechanisms by which predators regulate their prey, he constructed a mathematical model that describes temporal changes in prey and predator abundances. The model makes several simplifying assumptions such as (1) the populations are large enough so it makes sense to treat their abundances as continuous rather than discrete variables; (2) the populations are well mixed in the environment (which is the reason why this type of model is sometimes called mass action model in an analogy with chemical kinetics); (3) the populations are closed in the sense that there is no immigration or emigration; (4) the population dynamics are completely deterministic, that is, no random events are considered; (5) in absence of predators, prey grow exponentially; (6) the per predator rate of prey consumption is a linear

function of prey abundance; (7) predators are specialists and without the prey their population will decline exponentially; (8) the rate with which consumed prey are converted to new predators is a linear function of prey abundance; (9) both populations are unstructured (e.g., by sex, age, size, etc.); and (10) reproduction immediately follows feeding etc.

If $R(t)$ and $C(t)$ are the prey and predator abundance, respectively, then under the above assumptions the population dynamics are described by two differential equations

$$\begin{aligned}\frac{dR}{dt} &= (r-\lambda C)R \\ \frac{dC}{dt} &= (e\lambda R-m)C\end{aligned} \qquad [1]$$

where r is the per capita prey growth rate, λ is the rate of search and capture (hereafter search rate) of a single predator for an individual prey item so that λR is the per predator rate of prey consumption (i.e., the functional response), e is the rate with which consumed prey are converted into predator births, and m is the per capita predator mortality rate. Model [1], which was independently formulated by Alfred Lotka, is today known as the Lotka–Volterra prey–predator model. For initial population abundances $R(0)$ and $C(0)$, this model predicts future abundance of prey $R(t)$ and predators $C(t)$ (**Figure 1a**).

From the ecological point of view, the important information such a model can provide is whether or not population abundances tend to an equilibrium at which both species will coexist. At the equilibrium, predator and prey abundances do not change (i.e., $dR/dt = dC/dt = 0$), which gives

$$R^* = \frac{m}{e\lambda} \quad \text{and} \quad C^* = \frac{r}{\lambda}$$

This equilibrium (shown as the solid dot in **Figure 1b**) is at the intersection of the prey and predator isoclines; they are the lines in the phase space along which $dR/dt = 0$ and $dC/dt = 0$ (shown as dashed lines in **Figure 1b**). Interestingly, the prey equilibrium depends only on parameters that describe population growth of predators whereas the predator equilibrium depends on the prey per capita growth rate r. Thus, increasing the prey growth rate r (which is sometimes called enrichment in the ecological literature) does not change the prey equilibrium density, but it increases the predator equilibrium abundance.

Knowing the interior equilibrium does not tell us whether this equilibrium is stable with respect to perturbations in population abundances or not. In other words, we want to know if after some (random) perturbation from the equilibrium, population abundances will return to this equilibrium or not. For the Lotka–Volterra model [1] this question is easy to solve because the model is an example of a conservative system with the first integral

$$V(R, C) = m(R/R^* - 1 - \ln R/R^*) + r(C/C^* - 1 - \ln C/C^*) \qquad [2]$$

which is constant along the trajectories of the model (here ln denotes the natural logarithm). Indeed, the time derivative of V along a trajectory of model [1]

$$\frac{dV(R(t), C(t))}{dt} = \frac{\partial V}{\partial R}R(r-\lambda C) + \frac{\partial V}{\partial C}C(e\lambda R - m) = 0$$

which implies that function V is constant along the trajectories of the Lotka–Volterra model [1]. Moreover, $V(R, C) \geq 0$ for positive population abundances (because the inequality $x - \ln x \geq 1$ holds for every $x > 0$) and function V minimizes at the equilibrium point (R^*, C^*). Thus, solutions of the equation $V(R, C) = \text{const}$, which are closed curves in the prey–predator phase space (**Figure 1b**), correspond to solutions of model [1]. This analysis shows that both prey and predator numbers will oscillate periodically around the equilibrium with the amplitude and frequency that depend on the initial prey and predator densities. Moreover, the average values of

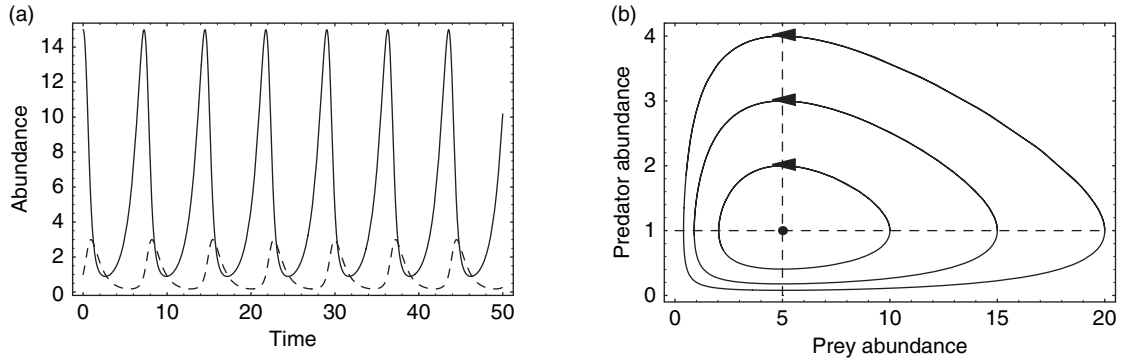

Figure 1 Solutions of the Lotka–Volterra model [1] in time domain (a, solid line shows prey abundance, dashed line predator abundance) and in the prey–predator abundance phase space (b). Dashed lines are the isoclines. Parameters: $r = 1$, $\lambda = 1$, $e = 0.2$, $m = 1$.

prey and predator densities over one period coincide with their equilibrium densities R^* and C^*. Indeed, the equation for prey can be rewritten as

$$\frac{\mathrm{d}\ln(R)}{\mathrm{d}t} = r - \lambda C$$

Integration of this equation over one population cycle of length T time units gives

$$\ln(R(T)) - \ln(R(0)) = rT - \lambda \int_0^T C(t)\mathrm{d}t$$

Since T is the period, the left hand side of the above equality is zero (because $R(0) = R(T)$) and

$$\bar{C} = \frac{1}{T}\int_0^T C(t)\mathrm{d}t = \frac{r}{\lambda} = C^*$$

where $\bar{C}$ denotes the average predator density. Similarly, the average prey density over each cycle equals the prey equilibrium density.

The above analysis shows that the prey–predator equilibrium is Lyapunov stable (i.e., after a small perturbation the animal abundances stay close to the equilibrium, **Figure 1b**), but it is not asymptotically stable because the population abundances do not return to the equilibrium. This particular type of equilibrium stability is sometimes called the neutral stability. The eigenvalues of the Lotka–Volterra model evaluated at the equilibrium are purely imaginary ($\pm\mathrm{i}\sqrt{rm}$) which implies that the period of prey–predator cycles with a small amplitude is approximately $2\pi/\sqrt{rm}$.

The mechanism that makes prey–predator coexistence possible in this particular model is the time lag between prey and predator abundances, with the predator population lagging behind the prey population (**Figure 1a**). The Lotka–Volterra model shows that (1) predators can control exponentially growing prey populations (this type of regulation is called the top-down regulation), (2) both prey and predators can coexist indefinitely, (3) the indefinite coexistence does not occur at equilibrium population densities, but along a population cycle. Can this model explain the question about the observed changes in predator and prey fish abundances during World War I? Volterra hypothesized that fishery reduces the prey per capita growth rate r and increases the predator mortality rate m, while the interaction rates e and λ do not change. Thus, ceased fishery during World War I should lead to a decrease in average prey fish population R^* and to an increase in the average predator fish population C^*, exactly as observed.

The Functional and Numerical Response

The Lotka–Volterra model assumes that the prey consumption rate by a predator is directly proportional to the prey abundance. This means that predator feeding is limited only by the amount of prey in the environment. While this may be realistic at low prey densities, it is certainly an unrealistic assumption at high prey densities where predators are limited, for example, by time and digestive constraints. The need for a more realistic description of predator feeding came from the experimental work of G. F. Gause on protist prey–predator interactions. He observed that to explain his experimental observations, the linear functional dependencies of the Lotka–Volterra model must be replaced by nonlinear functions.

To understand the nature of prey–predator interactions, M. E. Solomon introduced concept of functional and numerical responses. The functional response describes prey consumption rate by a single predator as a function of prey abundance, while the numerical response describes the effect of prey consumption on the predator recruitment. Most simple prey–predator models such as the Lotka–Volterra model assume that production of new predators is directly proportional to the food consumption. In this case, the numerical response is directly proportional to the functional response. The constant of proportionality, e in model [1], is the efficiency with which prey are converted to newborn predators.

C. S. Holling introduced three types of functional responses (**Figure 2**). The type I functional response is the most similar to the Lotka–Volterra linear functional response, but it assumes a ceiling on prey consumption rate

$$f_{\mathrm{I}}(R) = \min\{\lambda R, \mathrm{const}\}$$

where const is the prey consumption rate when prey abundance is high (**Figure 2a**). This functional response is found in passive predators that do not hunt actively (e.g., web-building spiders and filter feeders).

The type II functional response assumes that predators are limited by total available time T. During this time predators are assumed either to search for prey (for T_{s} time units), or to handle prey (for T_{h} units). If the predator search rate is λ and R is the current prey density then the encounter rate of a searching predator with prey is λR. If handling of a single prey item takes h time units then $T_{\mathrm{h}} = h\lambda R T_{\mathrm{s}}$. Thus, $T = T_{\mathrm{s}} + T_{\mathrm{h}} = T_{\mathrm{s}}(1 + h\lambda R)$ and the number of consumed prey by a predator during time T is $\lambda R T_{\mathrm{s}}$. The average consumption rate over time interval T is then

$$f_{\mathrm{II}}(R) = \frac{\lambda R T_{\mathrm{s}}}{T} = \frac{\lambda R}{1 + h\lambda R}$$

which is the Holling type II functional response (**Figure 2b**). This functional response is concave and for large prey abundances it converges to $1/h$, which is the upper limit on consumption. The form of the Holling type II functional response is equivalent with the

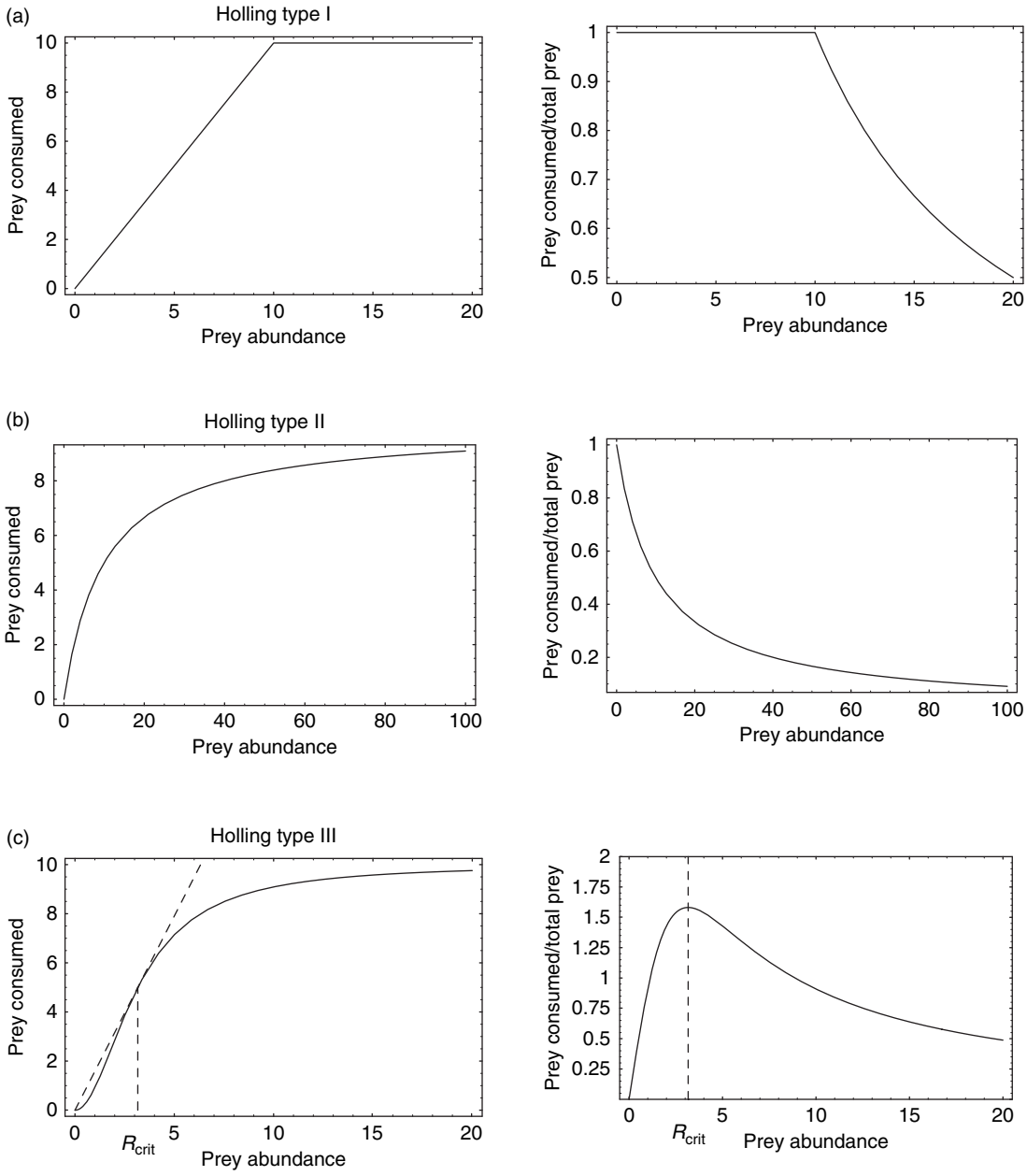

Figure 2 The three Holling type functional responses (left panel (a), type I; (b) type II; (c), type III). R_{crit} in panel (c) is the critical prey density below which the functional response is stabilizing. The right panel shows the effect of the functional response on the equilibrium stability. Stability condition [8] requires that the ratio of consumed prey to total prey abundance is an increasing function of prey abundance. Parameters: $\lambda = 1$, $h = 0.1$.

Michaelis–Menten rate of substrate uptake as a function of the substrate concentration.

The Holling type II functional response assumes that the predator search rate λ is independent of the prey density. However, there are several ecological processes that can make this parameter itself a function of prey abundance, that is, $\lambda(R)$. These processes include, for example, predator inability to effectively capture prey when at low densities, predator learning, searching images, predator switching between several prey types, optimal predator foraging, etc. When substituted to the Holling type II functional response, this added complexity can change the concave shape of the functional response to a sigmoid shape (**Figure 2c**). Sigmoid functional responses are called the Holling type III functional responses. A prototype of such a functional response is obtained when λ is replaced by $\lambda R^{\mu-1}$ in the Holling type II functional response, which then leads to

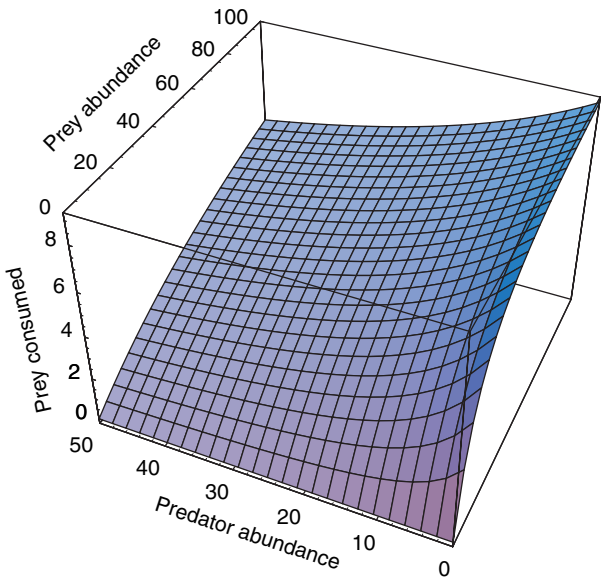

Figure 3 The Beddington–DeAngelis functional response [3]. Parameters: $\lambda = 1$, $h = 0.1$, $z = 0.2$.

a particular form of the Holling type III functional response

$$f_{\mathrm{III}}(R) = \frac{\lambda R^{\mu}}{1 + \lambda b R^{\mu}}$$

with parameter $\mu \geq 1$ ($\mu = 2$ in Figure 2c). For $\mu = 1$ the above functional response coincides with the Holling type II functional response, while for $\mu > 1$ the predator search rate increases with increasing prey density. This functional response is also called the Hill function. In enzymatic reaction kinetics the Hill function often describes a cooperative binding of several substrate molecules with an allosteric enzyme that has several binding sites.

The Holling functional responses consider a single predator and are thus functions of prey abundance only. If many predators are present, the per predator prey consumption rate can be influenced by predator interference that makes the functional response also dependent on predator density. A prototype of such a functional response is the Beddington–DeAngelis functional response

$$f(R, C) = \frac{\lambda R}{1 + \lambda b R + z C} \qquad [3]$$

where z is a positive parameter that models predator interference (Figure 3). Several other types of functional responses can be found in the literature.

Effects of Functional and Numerical Responses on Prey–Predator Stability

How does the shape of functional and numerical responses influence prey–predator stability? This question led G. F. Gause, A. N. Kolmogorov, W. W. Murdoch, and others to analyze prey–predator models where the linear functional and numerical responses of the Lotka–Volterra model are replaced by more general functions. A general representation of a prey–predator model is

$$\begin{aligned} \frac{dR}{dt} &= r(R)R - f(R, C)C \\ \frac{dC}{dt} &= (g(R, C) - m)C \end{aligned} \qquad [4]$$

where $r(R)$ is the per capita prey growth rate, $f(R,C)$ is the functional response, $g(R,C)$ is the numerical response, and m is the per capita predator mortality rate. For $r(R) = r$, $f(R,C) = \lambda R$ and $g(R,C) = e\lambda R$, the above model coincides with the Lotka–Volterra model [1].

In what follows we will assume that model [4] has a single positive equilibrium R^* and C^*. Then the question is, what is the long-term behavior of prey and predator abundances? Do they converge to this equilibrium? The usual starting point to answer this question is to study conditions under which the equilibrium is locally asymptotically stable. Conditions that guarantee local asymptotic stability of the equilibrium are given in terms of the Jacobian matrix evaluated at the equilibrium of model [4]:

$$\begin{pmatrix} \dfrac{dr(R^*)}{dR}R^* + r(R^*) - \dfrac{\partial f(R^*, C^*)}{\partial R}C^*, & -f(R^*, C^*) - \dfrac{\partial f(R^*, C^*)}{\partial C}C^* \\ \dfrac{\partial g(R^*, C^*)}{\partial R}C^*, & \dfrac{\partial g(R^*, C^*)}{\partial C}C^* \end{pmatrix}$$

To derive the above matrix we used the fact that at the equilibrium, $g(R^*,C^*) = m$. If the sum of the two diagonal elements (i.e., the trace) of the Jacobian matrix is negative and the determinant is positive then the equilibrium is locally asymptotically stable. This leads to the following two general conditions:

$$\frac{dr(R^*)}{dR}R^* + r(R^*) - \frac{\partial f(R^*, C^*)}{\partial R}C^* + \frac{\partial g(R^*, C^*)}{\partial C}C^* < 0 \qquad [5]$$

$$\begin{aligned} &\left(\frac{dr(R^*)}{dR}R^* + r(R^*) - \frac{\partial f(R^*, C^*)}{\partial R}C^*\right)\frac{\partial g(R^*, C^*)}{\partial C} \\ &\quad + \left(f(R^*, C^*) + \frac{\partial f(R^*, C^*)}{\partial C^*}C^*\right)\frac{\partial g(R^*, C^*)}{\partial R} > 0 \end{aligned} \qquad [6]$$

Although these two conditions look quite formidable, they will substantially simplify for particular cases of functional and numerical responses considered in the next section.

Prey Growth Is Density Independent

Here we assume that the per capita prey growth rate is density independent ($r(R) = r$), which implies that in stability conditions [5] and [6], $dr(R^*)/dR = 0$. This case

corresponds to the original Lotka–Volterra model in the sense that the only mechanism that regulates the exponential prey growth is predation (i.e., top-down regulation).

Effects of the Holling type functional responses on population dynamics

We will consider the effect of replacing the linear functional response by a nonlinear functional response in the Lotka–Volterra model. Prey–predator population dynamics are described by

$$\begin{aligned}\frac{dR}{dt} &= rR - f(R)C \\ \frac{dC}{dt} &= (g(R) - m)C\end{aligned} \qquad [7]$$

For these dynamics the stability conditions [5] and [6] substantially simplify because functional and numerical responses are independent of the predator density (i.e., $\partial f/\partial C = \partial g/\partial C = 0$). Substituting the predator equilibrium abundance $C^* = rR^*/f(R^*)$, in stability conditions [5] and [6] gives

$$\frac{df(R^*)}{dR} > \frac{f(R^*)}{R^*} \qquad [8]$$

and

$$\frac{dg(R^*)}{dR} > 0$$

The latter condition states that the numerical response should be an increasing function of prey abundance. This condition will be satisfied for any reasonable numerical response and let us focus on the first condition. This stability condition can be interpreted graphically. The equilibrium is locally asymptotically stable provided the slope of the tangent line to the graph of the functional response at the point $(R^*, f(R^*))$ is higher than is the slope of the line that passes through the origin and the point $(R^*, f(R^*))$. For example, in **Figure 2c** this happens if the prey equilibrium density is to the left of the critical value R_{crit} at which both slopes are the same (i.e., the tangent to the graph, shown as the dashed line, passes through the origin). Alternatively, stability condition [8] states that for a prey–predator equilibrium to be locally asymptotically stable it is sufficient that the ratio of consumed prey to total prey (i.e., $f(R)/R$, **Figure 2**, right panel) is an increasing function of prey density at the equilibrium. Indeed, condition [8] is nothing else than expression of the fact that derivative of function $f(R)/R$ with respect to prey density is positive. This is equivalent to saying that the prey zero isocline ($C = rR/f(R)$, shown as the horizontal dashed curve in **Figures 1b** and **4**–**8**) has a negative slope at the equilibrium. Clearly, a linear functional response used in the Lotka–Volterra model, the type I functional response and the type II functional response do not satisfy stability condition [8] (**Figures 2a**, **2b**, right panel). This means that the interior equilibrium is not asymptotically stable and we can ask what happens if populations are shifted of the equilibrium. In the case of the Lotka–Volterra model with the linear functional response we already know that after a perturbation trajectories oscillate around the equilibrium (**Figure 1**).

Now let us consider the effect of the Holling type II functional response on the prey–predator equilibrium stability. For small handling times the Holling type II functional response causes trajectories to spiral outward from the equilibrium (**Figure 4a**)

$$R^* = \frac{m}{(e - hm)\lambda}, \quad C^* = \frac{er}{(e - hm)\lambda}$$

When prey density is high, the Holling type II functional response is saturated and equal approximately to $1/h$. Substituting this value in [7] and integrating the model, it can be proved that for large handling times that satisfy $h > e\lambda/(r + m)$ there are trajectories along which both prey and predator populations grow to infinity (one such trajectory is shown in **Figure 4b**). In this latter case the prey population escapes completely the predator

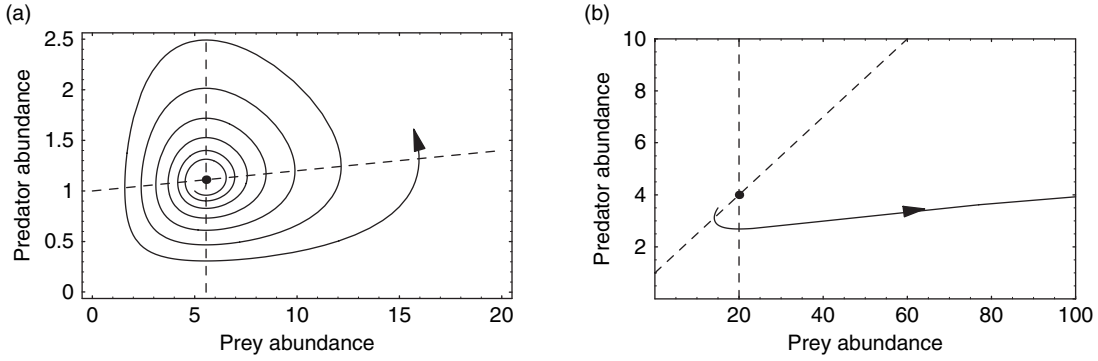

Figure 4 Prey–predator population dynamics [7] with the Holling type II functional response. Panel (a) assumes small handling time ($h = 0.02$) while panel (b) assumes large handling time ($h = 0.15$). The dashed lines are isoclines. Parameters: $r = 1$, $\lambda = 1$, $e = 0.2$, $m = 1$.

regulation. This happens, for example, when predators are inefficient when handling prey (i.e., when the handling time is large), or the intrinsic per capita prey growth rate is high. This analysis validates the statement that the Holling type II functional response is destabilizing, which means that when handling times are positive, the neutrally stable equilibrium of the Lotka–Volterra model becomes unstable.

The Holling type I functional response combines the effects of the linear functional response with the Holling type II functional response for large handling times. Thus, when the prey–predator equilibrium is in the part of the prey–predator phase space where the functional response increases linearly (Figure 5), small perturbations lead to periodic oscillations around the equilibrium while large perturbations lead to trajectories that diverge from the equilibrium.

The work of A. N. Kolmogorov shows that prey–predator coexistence can occur only either at the equilibrium, or along a limit cycle. However, in the case of the prey–predator model [7] with the Holling type II functional response it can be proved that no limit cycle exists. Indeed, because

$$\frac{\partial}{\partial R}\left(\frac{1}{RC}(rR-f(R)C)\right)+\frac{\partial}{\partial C}\left(\frac{1}{RC}(g(R)C-mC)\right) =\frac{1}{R}\left(\frac{f(R)}{R}-\frac{\mathrm{d}f(R)}{\mathrm{d}R}\right)>0 \qquad [9]$$

the Dulac criterion excludes prey–predator limit cycles.

The functional response that meets the local stability condition [8] is the Holling type III response. But, the stabilizing effects of predators will occur only at low to medium prey equilibrium densities (those that are to the left of R_{crit} in Figure 2c). This is the case shown in Figure 6a. At a higher prey equilibrium density, the functional response saturates and predators cannot regulate their prey (Figure 6b).

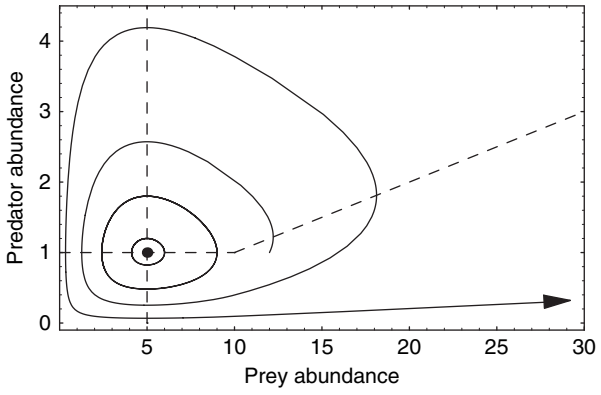

Figure 5 Prey–predator population dynamics [7] with the Holling type I functional response. Small perturbations from the equilibrium lead to cycles exactly as in the case of the Lotka–Volterra equation with the linear functional response. Larger perturbations lead to trajectories that diverge from the equilibrium. The dashed lines are isoclines. Parameters: $r=1$, $\lambda=1$, $e=0.2$, $m=1$, const $=10$ where *const* is the upper ceiling of the Holling type I functional response.

Functional and numerical responses depend on the predator density

A prototype of such functions is the Beddington–DeAngelis functional response [3]. To simplify analysis, it is assumed that the handling time in the Beddington–DeAngelis functional response is zero ($h=0$). Thus, prey–predator population dynamics are described by

$$\frac{\mathrm{d}R}{\mathrm{d}t}=rR-\frac{\lambda R}{1+zC}C$$
$$\frac{\mathrm{d}C}{\mathrm{d}t}=(g(R,C)-m)C \qquad [10]$$

At the population equilibrium the first stability condition [5] simplifies to $\partial g(R^*,C^*)/\partial C<0$ which means that the predator growth must be negatively density dependent. The second stability condition [6] simplifies to $\partial g(R^*,C^*)/\partial R>0$, which holds provided the numerical response increases with increasing prey density. This analysis implies that density-dependent predator growth stabilizes prey–predator population dynamics when handling times are neglected (Figure 7). Depending on the parameters, positive handling times can surpass the stabilizing effect of predator interference leading to an unstable equilibrium.

Prey Growth Is Density Dependent

Now let us consider the case where the per capita prey growth rate is density dependent and decreases with increasing prey density ($\mathrm{d}r(R)/\mathrm{d}R<0$). The simplest prototype of such dependence is a linear decrease in the per capita prey growth rate ($r(R)=r(1-(R/K))$, which is then the logistic equation with the carrying capacity K. This type of prey growth is also called the bottom-up regulation. Clearly, the negative density-dependent prey growth promotes prey–predator coexistence because the prey growth is now controlled by two independent mechanisms: top-down and bottom-up regulation. This is reflected in stability condition [5] where the left hand side of the inequality is smaller for the negative density-dependent prey growth rate when compared with exponentially growing prey.

The Lotka–Volterra model with the logistic prey growth rate is

$$\frac{\mathrm{d}R}{\mathrm{d}t}=rR\left(1-\frac{R}{K}\right)-\lambda RC$$
$$\frac{\mathrm{d}C}{\mathrm{d}t}=(e\lambda R-m)C \qquad [11]$$

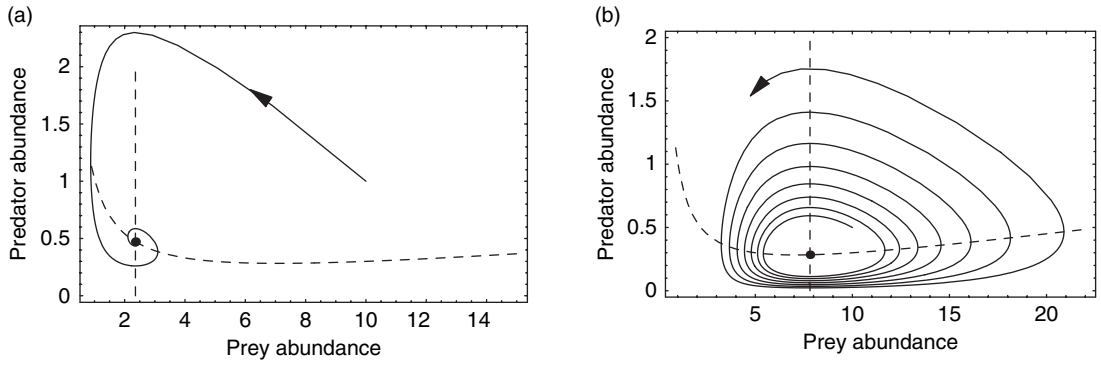

Figure 6 Prey–predator population dynamics [7] with the Holling type III functional response. (a) shows the stabilizing case where the prey population equilibrium is in the range of prey abundances where the Holling type III functional response is stabilizing (i.e., smaller than R_{crit} from **Figure 2c**). (b) Shows the case where the prey population equilibrium does not satisfy this condition and the equilibrium is unstable. Dashed lines are isoclines. Parameters: $r=1$, $\lambda=1$, $e=0.2$, $h=0.02$, $m=1$ in (a) and $m=5.5$ in (b).

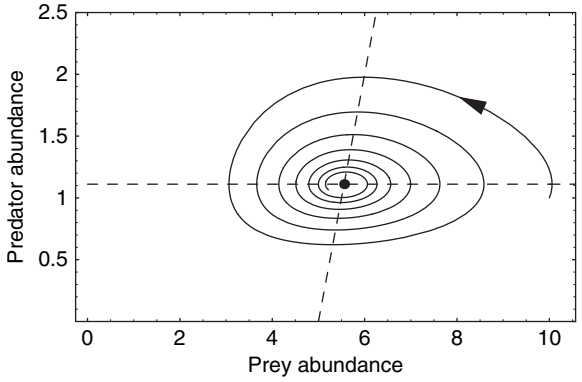

Figure 7 Prey–predator population dynamics [7] with the Beddington–DeAngelis functional response [3] when handling time is neglected ($h=0$). Dashed lines are isoclines. Parameters: $r=1$, $\lambda=1$, $e=0.2$, $z=0.1$, $m=1$.

Provided $K>(m/e\lambda)$, the interior equilibrium of this system is

$$R^*=\frac{m}{e\lambda} \quad \text{and} \quad C^*=\frac{(eK\lambda-m)r}{eK\lambda^2}$$

and local stability conditions [5] and [6] hold for all parameter values. Using the first integral V given by formula [2] as a Lyapunov function it is easy to see that this equilibrium is globally asymptotically stable (i.e., all trajectories of model [11] with initially both populations present converge to this equilibrium). Indeed, function V decreases along the trajectories of model [11] because

$$\frac{\mathrm{d}V(R(t),C(t))}{\mathrm{d}t}=-\frac{r}{K}(R(t)-R^*)^2\leq 0$$

Replacing the linear functional response in model [11] by the Holling type II functional response leads to the Rosenzweig–MacArthur prey–predator model

$$\begin{aligned}\frac{\mathrm{d}R}{\mathrm{d}t}&=rR\left(1-\frac{R}{K}\right)-\frac{\lambda R}{1+\lambda bR}C\\ \frac{\mathrm{d}C}{\mathrm{d}t}&=\left(\frac{e\lambda R}{1+\lambda bR}-m\right)C\end{aligned} \qquad [12]$$

Provided the following two conditions are met

$$m<\frac{e}{b} \quad \text{and} \quad K>\frac{m}{\lambda(e-mb)}$$

the prey–predator equilibrium is

$$R^*=\frac{m}{(e-bm)\lambda} \quad \text{and} \quad C^*=\frac{er(\lambda K(e-mb)-m)}{K(e-bm)^2\lambda^2}$$

Local stability condition [5] holds for carrying capacities that meet the following constraint:

$$K<\frac{e+bm}{\lambda b\,(e-bm)}:=K_{\mathrm{crit}}$$

Stability condition [6] holds for all parameter values. Using the Dulac criterion it can be proved that no limit cycles exist and the equilibrium is globally asymptotically stable (**Figure 8a**). At the critical carrying capacity K_{crit} the equilibrium undergoes the Hopf bifurcation and for higher carrying capacities a unique globally stable limit cycle exists (**Figure 8b**). This model shows that prey–predator coexistence is not limited to an equilibrium. In fact, as the environmental carrying capacity (K) increases, the stable interior equilibrium is destabilized and a globally stable limit cycle appears. This phenomenon was termed the paradox of enrichment because, contrary to the intuition, enriched environments (i.e., environments with a higher K) do not promote species coexistence at an equilibrium. This paradox is easy to understand, because an increase in the environmental carrying capacity means a weaker bottom-up regulation, thus a less-stable prey–predator population dynamics due to the destabilizing Holling type II functional response.

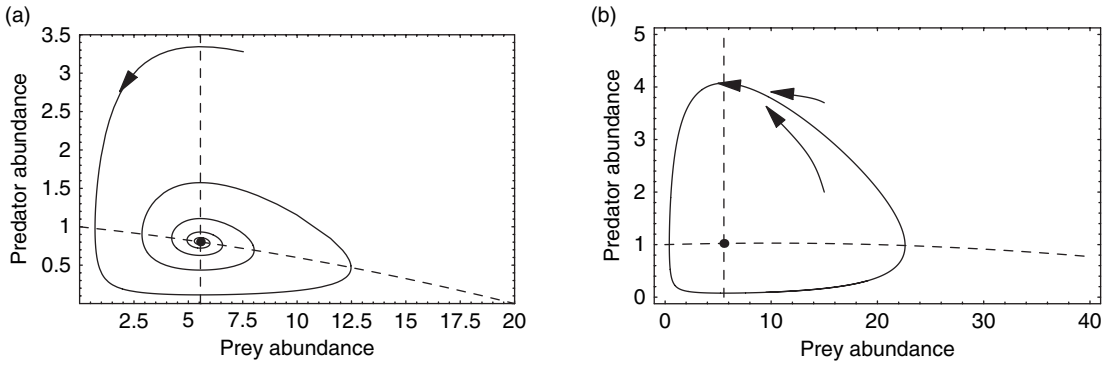

Figure 8 Prey–predator dynamics for the MacArthur–Rosenzweig model [12]. (a) Shows the case where the carrying capacity is below the critical level K_{crit} ($K = 20$) and the equilibrium is globally asymptotically stable. (b) Shows that for higher carrying capacities ($K = 70$) there exists a globally stable limit cycle along which prey coexist with predators. Dashed lines are isoclines. Parameters: $r = 1$, $\lambda = 1$, $e = 0.2$, $h = 0.02$, $m = 1$.

Inhomogeneous Environment

The original Lotka–Volterra model does not consider space explicitly. Instead, it assumes that populations are uniformly distributed in space. Spatial structure can be incorporated in the model either as a continuous variable which then leads to reaction–diffusion models, or as a discrete variable, which leads to patch models. A key question addressed by spatial prey–predator models is the effect of animal dispersal on the stability of prey–predator dynamics.

Stability Caused by Asynchrony in Local Dynamics

Here let us start with a simple example that considers two spatial patches. In one patch prey are vulnerable to predation while the other patch is a complete refuge. We assume that up to S prey can be in the refuge and both vulnerable and invulnerable prey reproduce at the same positive rate r. This means that the refuge is always fully occupied and animals born in the refuge must disperse to the open patch. Population dynamics in the open patch are described by the Lotka–Volterra model

$$\frac{dR}{dt} = r(R+S) - \lambda RC$$
$$\frac{dC}{dt} = (e\lambda R - m)C$$

with equilibrium densities

$$R^* = \frac{m}{e\lambda}, \quad C^* = \frac{r}{\lambda} + \frac{re}{m}S$$

The recruitment of prey to the open patch makes the per capita prey growth rate $r(1 + S/R)$ in the open patch negatively density dependent (i.e., it decreases with increasing prey abundance R) similarly as in the case of the logistic growth. The stability conditions [5] and [6] hold and the above equilibrium is locally asymptotically stable, which supports the general conclusion from other theoretical studies that refugia that protect a constant number of prey have a strong stabilizing effect on prey–predator population dynamics.

This example nicely illustrates stabilizing mechanism of asynchronous oscillations in population densities. In this example, the abundance of prey in the refuge is constant while the abundance of vulnerable prey varies due to demographic changes and recruitment of prey from the refuge. This asynchrony, then leads to the negative density-dependent recruitment rate of prey to the vulnerable patch. While this mechanism is clear in this simple example, it is much less obvious in many models that consider space explicitly.

Now let us consider a more complex prey–predator model in a heterogeneous environment consisting of N patches. The simplest possible case assumes that all patches are identical and animal dispersal is unconditional (random). The question is whether animal dispersal can stabilize population dynamics that are unstable without dispersal. Because unconditional animal dispersal tends to equalize prey and predator abundance across patches, animal dispersal tends to synchronize animal population dynamics and the answer to the above question is negative. However, patch-dependent dispersal rates and/or differences in local population dynamics can lead to asynchrony in local population dynamics, thus to negative density-dependent recruitment rates that can stabilize prey–predator population dynamics on a global scale exactly as in the example with the refuge. Figure 9 shows the stabilizing effect of prey dispersal in a

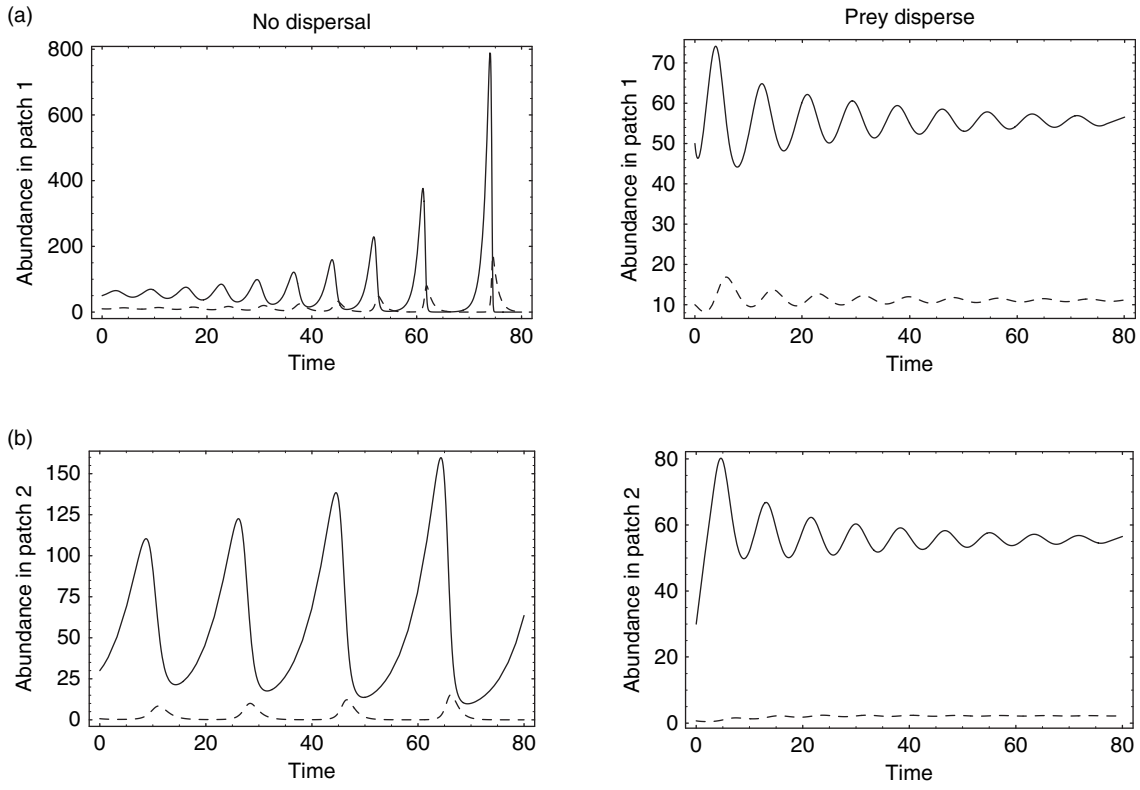

Figure 9 This figure shows the stabilizing effect of dispersal. The left panel shows prey (solid line) and predator (dashed line) dynamics in patch 1 (top panel) and patch 2 (bottom panel) without any dispersal ($\varepsilon_1 = \varepsilon_2 = 0$). These dynamics assume the Holling type II functional response which excludes prey and predator coexistence in either patch. The right panel shows the same system where prey disperse between patches ($\varepsilon_1 = \varepsilon_2 = 1$). Parameters: $r_1 = 1$, $r_2 = 0.2$, $\lambda_1 = \lambda_2 = 0.1$, $e_1 = e_2 = 0.2$, $h_1 = h_2 = 0.02$, $m_1 = m_2 = 1$.

two-patch environment. Population dynamics are described by the following model:

$$\begin{aligned}
\frac{dR_1}{dt} &= r_1 R_1 - \frac{\lambda_1 R_1}{1 + h_1 \lambda_1 R_1} C_1 + \varepsilon_2 R_2 - \varepsilon_1 R_1 \\
\frac{dC_1}{dt} &= \frac{e_1 \lambda_1 R_1}{1 + h_1 \lambda_1 R_1} C_1 - m_1 C_1 \\
\frac{dR_2}{dt} &= r_2 R_2 - \frac{\lambda_2 R_2}{1 + h_2 \lambda_2 R_2} C_2 + \varepsilon_1 R_1 - \varepsilon_2 R_2 \\
\frac{dC_2}{dt} &= \frac{e_2 \lambda_2 R_2}{1 + h_2 \lambda_2 R_2} C_2 - m_2 C_2
\end{aligned}$$

where ε_i $(i = 1, 2)$ describes prey dispersal between patches. Without dispersal, the local prey–predator population dynamics are unstable due to the Holling type II functional response (**Figure 9**, left panel). Prey dispersal (**Figure 9**, right panel) can stabilize population dynamics at an equilibrium. Similarly, dispersal of predators (or both prey and predators) can (but does not necessarily) stabilize population densities. This mechanism is in the roots of deterministic metapopulation dynamics where populations can coexist on the global spatial scale despite local extinctions. The necessary conditions for such global stability are differences in patch or migration dynamics and dispersal rates that are not too high to synchronize local patch dynamics.

Predator Aggregation

Another mechanism that promotes prey–predator coexistence in spatially heterogeneous environments is the tendency of predators to aggregate in patches where prey abundance is high. If u_i denotes the proportion of predators in patch i, the Lotka–Volterra model for two patches is then described by the following set of equations

$$\begin{aligned}
\frac{dR_1}{dt} &= r_1 R_1 - \lambda_1 R_1 u_1 C \\
\frac{dR_2}{dt} &= r_2 R_2 - \lambda_2 R_2 u_2 C \\
\frac{dC}{dt} &= e_1 \lambda_1 R_1 u_1 C + e_2 \lambda_2 R_2 u_2 C - mC
\end{aligned} \qquad [13]$$

provided the predator mortality rate is patch independent. Here R_i is prey abundance in patch i, and all other parameters have the same meaning as those in the Lotka–Volterra model, but they are patch dependent now. The above model assumes that prey do not move between the two patches. When predator distribution (u_i) is fixed, the above model has no interior equilibrium and the prey with smaller value of $r_i/(u_i\lambda_i)$ is always driven to extinction. This is documented in **Figure 10a** where the second prey type (dashed line) is outcompeted by the first prey (solid line). This indirect interaction is

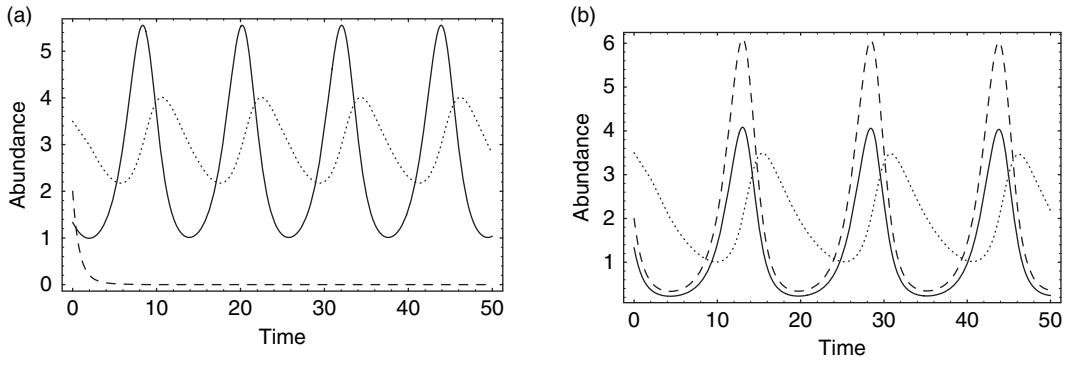

Figure 10 The effect of predator aggregation on population dynamics [13]. (a) Shows the extinction of the competitively weaker prey species due to apparent competition when predator preferences for the two prey are fixed ($u_1 = u_2 = 0.5$). (b) Shows the coexistence of all species when predator preferences are adaptive. Parameters: $r_1 = 1.5$, $r_2 = 0.5$, $m = 0.2$, $e_1 = 0.15$, $e_2 = 0.1$, $\lambda_1 = \lambda_2 = 1$.

mediated by predators (dot line). This kind of indirect competition between the two prey populations, which is mediated by the shared predators, is called apparent competition.

The above analysis assumes that the proportion of predators u_i in each patch is fixed and independent of prey abundances. Now let us consider the situation where predator patch preferences are adaptive. If predator fitness is measured by the per capita predator population growth rate $dC/(Cdt)$ then predators will aggregate in patch 1 ($u_1 = 1$ and $u_2 = 0$) if $e_1\lambda_1 R_1 > e_2\lambda_2 R_2$ and in patch 2 if the opposite inequality holds (here we neglect the travel time between the two patches). This makes predator preferences for either patch dependent on prey abundances and predators switch between the two patches. Predator switching then changes population dynamics of model [13]. It can be proved that prey dynamics get synchronized and prey–predator population dynamics in both patches are described by the Lotka–Volterra-like cycles (**Figure 10b**). In particular, both prey populations coexist with predators indefinitely. This clearly shows that predator aggregation can promote species coexistence without necessarily leading to an equilibrium. In this example, adaptive predator switching relaxes apparent competition between the two prey because at low prey density in one patch predators switch to the other patch. In fact, this type of predator behavior drives the two prey populations to the levels where predator fitness is the same in both patches and predators will distribute across both patches following the 'ideal free distribution'.

Stability and Complexity in Prey–Predators Models

Are more complex systems more stable? R. M. May used an extension of the Lotka–Volterra model to show that this is not the case. Let us consider a food web consisting of n prey and n generalist predators. Assuming that such a system has an equilibrium at which all species exist at positive densities it can be shown that the corresponding eigenvalues occur in pairs, each pair having the form $\xi + i\eta$ and $-\xi - i\eta$. Thus, there are two possibilities. Either real parts of all eigenvalues are zero in which case the equilibrium is neutrally stable exactly as in the case of the Lotka–Volterra prey–predator model [1]. If there exists an eigenvalue with a negative real part, then there must be also an eigenvalue with a positive real part which means that the equilibrium in n-prey–n-predator model is unstable. Thus, it is clear that the n-prey–n- predator system at best has the same stability property as the corresponding Lotka–Volterra prey–predator model. As the number of species increases it is more likely that among the eigenvalues there will be an eigenvalue with a positive real part and the equilibrium will be unstable. This and other models lead to prediction that complexity destabilizes food webs. These studies considered only the case where interaction strengths are fixed. In other words, they exclude the possibility of adaptive predator foraging behavior, or prey escape strategies. Recent studies show that when predator foraging behavior is adaptive (similarly as described in model [13]), the negative relation between food web complexity and community persistence can be reversed.

Summary

The original Lotka–Volterra predator–prey model was extended in many directions, resulting in a vast theoretical literature on prey–predator interactions. Some of these models relax the original assumptions and analyze the properties of the resulting models. Others try to adapt the existing models to some particular empirical prey–predator systems. In this article surveyed some basic theory resulting from the Lotka–Volterra formalism that considers time as a continuous variable was surveyed.

This corresponds to the situation where populations reproduce and die continuously. Alternative formalism is based on models that consider time as a discrete variable. These models can better fit situations where individual reproduction is synchronized. A discrete time alternative to the Lotka–Volterra model is the Nicholson–Bailey host–parasitoid model that can be as well used as a description of prey–predator interactions.

See also: Coexistence; Competition and Competition Models; Dispersal–Migration; Fishery Models; Indirect Effects in Ecology; Mathematical Ecology; Metapopulation Models; Stability.

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Principal Components Analysis

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Introduction

Principal components analysis (PCA) is an ordination technique used primarily to display patterns in multivariate data. It aims to display the relative positions of data points in fewer dimensions while retaining as much information as possible, and explore relationships between dependent variables. In general, it is a hypothesis-generating technique that is intended to describe patterns, rather than test formal statistical hypotheses. Although PCA was originally developed to analyze continuous variables, it can also be used on ordinal and presence–absence data.

PCA is carried out on the response of dependent variables in a multivariate data set. Consequently it is an unconstrained ordination, in which hypothetical causal independent variables are not explicitly included in the analysis. For example, if the abundance of several species of fish (the response or dependent variables) were measured at a range of different sites with different characteristics such as wave exposure (causal or independent variables), the exposure information would not be explicitly included in the analysis. Patterns recovered in PCA are solely a function of relationships between the dependent variables. For this reason, PCA can also be classified as an indirect gradient analysis, in which