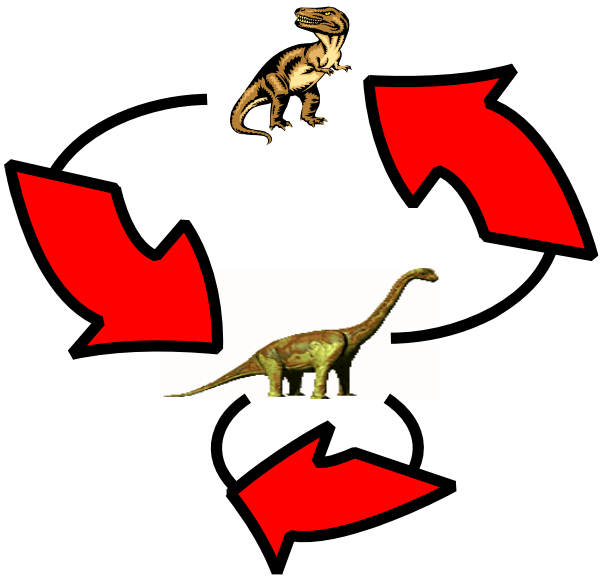
Chapter 5: *PREDATOR-PREY COMMUNITIES*

Including predator dynamics: the numerical response

In the previous chapter, we made the assumption that the predator was not closely coupled with its prey because it was a generalist or had dynamics on a different spatial or temporal scale than its prey. Now we shall make the opposite assumption and assume that the predator is a specialist on this prey species and that its population growth is limited only by the availability of the prey. We have the following foodweb:



Thus, we have the predator being affected by the prey, the prey affected by the predator, and the prey limiting its own abundance, for example, by competition for a resource whose dynamics are fast relative to those of the prey population.

The *numerical response* refers to the fact that the predator is limited by the prey. An increase in the prey density increases food availability to the predator, leading to a higher predator growth rate. If this higher predator growth rate is positive, the predator density will increase, and as a consequence there will be a higher predation rate on the prey. With a numerical response, there is a full feedback loop from prey to predator back to the prey. All links in this foodweb module are strong, and all feedbacks are important. Unlike the case in the previous chapter, neither predator nor prey can be considered constant for point of view of understanding the dynamics of the other. For example, from the prey point of view, density dependence can be generated by the effect that the prey population has on the predator density.

The equations that we shall be considering to exemplify these interaction are as follows:

$$\frac{1}{N}\frac{dN}{dt} = r\left(1-\alpha N\right) - a(N)P,$$
$$\frac{1}{P}\frac{dP}{dt} = cf(N) - m. \qquad (5.1)$$

You will probably notice some similarity between these equations, and the equations (4.1) in Chapter 4 for the case of consumer limited by a single resource. The resemblance is not accidental. Equations (5.1) are a generalization of the consumer-resource equations of Chapter 4, the only differences being that the prey here takes the place of the resource — it is in fact the resource of the predator — and a type I functional response is used in the consumer-resource equations compared with a general functional response here. We could in fact have used a general functional response in the consumer resource equations. What we learn here about predation with various sorts of functional responses can also be applied to consumer-resource dynamics as generalizations of our treatment of them. Our focus now is different. We are interested in the prey and predator as a community module, and so we ask about the joint dynamics of the predator and prey. When focusing on just one of the two species in the interaction, either the predator or the prey, we can think of the other partner as providing a source of density dependence.

Equilibrium properties

These equations commonly have an equilibrium point for the predator and the prey. An important issue here is that the equation for predator growth is equal to zero at a particular prey density, i.e. when

$$cf(N) - m = 0, \text{ i.e. } f(N) = m/c\,. \qquad (5.2)$$

This value of N is the prey equilibrium N^*. To get it we need to solve equation (5.2), which normally means finding the inverse of the function f, which can be in most cases with simple algebra:

$$N^* = f^{-1}(m/c)\,.$$

So for the predator to be at equilibrium, the prey must be at a particular density. From the prey equation we can solve for the predator equilibrium. Equating the prey per capita growth rate to zero we see that

$$r\left(1-\alpha N\right) = a(N)P,$$

which rearranges to

$$P = \frac{r\left(1-\alpha N\right)}{a(N)} = \frac{r\left(1-\alpha N\right)N}{f(N)}. \qquad (5.3)$$

These equations, (5.2) and (5.3) define curves in phase space where the prey population or predator population has zero growth. At their intersection is the equilibrium point. At this point, neither population grows, and so the system stays at this point unless perturbed. The system will not stay at other points on these isoclines because at those points one species is still changing.

These are points then signal that growth of the species changes from positive to negative or negative to positive at this point.

To get the predator equilibrium, we just substitute the prey equilibrium, N^*, into (5.3), to get

$$P^* = \frac{r(1-\alpha N^*)}{a(N^*)}$$

There are some important things to note here about the equilibrium point. The predator density is the ratio of the per capita growth rate, not accounting for predation, viz $r(1 - \alpha N)$, and the attack rate. We can also think of it as the total growth rate without predation, viz $r(1 - \alpha N)N$ divided by the functional response $f(N)$. Thus, the predator density at equilibrium consists of the productivity, as discussed in the previous chapter, divided by the per unit predator harvesting rate. This all makes a whole lot sense. The equilibrium prey density, however, is not so intuitive at first sight. It is determined here by the predator equation, and depends on the maintenance requirement of the predator and the conversion of predator to prey. So if the maintenance requirement is high, the prey density will be high. This has a certain sense. If the predator has a lot of costs or metabolic inefficiency, it will allow the prey population to be high. The way to read it, though, is that the predator needs a large prey population if its maintenance requirement is high.

Predator-prey dynamics

The isoclines given above, divide the plane into regions where the predator and prey populations are increasing versus decreasing. Some examples are given in Figures 5.11 to 5.13, but it is a good exercise to derive them for other cases. These isoclines are normally represented on diagrams called phase plane diagrams, of which Figures 5.2-5.13 are examples.

Although the numerical response leads to density dependence in the prey population, it need not necessarily lead to stable dynamics alone. To see this, we first specialize equations (5.3) to the case of no prey self-limitation ($K = \infty$), and a linear functional response. We then obtain the equations

$$\begin{aligned} \frac{1}{N}\frac{dN}{dt} &= r - a_s P, \\ \frac{1}{P}\frac{dP}{dt} &= c a_s N - m, \end{aligned} \qquad (5.4)$$

which are the classic Lotka-Volterra predator-prey equations. The Lotka-Volterra equations give population cycles as shown in Figures 5.1 and 5.2. Cycles like these can be understood

intuitively in terms of each species responding to the density of the other, but with the entire feed back loop from predator to prey back to prey again having a lag to it. To understand how it works, note that according these equations, the predator population has a zero growth rate at a prey density of $N^* = m/(ca_s)$. Similarly, the prey population has a zero growth rate at $P^* = r/a_s$. Now imagine that at time zero the prey population is growing ($P < P^*$), but the predator population is declining ($N < N^*$). The prey population will continue to grow under this declining predation pressure, but the predator growth rate will be increasing because it prey availability is going up. Eventually, there are enough prey for the predator population to start to increase ($N > N^*$), but not yet too many predators for the prey population to decrease ($P < P^*$). Thus, for a time predator and prey populations increase together until the predator population is so dense that the prey growth rate becomes negative ($P > P^*$), and so the prey starts to decrease. The predator, however, will continue to increase until the prey have dropped below N^*. Then the predator will decrease eventually leading back to the situation where ($P < P^*$) so that the prey are increasing again, while the predators are decreasing ($N < N^*$) where we began the cycle.

In the Lotka-Volterra system, the amplitude of these fluctuations remains constant over time and a cycle occurs. These cycles as explained here simply involve the feedback loop from the predator to the prey, and so are a property of the numerical response. As we shall see, not all predator-prey systems with a numerical response have cycles, but nevertheless a tendency towards fluctuations of some form is a general feature of the numerical response, with the feedback loop working approximately as described above in all cases. The greater complexity that arises in other situations is due the fact that the predator and prey per capita growth is not simply a function of the other species density, and so the change over from increasing to decreasing populations occurs in more complex ways.

The critical issue causing fluctuations in all cases is the fact that when growth of one species changes sign, the growth rate of the other species maintains the same sign for some nonzero amount of time before it changes sign. For example, if the predator and prey are both increasing, according to the equations above, the prey growth rate will be the first to become negative, and the predator will continue to increase after the prey has stopped increasing. The predator will only start decreasing when the prey density has fallen sufficiently, because the predator growth is a function of the prey density, not the prey growth rate. Thus, the predator decline will lag behind the prey decline, and the continuing increase of the predator while the prey is decreasing will tend to hasten the decline of the prey. The prey will eventually increase again either when its own density is low enough or the predator density has become low enough, but again the change in the growth rate of the predator will not occur until prey density has increased sufficiently to allow the predator to increase. This lag in the change in sign of the predator growth rate encourages the trend in the prey by continuing to increase the number of predators while the prey are falling, or continuing to decrease the number of predators while the prey are increasing, enhancing fluctuations, and tending to create a cycle.

Originally Lotka-Volterra cycles were assumed to be an explanation of predator-prey cycles in nature, but it turns out that these cycles are neutrally stable, as shown in Figure 5.3: all curves in this figure are different solutions of the equations with the same parameter values, but differing

initial values, and each of these curves is a neutrally stable equilibrium cycle. Indeed, there is a continuous family of equilibrium cycles expanding from the equilibrium point to cycles of arbitrarily large amplitude. A perturbation of the system, other than all the way to extinction of prey or predator, merely takes the system from one equilibrium cycle to another. This neutral stability does not guarantee persistent cycles in nature, and so has been abandoned as an explanation of those cycles. Moreover, neutrally stable models have the property that some slight modification can destroy the neutral stability. For example, if we replace the type I functional response in equations (5.4) by a type II response, to give

$$\frac{1}{N}\frac{dN}{dt} = r - \frac{a_s P}{1 + a_s \eta N},$$

$$\frac{1}{P}\frac{dP}{dt} = \frac{c a_s N}{1 + a_s \eta N} - m, \tag{5.5}$$

we find that the cycles have become unstable (Figure 5.4), and the populations fluctuate with ever increasing amplitude. The cycles get closer and closer to the predator and prey axes, although in principle they never hit the axes. However, we must admit that the model breaks down at small absolute population numbers because real populations are finite and discrete, not continuous the way such differential equation models assume. Thus, we must interpret such ever expanding cycles as extinction of the predator, or both the predator and the prey. The type II functional response without prey self-limitation does not always give ever expanding cycles. It is also possible for the prey to escape control of the predator (Figure 5.5), i.e. the prey population simply continues growing and the predator never catches up to the point where it causes the prey population to decline. This can occur if

$$r > \frac{c}{\eta} - m$$

for then, high prey abundance and low predator to prey ratio means that the prey grows faster than the predator, which maintains high prey abundance and a low predator to prey ratio. Both populations then increase exponentially.

These findings for a coupled predator and prey system with a type II functional response and no prey self-limitation are consistent with our findings for the uncoupled system of the previous chapter. Here, however, we see a lesser role of initial population densities and a greater role of the specific parameters of the system. The reason is simply due to the numerical response of the predator: It now adjusts to prey density rather than being independent of it. The fact that it may do so with a time lag, combined with the destabilizing effects of a type II functional response, leads to instability.

If we change the functional response to a type III, stability becomes possible, even in the absence of self-limitation, as we might expect from the results of the uncoupled system (Figure 5.6). Again, however, as we might expect from the results of the decoupled system, with larger

handling times, escape from control of the predator is possible (Figure 5.7).

All of the cases of a numerical response that we have considered so far ignore prey self-limitation. Naturally, when prey self-limitation is added, increasingly large population fluctuations, and exponentially increasing populations, are not possible in the long run, even though these effects may still occur in the short term.

When prey self limitation is added to the Lotka-Volterra model, the neutrally stable cycles are replaced by a stable equilibrium (Figure 5.8). There are two possible outcomes for prey self-limitation and a type II functional response. There may be a stable equilibrium point (as shown in Figure 5.9) or a stable equilibrium cycle (Figure 5.10). Whether or not it is a stable equilibrium cycle (often called a *limit cycle*) or a stable equilibrium point depends on the value of K. For small values of K, it is a stable equilibrium point, but as K is increased the dynamics change over to the stable equilibrium cycle. This phenomenon is sometimes referred to as the *paradox of enrichment* (Rosenzweig 1971). The parameter K may be regarded as the richness of the resource base for the prey population. For example, in the mechanistic model of exploitative competition, we found that K was proportional to the intrinsic rate of increase r_R of the resource. A large value of K means that the prey are only weakly self limited, and therefore the destablizing effects of the type II functional response make the equilibrium unstable. However, ever increasing fluctuations are prevented by self-limitation. This idea has an important general prediction: changes in a system that cause a shift from one sort of density dependence to another may alter stability properties, especially when one form of density dependence is stabilizing and the other is destabilizing.

With a type III functional response, both stable equilibrium points and stable equilibrium cycles are possible. The stability of the equilibrium can result from the stabilizing effect of the functional response (Figure 5.11) or from the stabilizing effects of prey self limitation (Figure 5.13). If the equilibrium point occurs where the destabilizing effects of handling time are dominant, then the equilibrium is unstable and a stable equilibrium cycle occurs (Figure 5.12).

Figures 5.11-5.13 show the predator and prey *zero-isoclines*, which are the curves defining the sets of predator and prey densities at which the predator and prey respectively have zero growth rates. The predator isocline is found by equating the predator growth rate to zero, and the prey isocline is found by setting the prey growth rate to zero. The equilibrium point is found at the intersection of the zero isoclines. The stability of the equilibrium can be determined by where it lies on the prey isocline. If it is on a decreasing part of the isocline, then the equilibrium is locally stable, but if it is found on an increasing part of the isocline, it is unstable (see the appendix). Many books demonstrate graphical techniques for determining the stability of predator-prey equations based on the angle of intersection of the predator and prey isoclines. These methods, however, should be used with caution as Bulmer (1976) and Freedman (1987) have pointed out that they are not reliable in general.

We have been studying predation from the perspective of density dependence in the prey population. Functional responses may lead to density dependence as a consequence of

behavioral reactions of the predator to the prey density. The numerical response leads to density dependence through the feed back loop between the prey and the predator. Although this feed back loop leads to an equilibrium and prevents exponential growth, it is not a source of stability on its own, and needs to be combined with a stabilizing functional response, prey self limitation, or some other factor, for stability to arise. Our study of predation shows that density-dependent population growth may well be much more complex than the simple logistic equation implies when that density dependence arises through predation. As we have emphasized repeatedly, density dependence must act on a fast timescale for logistic-like dynamics.

An important feature of our study of predation is that through the numerical response, the predator can lead to any particular prey equilibrium, less than the prey's carrying capacity, based solely on the predator's needs. This is because of the assumption that the predator is limited solely by the prey and will increase or decrease until the prey is at the equilibrium determined by the predator's growth rate as a function of prey density. If the predator's needs are higher than the carrying capacity of the prey, however, the predator cannot persist in the system: it would go extinct. The fact that the predator determines the prey equilibrium independently of the prey dynamics is a somewhat controversial feature of predator-prey models of the sort considered here and has led to the introduction of a body theory called *ratio-dependent predation* in which some form of interference among predators occurs preventing them from being quite so effective that prey density is determined solely by their needs (Hanski 1991).

*He**terogeneity as a stabilizing mechanism: the dynamics of parasitoids and their host*s.

So far we have examined population dynamics of predators and their prey as if all the populations in question have no internal structure. Thus, we have not considered the effects of age-structure, or size structure (Hastings 1984; Hastings and Wollkind 1982), nor have we considered in any detailed way the very real possibility that the populations in question are patchily distributed in space and that the patchiness might matter. The closest we have come is to note that one explanation for a type III functional response has to do with patchy distributions of prey in space, and the reaction of predators that patchiness. From the very early days of predator prey theory, the potential that patchiness might be involved in stabilizing predator-prey dynamics has been suggested (Huffaker 1958; Huffaker et al. 1963). The first mention that I am aware of, however, is not for an ordinary predator of the type that we have considered above, but a parasitoid (Nicholson and Bailey 1935). A parasitoid is a parasite lays its eggs in the body of the host leading ultimately to death of the host. Most insects are attacked by parasitoids, which are usually other insects, either wasps or flies of some sort (Hassell 2000). Although there has been much theoretical consideration of predation in a patchy environment (De Roos et al. 1991; De Roos et al. 1998; Hastings 1977; Hastings 1979), the most well-developed theory is for the special case of parasitoids.

Nicholson and Bailey (1935) were the first to put forward a model for the dynamics of a parasitoid and its host, and their formulation has been the basis of nearly allsubsequent theoretical work. Their model is given in discrete time as follows:

$$N(t+1) = \lambda N(t) e^{-aP(t)}$$
$$P(t+1) = N(t)(1 - e^{-aP(t)}) \quad (5.6)$$

This model is usually explained in the following terms: The hosts are annual organisms (or live just one time period, whatever that may be). A female host has an expected number λ of offspring successfully surviving to the next time period in the absence of parasitism. In the presence of parasitism, this expected number surviving becomes $\lambda\exp(-aP(t))$. The quantity $\exp(-aP(t))$ is usually interpreted as the probability that the host is not found by a parasitoid. It happens to be the zero probability for a Poisson distribution with mean $aP(t)$. So you can think of hosts as receiving Poisson numbers of viable parasitoid eggs, and with host death guaranteed by the presence of any viable parasitoid eggs. Hence, zero parasitoid eggs must be laid in a host for it to survive.

The equations as written assume that exactly one parasitoid offspring emerges from each killed host, the fraction of hosts killed being $1 - \exp(-aP(t))$. There is no problem modifying this equation to have some different number of parasitoids emerging on average from each host killed. So long as that number does not vary with host or parasitoid density, the behavior of the equations is unchanged.

These equations are unstable. They have an equilibrium point, which you should be able to calculate without much problem, but the populations undergo exploding oscillations for all parameter values where a positive equilibrium point exists, viz all situations in which $\lambda > 1$. One example of these oscillations is shown as the thin lines in Figure 5.14 (dashed hosts, solid parasitoids). Nicholson and Bailey (1935) reasoned that this instability might disappear if not all hosts were easily accessible to parasitoids, which was later formalized in a mathematical model (Bailey et al. 1962). The thick lines in Figure 5.14 are the output of a simplified version of this model where half the hosts are completely protected from parasitism, and the other half are fully exposed. As you will note, extreme instability has been replaced by strong stability. An understanding of how this works is given in Figure 5.15. The solid curve gives the host surivival fraction defined by the Nicholson-Bailey model, viz $\exp(-aP(t))$. The point A is the host surival fraction for those hosts that are not exposed to parasitoids, viz 1. Naturally, the solid curve gives host surivival in patches that are exposed to parasitoids. Overall survival, as a function of average exposure to parasitoids is given by the dashed curve, which is obtained as the mid-point of the lines joining the point A to the solid curve. For example, consider the point B. A parasitoid exposure of 75 for exposed patches will give an average for the system of 75/2, which is on the x axis below the point M'. The average survival, however, for parasitoids in the system is given by the point M. Rather than M' being the survival level for average exposure, the result is actually M. Following the same procedure for all other points on the solid curve yields the dashed curve.

The thing to note about the dashed curve is that it is not as steep at the solid curve, and in fact levels off above zero (in this case at 50%, but that is not so important as the fact that it has an

asymptote above zero). Thus, the effect that increases in parasitoid density can have on host survival is greatly moderated relative to the Nicholson-Bailey model. Host crashes due to increases in parasitoid numbers are thus greatly reduced, and the result is damped oscillations in numbers rather than the wildly expanding oscillations seen in the Nicholson-Bailey model.

One version of the model of Bailey et al (1962), which has been popularized by May (1978), replaces the Poisson zero term defining host survival with the zero term of the negative binomial distribution. This change means that the equations above are replaced by

$$N(t+1) = \lambda \frac{N(t)}{\left(1 + aP(t)/k\right)^k}$$
$$P(t+1) = N(t) - \frac{N(t)}{\left(1 + aP(t)/k\right)^k} \qquad (5.7)$$

where k is parameter of the negative binomial that determines the degree to which eggs are clumpily distributed over hosts. Smaller k means more clumped. Whenever k is less than one in value, these equations give a stable equilibrium. For k greater than 1, the system remains unstable. This negative binomial model can be interpreted in a number of ways. First clumping of eggs could be due the fact that some hosts are simply more difficult to find than others, which is the original explanation of Bailey et al (1962). Alternatively, it could be what Chesson and Murdoch (1986) called aggregation independent of host density. This means that, for whatever reason, the parasitoid distribution is patchy in space, but patchy in a way that is independent of the host distribution in space. Surprisingly, this seems to be the most commonly observed pattern of host and parasitoid distributions in nature (Pacala and Hassell 1991). Note that being more difficult to find, or happening to be in a patch not greatly visited by parasitoids, both lead to lower levels of risk for individual hosts than the average risk experienced by hosts. In other words there is variation between host individuals in the level of risk they experience. Such variation in risk has a stabilizing effect in host-parasitoid models, almost regardless of the cause (Chesson and Murdoch 1986; Hassell 2000; Hassell et al. 1991). Such variation in risk might also be caused by aggregation of parasitoids in relation to host density, and also by aggregation inversely to host density.

Hassell et al (1991) proposed that the coefficient of variation in risk of parasitism over host individuals might provide a suitable approximate predictor of stability in host-parasitoid systems. A values greater than 1 should mean that the system is stable, and less than 1 unstable. For the negative binomial model, these situations correspond to $k < 1$, and $k > 1$, respectively. Although one such fixed value of the coefficient of variation is hardly likely to be applicable broadly, it does work out roughly correctly for simple models derived from the Nicholson-Bailey model, with a variety of potential causes of variation in risk. Following on from these models are a variety of other models of patchy distributions and metapopulation dynamics of hosts and parasitoids, all of which do create variation in risk, and many of which lead to stable dynamics on large spatial scales (Hassell 2000).

Results similar to those for host-parasitoid systems have been found for predator-prey systems, although they are very much less studied. Spatially-explicit models of predator-prey systems, with locally dispersing predators and prey, local interactions between predators and prey and finite local population sizes, often show stable behavior of the predator-prey interaction at the landscape level, even though dynamics locally in space are unstable (McCauley et al. 1993). These models do not incorporate environmental heterogeneity, but populations are patchy locally in space simply due various components of demographic stochasticity (stochasticity in reproduction, local dispersal and mortality). Such patchiness leads to variation in risk, which is a potential explanation also for the stability of these models.

Predator-prey metapopulation models

Many of the predator models currently being studied theoretically are rather too complex for easy summary and synthesis, but some of the essential features can be deduced from the study of presence-absence metapopulation models. Starting out with the Levins model from Chapter 3 (where p here is the proportion of patches occupied by a particular species),

$$\frac{1}{p}\frac{dp}{dt} = c(1 - p) - \varepsilon , \tag{5.8}$$

we can modify the extinction rate by adding a predator. We can do this by replacing ε with $\varepsilon + ay$, where y is the density of predators in the system as a whole. The equation then becomes

$$\frac{1}{p}\frac{dp}{dt} = c(1-p) - \varepsilon - ay . \tag{5.9}$$

The prey equilibrium now becomes

$$p^* = 1 - \frac{\varepsilon}{c} - \frac{ay^*}{c} , \tag{5.10}$$

assuming that the predator does come to an equilibrium. To find out whether this happens, and what the equilibrium value will be, we have to specify the predator equation. The predator need not have a metapopulation structure or at least not one on the same scale as the prey. Indeed, it is not uncommon for predators to have larger scales of movement than their prey organisms. One real world example is potentially the system in southeastern Australian forests involving the greater glider as prey and powerful owls as the predator (Possingham et al. 1994).

Simple assumptions for the predator are then a linear per capita rate of increase as a function of the frequency of prey patches, to give the predator equation

$$\frac{1}{y}\frac{dy}{dt} = \kappa ap - d, \tag{5.11}$$

where κ is the rate of conversion of decimated prey populations into predators, and d is the predator maintenance requirement, namely the number of predator units per predator that must be gained per unit time through prey consumption to balance losses due to mortality and metabolism.

In this model, note that the predator equation determines the prey equilibrium as

$$p^* = \frac{d}{\kappa a} \tag{5.12}$$

a circumstance that comes directly from the fact that the predator is limited only by the prey, and therefore can only be at equilibrium for one particular value of prey abundance. The predator equilibrium found from substituting back in equation (5.10) is

$$y^* = \frac{1}{a}\left[c\left(1 - \frac{d}{\kappa a}\right) - \varepsilon \right], \tag{5.13}$$

which is simply per patch rate at which prey are in excess for equilibrium without the predator, scaled by the per unit rate at which predators dispatch prey patches.

This equilibrium is positive provided simply that

$$1 - \frac{\varepsilon}{c} > \frac{d}{\kappa a} \tag{5.14}$$

which makes sense in saying that the prey equilibrium without the predator must be higher than the prey equilibrium with the predator. This condition is discussed in more detail below for second example, but you should note that when this equilibrium is feasible, it is globally stable. A couple of notable things about the equilibria are

1. The prey parameters have no effect on prey equilibrium beyond the constraint above, which is otherwise simply determined by the predator equilibrium–characteristic of predation limited only the prey.
2. The total prey consumption rate by the predator at equilibrium is p^*ay^*, which equals

   $$cp^*\left(1 - p^*\right) - \varepsilon p^*.$$

   This is the amount by which prey patch occupancy grows before predation is accounted for, as it clearly must be. In per capita prey terms, this is

   $$c\left(1 - p^*\right) - \varepsilon.$$

   The predator reduces p^*, which reduces competition for unoccupied patches between

propagules, the predator takes the excess production freed up by competition. If not enough production can be freed up by competition to allow the predator and prey to be at equilibrium, i.e. if p^* from the predator equation is not less than $1 - \varepsilon/c$, the prey equilibrium without predation, then predator cannot persist in the system and will go extinct.

3. Favorable prey parameters (high c and low ε) lead to a high predator density.
4. A high predator attack rate leads to both high prey suppression and low predator densities, but predator density is not monotonic in the attack rate, and intermediate attack rates lead to the highest predator densities–why should this be so?
5. Low predator maintenance requirements (d) and high conversion of prey to predator (κ) lead to both low prey frequency and high predator density.
6. Habitat fragmentation will make the prey less abundant on the landscape, and the predator can go extinct because it cannot meet its energy needs when its encounter rate with prey patches is too low. That will occur even if the predator can distinguish the correct habitat, and does not waste time in or traveling between incorrect habitat, because the prey remains less frequent even in the remaining available habitat, after destruction.

A model in which predators and prey both have a metapopulation structure is

$$\begin{aligned} \frac{1}{p}\frac{dp}{dt} &= c(1 - p - y) - \varepsilon - ay \\ \frac{1}{y}\frac{dy}{dt} &= ap - d, \end{aligned} \tag{5.15}$$

where y is the frequency of patches occupied by predators, and p is the frequency of patches occupied by prey alone. For our analysis of these equations is it immaterial whether or not prey are present in the predator patches, but the important thing to note is they do not contribute prey colonists of other patches, and they are not colonizable by prey. In effect, once a predator has found a patch of prey, those prey are no longer able to colonize other patches, and are not counted in p. Moreover, the fraction, y, of patches occupied by predators, is unavailable for colonization by prey. In other words, a patch occupied by a predator is unavailable for conversion back into a prey-only until the predator goes extinct in that patch, which it will with probability d per unit time. This model is identical to the predator-prey model of Nee *et al* (1997).

It is worth noting that this model is equivalent at the whole population level to Lotka-Volterra predator-prey dynamics with prey self-limitation even though the assumptions at the level of an individual patch are rather different. This is most apparent if you rewrite the RHS of the prey equation in the form $c(1 - p) - \varepsilon - (c + a)y$. Note that the meanings of the parameters in the two situations are quite different and would be measured empirically in very different ways. Thus, when the metapopulation model is appropriate, small-scale studies estimating prey self-limitation, attack rates and predator maintenance requirements, would not be at all suitable for predicting outcomes at the level of the whole system. Instead, to scale up to the whole system,

local colonization and extinction rates would be the critical parameters.

The model above of logistic self-limitation and a linear functional response in homogeneous space is mathematically equivalent to the equations (5.15), and so we can understand how they behave from our knowledge of those situations. The first thing to note is that the predator equation specifies the prey equilibrium:

$$p^* = \frac{d}{a}. \tag{5.16}$$

It specifies the frequency of prey patches necessary to sustain the predator. For prey frequencies above this level, the predator increases in frequency. Below this level, the predator decreases in frequency.

Substituting the prey equilibrium in the prey equation and solving for the predator equilibrium gives the value

$$y^* = \frac{c(1 - d/a) - \varepsilon}{c + a}. \tag{5.17}$$

For this predator equilibrium to be feasible, it is necessary that

$$\frac{d}{a} < 1 - \frac{\varepsilon}{c}. \tag{5.18}$$

In other words, it is necessary that the prey equilibrium in the presence of the predator be less than the prey equilibrium the absence of the predator. This makes sense, because the growth of the prey is necessarily negative when the prey is below the equilibrium it has without the predator. Hence, it could not possibly sustain the predator when its equilibrium alone is less than its equilibrium with the predator. This condition , however, is the sole condition for a feasible equilibrium.

There are a number of ways in which you can see that the equilibrium is stable, indeed globally stable. You can say simply that you know that it is from previous information on Lotka-Volterra models with prey self-limitation, or you can satisfy yourself again by doing an isocline analysis, and perhaps a local stability analysis as a cross check, or you could find a Lyapunov function for the system.

How do the parameters affect the equilibria? There are a lot of similarities with the case where the predator does not have a metapopulation structure. One difference is that there is no κ here, because on attack, one prey patch is converted into one predator patch. If you like, you could say $\kappa = 1$, but you could not vary it. The only other difference is that because the predator affects colonization as well as extinction, the denominator of the predator equilibrium (5.17) is c

+ a. This difference means that the predator equilibrium no longer increases linearly with the prey colonization rate. It does increase still, but it asymptotically approaches the value $1 - d/a$ at which point every patch has either the predator or prey present because as soon as the predator goes extinct in a patch, it is colonized by prey because of their very large c. Despite the difference here, this predator is affected by habitat fragmentation in pretty much the same way as a free ranging predator. If the frequency of suitable patches becomes too low for its equilibrium, it must go extinct too.

These examples are just two of a variety of different ways of formulating predator-prey metapopulation models. Hastings (1977) gives some other formulations.

Other topics

There are many other topics in the study of predation that we might have considered including interference among predators, prey avoidance behavior, and prey refuges. All of these are either sources of density dependence or modify the effects of density dependence. Check out Taylor (1984), Taylor and Pekins (1991) and Hassell (1978; 2000), for more details.

APPENDIX: Stability analysis of predator-prey equations

In Chapter 5 we studied the behavior of predator-prey equations in the following form

$$\frac{1}{N}\frac{dN}{dt} = g(N) - a(N)P,$$
$$\frac{1}{P}\frac{dP}{dt} = cf(N) - m, \qquad (1)$$

where the logistic term present in the text equations (1) has been generalized to $g(N)$, which is simply some decreasing function of N representing self-limitation.

Phase-Plane Diagrams for Predator-Prey Models

In the previous chapters, we found the idea of equilibrium especially useful. For the most part, we dealt only with a single differential equation with only one species population changing over time. Here we have a system of two differential equations, and an equilibrium is not a single number but a pair (N^*, P^*) representing the values of N and P for which the growth rates dN/dt and dP/dt are simultaneously zero. Thus, if prey and predator densities are at the respective values N^* and P^*, the predator and prey populations do not change with time. The pair (N^*, P^*) can be plotted as a point in two-dimensional space, where the dimensions are respectively predator and prey density. This particular two-dimensional space is called the phase plane for the system of differential equations. For example, Figures 5.2 to 5.13 are all given in phase plane, and the equilibrium is given as a dot in the middle of each figure . The point **0** = (0, 0) is always an equilibrium in a closed system predator-prey system, and also there are equilibria of

the form (N^{**}, 0), (0, P^{**}) in both or one of the species is extinct, and the remaining species is at its single-species positive equilibrium point, indicated by ** in this notation. Of most interest, however, are equilibria in which both predator and prey have positive densities, and these are obtained as a solution to the pair of equations

$$\frac{1}{N}\frac{dN}{dt} = 0,$$
$$\frac{1}{P}\frac{dP}{dt} = 0. \tag{2}$$

Each of these equations defines a curve in phase space, called the zero growth isocline for the species. These zero-growth isoclines are depicted in Figures 5.11-5.13. Their intersections define positive equilibrium points. However, they provide much more information than simply locating the equilibrium: they divide phase space into the region where the population increases and the region where it decreases. The isocline itself is the line where the population is neither increasing nor decreasing.

For our predator-prey model, the equilibrium equations (2) can be written explicitly as

$$g(N) - a(N)P = 0,$$
$$cf(N) - m = 0. \tag{3}$$

and these rearrange to

$$P = g(N)/a(N) \tag{4}$$

(the equation for the prey zero-growth isocline) and

$$f(N) = m/c \tag{5}$$

(the equation for the for predator-zero growth isocline).

A simple example: Lotka-Volterra isoclines

For the Lotka-Volterra model, equations (4) and (5) become simply

$P = r/a_s$

and

$N = m/ca_s$.

Thus, they plot as a horizontal line and a vertical in phase space. If you think about what these mean for the joint growth of these populations, you can see that oscillations in population densities must occur with the predator oscillations following the prey oscillations. A more detailed analysis (e.g. linear stability analysis--see discussed below) is needed to see that the cycles are neutrally stable.

Returning to the general equations (4) and (5), it is important to note that the predator zero growth isocline specifies just one value of N. Because the functional response is monotonic, equation (5) has a unique solution N^*, which is the prey equilibrium. For this reason, the predator isocline just appears as a vertical line at N^* in the Figures 5.11-5.13. This outcome reflects the fact that the predator equation depends on the prey density but not the predator density--it is limited by prey, but not itself in a direct way. (It does of course limit itself indirectly by feed back through the prey.) The prey isocline takes an intuitive form too: it specifies the value of P to be the ratio of the attack rate to self-limited growth, and it is thus just the predator density that makes predation balance self-limited growth. The equilibrium point for the system is defined by the intersection of these two isoclines.

Note that the isoclines do not always intersect at positive values for both N and P, and in such cases there is no positive equilibrium for the populations--the predator and prey populations cannot possibly remain constant at any positive predator and prey densities. This scenario has not been graphed here. Note that the ratio m/c determines whether there is a prey equilibrium. If m/c is larger than the maximum value that the functional response, f, can take, then it is impossible for the predator get enough to eat no matter how many prey there are. Thus, the predator must go extinct. In this case there is no predator isocline--there are no points in phase space where the predator's growth rate is zero. Another situation in which there is no equilibrium point is when $f(N^{**}) < m/c$, i.e. when the value of the functional response at the prey equilibrium, in the absence of predation, is less than the prey intake needed to sustain the predator. In this case, it is not the case that predator cannot consume enough of the prey no matter how abundant they may be, but the prey are self-limited below the minimum level needed to sustain the predator. You should think about where the predator isocline would be located in this instance.

You can use the isoclines to mark the phase-plane diagrams with angle brackets to indicate the possible directions of community change. Vertical arrows indicate the sign of predator population change, while horizontal arrows refer to the prey. The actual direction of the population trajectories in phase-space is bracketed by the predator and prey arrows. Note that the predator is decreasing if to the left of the predator isocline (there are too few prey there to sustain the predator), and increasing to the right of the predator isocline. Similarly the prey are increasing below the prey isocline, where the prey growth rate exceeds mortality due to predation. Prey decrease above the prey isocline.

These qualitative properties of the growth rate can be used to draw some conclusions about the

general properties of the predator-prey interaction. In the case where there is a positive equilibrium, it will be the only positive equilibrium in this model (can you see why?), and the predator and prey population densities will exhibit a general anticlockwise motion about this equilibrium. This means that predator and prey numbers will oscillate through time with oscillations in predator density lagging behind those of the prey. This makes intuitive sense doesn't it? Although we can see that oscillatory behavior should be found away from equilibrium, we cannot tell whether these oscillations will become dampened over time such that the population densities approach the equilibrium value, or will behave in some other manner. To tell this we need to delve more deeply into the equations.

The shape of the prey isocline

In this predator-prey model, the equilibrium, if it exists, is locally stable if it occurs on a decreasing part of the prey isocline. Conversely, if the equilibrium is on an increasing part of the prey isocline the equilibrium is unstable. These facts are demonstrated below in the section on linear stability analysis, but you can see them intuitively by tracing possible trajectories on a phase plane diagram using the angle bracket method mentioned above. One important issue here is that when there is self-limitation, prey density has to be bounded -- it can never get above carrying capacity because growth rates are always negative there. It is then not too difficult to show that predator the predator population is bounded too, and neither prey nor predator can approach zero (Bulmer 1976). Instability of the equilibrium then means that a limit cycle must occur. You can understand this intuitively from the fact that the trajectories of the solutions of a differential equation cannot cross one another for do so would mean that two sets of population growth rates would occur at the same point in space at different times. Since we are not allowing such time dependence here, this cannot happen. Thus, as population trajectories spiral out from equilibrium but are bounded in finite space, they must pile up as they expand out, and the curve at which they pile is in fact the equilibrium cycle that they approach.

When is the prey isocline increasing or decreasing? We can figure this out from equation (4). We just need to know when P, according to (4) is increasing or decreasing as a function of N. This is just the same as when $\ln P$ is increasing or decreasing as a function of N. Taking logs and differentiating, we find

$$\frac{d \ln P}{dN} = \frac{g'(N)}{g(N)} - \frac{a'(N)}{a(N)} \tag{6}$$

which is just a comparison of relative rate of change of self-limitation and the relative rate of change of the attack rate as N changes. Since $g'(N)$ is negative (normally), then the equilibrium will certainly be stable if the attack rate is increasing at equilibrium--a type III functional response and low N. For a type II functional response, or a type III at high N, $a'(N)$ will be negative because $a(N)$ will be decreasing. Thus, whether or not the equilibrium is stable depends on which is changing relatively more rapidly at equilibrium, the attack rate (which is destabilizing in these cases) or self-limitation (which is stabilizing).

Prey isocline for type II functional response and logistic self-limitation

The sign of the slope of the prey isocline is $g'(N)/g(N) - a'(N)/a(N)$ because this is the slope of $\ln P$ as a function of N. For a model with a type II functional response and logistic self limitation of prey, this slope is

$$\frac{a_s\eta}{1+a_s\eta N} - \frac{1}{1/\alpha - N} = \frac{a_s\eta/\alpha - 1 - 2a_s\eta N}{(1+a_s\eta N)(1/\alpha - N)} \tag{7}$$

which is zero if

$$N = \frac{1}{2}\left(\frac{1}{\alpha} - \frac{1}{a_s\eta}\right). \tag{8}$$

Now the formula (4) for the prey isocline is in this case

$$P = \frac{r}{a_s}\left(1-\alpha N\right)\left(1+a_s\eta N\right) \tag{9}$$

which is a quadratic equation in N. For $N < 1/\alpha$, it is positive, but for $N = 1/\alpha$ it becomes zero, i.e. cuts the prey axis. For $N = 0$, $P = r/a_s$, i.e. cuts the predator axis at $P = r/a_s$. The paradox of enrichment arises as α decreases (i.e. K increases). We can see that for $\alpha > a_s\eta$, the prey isocline always has negative slope. But for $\alpha < a_s\eta$ it is hump shaped, and as α is decreased (K is increased), this hump moves to the right. With all other parameters fixed, we see that the first of all increasing K first leads to a situation where the predator can support the prey, i.e. when $f(K) > m/c$, meaning $N^* > 0$. At first, this will be a stable interaction (Figure 5.9). As K is increased further then the position (8) of the hump will move to the right of N^* and the equilibrium will become unstable. The outcome will then be a stable equilibrium cycle (Figure 5.10), and these cycles become indefinitely larger in magnitude as K is increased. As mentioned in the text, this set of transitions as K is increased is referred to as the paradox of enrichment.

Linear stability of the general predator-prey model

In general, the predator-prey equations (1) cannot be solved explictly and so we must use various other techniques to determine the properties of the solution. Numerical solution using a computer is a useful technique but is limited in the generality that it can achieve. Nevertheless, those of you with interests in numerical analysis can gain a great deal of insight from numerical solutions of predator prey equations. The first technique that we shall apply in class is approximation of the system near equilibrium by a linear system. We have already made use of this technique in analyzing single-species models.

The equations are of the general form

$$\frac{dN}{dt} = F(N, P)$$
$$\frac{dP}{dt} = G(N, P) \tag{10}$$

and we seek to replace $F(N, P)$ and $G(N, P)$ by linear functions that are accurate approximations near the equilibrium point (N^*, P^*). In the case of a function of one variable, we found that the linear approximation through any particular point is the tangent line through that point. For a function of two variables, the tangent plane gives the appropriate linear approximation. To write down the equation for the tangent plane we define $F_1(N, P) = \partial F/\partial N$, $F_2(N, P) = \partial F/\partial P$ and then the equation can be written as

$$R = F(N^*, P^*) + (N - N^*)F_1(N^*, P^*) + (P - P^*)F_2(N^*, P^*) \tag{7}$$

Recalling that at equilibrium $F(N^*, P^*) = 0$, and defining $n = (N - N^*)$, $p = (P - P^*)$, we have

$$R = nF_1(N^*, P^*) + pF_2(N^*, P^*). \tag{8}$$

Since $dx/dt = dN/dt$, $dy/dt = dP/dt$, this gives us the approximation

$$\frac{dn}{dt} = nF_1(N^*, P^*) + pF_2(N^*, P^*)$$
$$\frac{dp}{dt} = nG_1(N^*, P^*) + pG_2(N^*, P^*) \tag{9}$$

or

$$\frac{d\mathbf{z}}{dt} = \mathbf{A}\mathbf{z} \tag{10}$$

where $\mathbf{z} = (n, p)'$ and

$$\mathbf{A} = \begin{bmatrix} F_1(N^*, P^*) & F_2(N^*, P^*) \\ G_1(N^*, P^*) & G_2(N^*, P^*) \end{bmatrix}. \tag{11}$$

The linear differential equation (11) has the solution

$$\mathbf{z}(t) = e^{\mathbf{A}t}\mathbf{z}(0) \tag{12}$$

which can be expressed in terms of eigenvalues and projections on eigenvectors as

$$\mathbf{z}(t) = e^{\lambda_1 t}\mathbf{M}_1\mathbf{z}(0) + e^{\lambda_2 t}\mathbf{M}_2\mathbf{z}(0)\,, \tag{13}$$

where the λs are the eigenvalues of **A,** and the **M**s project the initial value **z**(0) on the corresponding eigenvectors. If the real parts of these eigenvalues are negative, then **z**(*t*) converges to **0**, i.e. the original system of equations (10) is locally stable at the equilibrium $F(N^*, P^*)$. If one of these real parts is positive, then the equilibrium is unstable. If eigenvalues are complex, then the solutions to the equation will spiral toward or away from equilibrium. Otherwise, near the equilibrium, the trajectories are asymptotically straightlines. Further away from equilibrium, however, they must show a spiral tendency in these predator-prey models.

To evaluate the matrix **A** for the predator-prey model we note that $F(N, P) = N[g(N) - a(N)P]$ and $G(N, P) = P[-m + cf(N)]$, where the terms in parentheses are 0 at equilibrium. Using these facts and differentiating, we obtain

$$\mathbf{A} = \begin{bmatrix} N^*[g'(N^*) - a'(N^*)P^*] & -f(N^*) \\ cP^*f'(N^*) & 0 \end{bmatrix} \tag{14}$$

To judge stability from $\mathbf{A} = (a_{ij})$ we use the Routh-Hurwitz criterion which says that the eigenvalues have negative real parts if

$$a_{11}a_{22} - a_{12}a_{21} > 0 \tag{15a}$$

and

$$a_{11} + a_{22} < 0. \tag{15b}$$

Note that (15a) implies that the real parts of the eigenvalues have the same sign, while $a_{11} + a_{22}$ is the sum of these real parts.

For our predator-prey model we find that

$$a_{11}a_{22} - a_{12}a_{21} = cP^*f(N^*)f'(N^*) \tag{16a}$$

which in general will be positive because the functional response is generally strictly increasing. For (15b) we get

$$a_{11} + a_{22} = N^*[g'(N^*) - a'(N^*)P^*]. \tag{16b}$$

Now from equation (4) we know that $P^* = g(N^*)/a(N^*)$. Thus $g'(N^*) - a'(N^*)P^* = g(N^*)[g'(N^*)/g(N^*) - a'(N^*)/a(N^*)]$, which from equation (6) and the fact that $g(N^*)$ has to be positive at equilibrium, means that the sum of the eigenvalues has the same slope as the prey isocline at equilibrium. This means that if the slope of the prey isocline is negative at equilibrium, the equilibrium will be locally stable. On the other hand if this slope is positive, the eigenvalues have positive real parts and the equilibrium is unstable. The case where the slope is

0 corresponds to 0 real parts because the real parts of the eigenvalues must have the same sign. These results tie in exactly with those derived in Chapter 4 for the case of no numerical response, for condition (16b) is simply the condition that the per capita growth rate have negative slope as a function of N at equilibrium which is the condition we used to determined stability graphically in Chapter 4.

These results tell us whether the equilibrium is stable or not. Further information tell us whether the system spirals into or away from equilibrium, or moves towards or away from equilibrium monotonically. This information comes from the discriminant,

$$(a_{11} + a_{22})^2 - 4(a_{11}a_{22} - a_{12}a_{21}). \tag{17}$$

If this quantity is positive the eigenvalues are real, and the system will not spiral, but instead approach or move away from equilibrium without spiraling around it. However, if the discriminant (17) is negative, the eigenvalues are complex, and the system will spiral when close to equilibrium.

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