Chapter 10

The Population Dynamics of Predation

10.1 Introduction: patterns of abundance and the need for their explanation

We turn now to the effects of predation on the population dynamics of the predator and its prey, where even a limited survey of the data reveals a varied array of patterns. There are certainly cases where predation has a profoundly detrimental effect on a prey population. For example, the 'vedalia' ladybird beetle (*Rodolia cardinalis*) is famous for having virtually eradicated the cottony cushion-scale insect (*Icerya purchasi*), a pest that threatened the California citrus industry in the late 1880s (see Section 15.2.5). On the other hand, there are many cases where predators and herbivores have no apparent effect on their prey's dynamics or abundance. For example, the weevil *Apion ulicis* has been introduced into many parts of the world in an attempt to control the abundance of gorse bushes (*Ulex europaeus*), and it has often become well established. The situation in Chile, however, is fairly typical, where, despite eating on average around half, and sometimes up to 94%, of the seeds produced, it has had no appreciable impact on gorse invasiveness (Norambuena & Piper, 2000).

There are also examples that appear to show predator and prey populations linked together by coupled oscillations in abundance (Figure 10.1), but there are many more examples in which predator and prey populations fluctuate in abundance apparently independently of one another.

It is clearly a major task for ecologists to develop an understanding of the patterns of predator–prey abundance, and to account for the differences from one example to the next. It is equally clear, though, that none of these predator and prey populations exist as isolated pairs, but rather as parts of multispecies systems, and that all these species are affected by environmental conditions. These broader issues of what determines a species' abundance are taken up again in Chapter 14. However, as with any complex process in science, we cannot understand the full complexity without a reasonable understanding of the components – in this case, populations of predators and prey. Hence, this chapter deals with the consequences of predator–prey interactions for the dynamics of the populations concerned.

The approach will be firstly to use simple models to deduce the effects produced by different components of the interactions, teasing out the separate effects before seeking to understand those effects in combination. Then, field and experimental data will be examined to see whether the deductions appear to be supported or refuted. In fact, simple models are most useful when their predictions are *not* supported by real data – as long as the reason for the discrepancy can subsequently be discovered. Confirmation of a model's predictions provides consolidation; refutation with subsequent explanation is progress.

10.2 The basic dynamics of predator–prey and plant–herbivore systems: a tendency towards cycles

There have been two main series of models developed as attempts to understand predator–prey dynamics. Both will be examined here. The first (Section 10.2.1) is based on differential equations (and hence, applies most readily to populations in which breeding is continuous), but relies heavily on simple graphical models (Rosenzweig & MacArthur, 1963). The second (Section 10.2.3) uses difference equations to model host–parasitoid interactions with discrete generations. Despite this taxonomic limitation, these models have the advantage of having been subject to rigorous mathematical exploration. (We have also noted previously that there are a very large number of important parasitoid species.) Although the two series of models are explained separately, they have, of course, a common aim (to advance our understanding of predator–prey dynamics), and they can increasingly be seen as ends of a discrete-to-continuous spectrum of mathematical approaches.

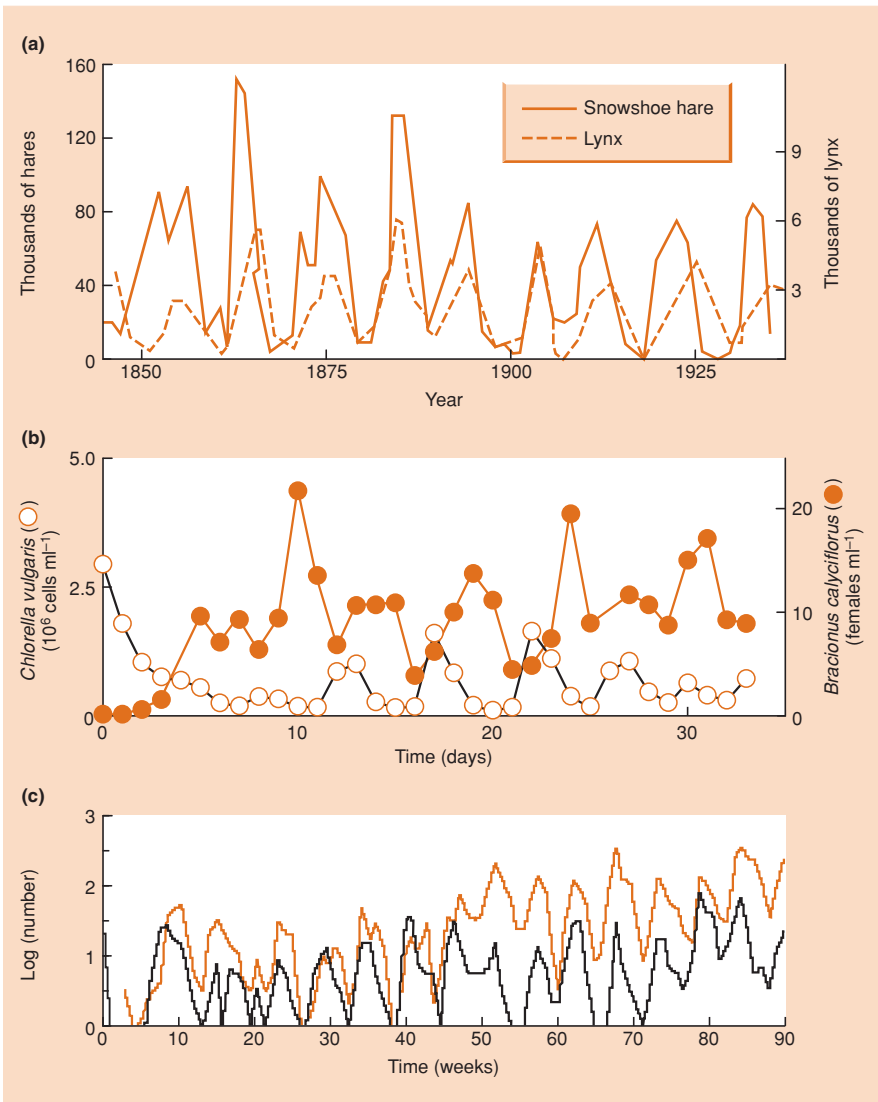

Figure 10.1 Coupled oscillations in the abundance of predators and prey. (a) The snowshoe hare (*Lepus americanus*) and the Canadian lynx (*Lynx canadensis*) as determined by the number of pelts lodged with the Hudson Bay Company. (After MacLulick, 1937.) (b) Parthenogenetic female rotifers, *Bracionus calyciflorus* (predators, ●), and unicellular green algae, *Chlorella vulgaris* (prey, ○) in laboratory cultures. (After Yoshida *et al.*, 2003). (c) The parasitoid *Venturia canescens* (——) and its moth host *Plodia interpunctella* (——) in laboratory cultures. (After Bjørnstad *et al.*, 2001.)

10.2.1 The Lotka–Volterra model

The simplest differential equation model is known (like the model of interspecific competition) by the name of its originators: Lotka–Volterra (Volterra, 1926; Lotka, 1932). This will serve as a useful point of departure. The model has two components: P, the numbers present in a predator (or consumer) population, and N, the numbers or biomass present in a prey or plant population.

We assume initially that in the absence of consumers the prey population increases exponentially (see Section 5.9):

$$dN/dt = rN. \qquad (10.1)$$

But prey individuals are removed by predators at a rate that depends on the frequency of predator–prey encounters. Encounters will increase with the numbers of predators (P) and the numbers of prey (N). However, the exact number encountered and successfully consumed will depend on the searching and attacking efficiency of the predator: a, sometimes also called the 'attack rate'. The consumption rate of prey will thus be aPN, and overall:

$$dN/dt = rN - aPN. \qquad (10.2)$$

the Lotka–Volterra prey equation

In the absence of prey, predator numbers in the model are assumed to decline exponentially through starvation:

$$dP/dt = -qP, \qquad (10.3)$$

where q is the predator mortality rate. This is counteracted by predator birth, the rate of which is assumed to depend on only

two things: the rate at which food is consumed, *aPN*, and the predator's efficiency, *f*, at turning this food into predator offspring. Predator birth rate is therefore *faPN*, and overall:

the Lotka–Volterra predator equation

$$dP/dt = faPN - qP. \quad (10.4)$$

Equations 10.2 and 10.4 constitute the Lotka–Volterra model.

The properties of this model can be investigated by finding zero isoclines. Zero isoclines were described for models of two-species competition in Section 8.4.1. Here, there are separate zero isoclines for the predators and prey, both of which are drawn on a graph of prey density (*x*-axis) against predator density (*y*-axis). Each is a line joining those combinations of predator and prey density that lead either to an unchanging prey population ($dN/dt = 0$; prey zero isocline) or an unchanging predator population ($dP/dt = 0$; predator zero isocline). Having drawn, say, a prey zero isocline, we know that combinations to one side of it lead to prey decrease, and combinations to the other to prey increase. Thus, as we shall see, if we plot the prey and predator zero isoclines on the same figure, we can begin to determine the pattern of the dynamics of the joint predator–prey populations.

In the case of the prey (Equation 10.2), when:

$$dN/dt = 0,\ rN = aPN \quad (10.5)$$

or:

$$P = r/a. \quad (10.6)$$

properties revealed by zero isoclines

Thus, since *r* and *a* are constants, the prey zero isocline is a line for which *P* itself is a constant (Figure 10.2a). Below it, predator abundance is low and the prey increase; above it, predator abundance is high and the prey decrease.

Likewise, for the predators (Equation 10.4), when:

$$dP/dt = 0,\ faPN = qP \quad (10.7)$$

or:

$$N = q/fa. \quad (10.8)$$

The predator zero isocline is therefore a line along which *N* is constant (Figure 10.2b). To the left, prey abundance is low and the predators decrease; to the right, prey abundance is high and the predators increase.

Putting the two isoclines together (Figure 10.2c) shows the behavior of joint populations. Predators increase in abundance when there are large numbers of prey, but this leads to an increased predation pressure on the prey, and thus to a decrease in prey abundance. This then leads to a food shortage for predators and a decrease in predator abundance, which leads to a relaxation of predation pressure and an increase in prey abundance, which leads to an increase in predator abundance, and so on (Figure 10.2d). Thus, predator and prey populations undergo 'coupled oscillations' in abundance, which continue indefinitely.

an underlying tendency towards coupled oscillations – which are structurally unstable in this case

The Lotka–Volterra model, then, is useful in pointing to this underlying tendency for predator–prey interactions to generate fluctuations in the prey population tracked by fluctuations in the predator population. The detailed behavior of the model, however, should not be taken seriously, because the cycles it exhibits are 'structurally unstable', showing 'neutral stability'. That is, the populations would follow precisely the same cycles indefinitely, but only until some external influence shifted them to new values, after which they would follow new cycles indefinitely (Figure 10.2e). In practice, of course, environments are continually changing, and populations would continually be 'shifted to new values'. A population following the Lotka–Volterra model would, therefore, not exhibit regular cycles, but, because of repeated disturbance, fluctuate erratically. No sooner would it start one cycle than it would be diverted to a new one.

For a population to exhibit regular and recognizable cycles, the cycles must themselves be stable: when an external influence changes the population level, there must be a tendency to return to the original cycle. In fact, as we shall see, predator–prey models (once we move beyond the very limiting assumptions of the Lotka–Volterra model) are capable of generating a whole range of abundance patterns: stable-point equilibria, multigeneration cycles, one-generation cycles, chaos, etc. – a range repeated in surveys of real populations. The challenge is to discover what light the models can throw on the behavior of real populations.

10.2.2 Delayed density dependence

numerical responses

The basic mechanism generating the coupled oscillations in these predator–prey interactions is a series of time-delayed '*numerical responses*', i.e. changes in one species' abundance in response to the abundance of the other species. The first is a time delay between 'many prey' and 'many predators' (arising because the response of predator abundance to high prey abundance cannot occur instantaneously). There may then be another time delay between 'many predators' and 'few prey', and then between 'few prey' and 'few predators', and so on. In practice, therefore, even where coupled oscillations exist, their exact shape is likely to reflect the varying delays, and strengths, of the different numerical responses. Certainly, the shapes of apparent coupled oscillations in real populations are varied, and not all are symmetric like those generated by the Lotka–Volterra model (see Figure 10.1).

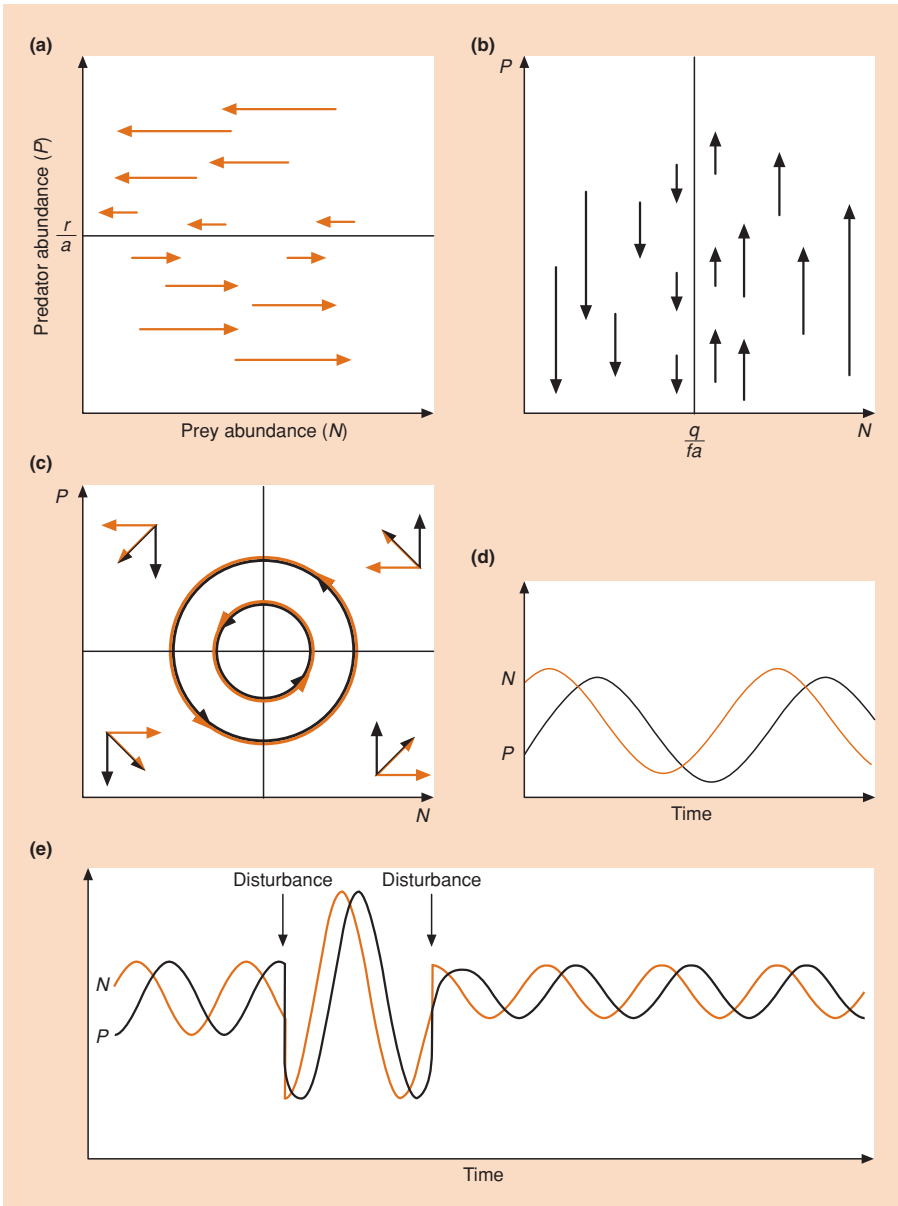

Figure 10.2 The Lotka–Volterra predator–prey model. (a) The prey zero isocline, with prey (*N*) increasing in abundance (arrows left to right) at lower predator densities (low *P*) and decreasing at higher predator densities. (b) The predator zero isocline, with predators increasing in abundance (arrows pointing upwards) at higher prey densities and decreasing at lower prey densities. (c) When the zero isoclines are combined, the arrows can also be combined, and these joint arrows progress in anticlockwise circles. In other words, the joint population moves with time from low predator/low prey (bottom left in (c)), to low predator/high prey (bottom right), to high predator/high prey, to high predator/low prey and back to low predator/low prey. Note, however, that the lowest prey abundance ('9 o'clock') comes one-quarter of a cycle before the lowest predator abundance ('6 o'clock' – anticlockwise movement). These coupled cycles of predator–prey abundance, continuing indefinitely, are shown as numbers against time in (d). However, as shown in (e), these cycles exhibit neutral stability: they continue indefinitely if undisturbed, but each disturbance to a new abundance initiates a new, different series of neutrally stable cycles, around the same means but with a different amplitude.

the regulatory tendencies of delayed density dependence are relatively difficult to demonstrate

These responses are density dependent (see Section 5.2): they act to reduce the size of relatively large populations and allow relatively small populations to increase. Varley (1947) introduced the term 'delayed density dependence' to describe them. The strength of a delayed density-dependent effect is related not to the current abundance (that would be *direct* density dependence) but to abundance at some time in the past (i.e. the delay-length ago). Compared to direct density dependence, delayed density dependence is relatively difficult to demonstrate. To see this, we can examine the coupled oscillations produced by a particular parasitoid–host model, shown in Figure 10.3a (Hassell, 1985). The details of the model need not concern us, but note that the oscillations are damped: they get gradually smaller over time until a stable equilibrium is reached. The prey population, subject to delayed density dependence, is regulated in size by the predator. In Section 5.6, we demonstrated density dependence by plotting *k* values against the log of density; but when we plot the *k* values of predator-induced mortality

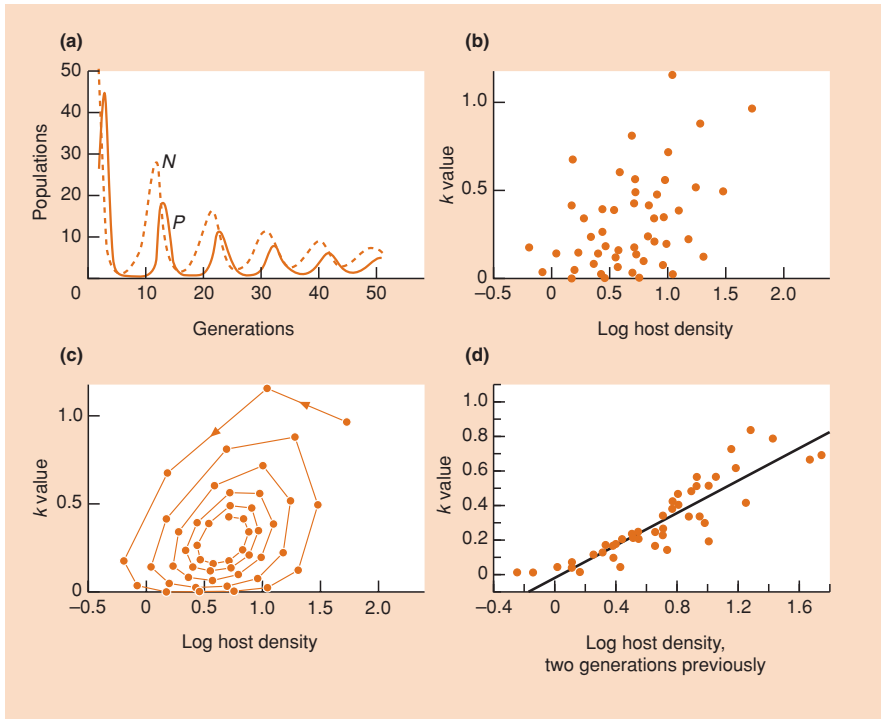

Figure 10.3 Delayed density dependence. (a) A parasitoid–host model followed over 50 generations: despite oscillations, the parasitoid has a regulatory effect on the host population. (b) For the same model, the *k* value of generation mortality plotted against the log of host density: no clear density-dependent relationship is apparent. (c) The points from (b) linked serially from generation to generation: they spiral in an anticlockwise direction – a characteristic of delayed density dependence. (After Hassell, 1985). (d) The *k* value of generation mortality plotted against the log of host density two generations previously: a clear delayed density-dependent relationship is again apparent.

against the log of prey density in that generation (Figure 10.3b), no clear relationship is apparent. On the other hand, when the same points are linked together, each generation to the next (Figure 10.3c), they can be seen to describe an anticlockwise spiral. This spiraling is characteristic of delayed density dependence. Here, because the oscillations are damped, the points spiral inwards to the equilibrium point. Moreover, when we plot the *k* values of predator-induced mortality against the log of prey density *two generations previously* (Figure 10.3d), the delayed density dependence is clearly revealed by the positive relationship characteristic of density dependence in general. Indeed, the fact that a two-generation delay gives a better fitting relationship than delays that are either shorter or longer, tells us that two generations is our best estimate of the delay in this case.

The regulatory effects of delayed density dependence are relatively easy to reveal for the model population of Figure 10.3, because it is not subject to the fluctuations of a natural environment, it is not subject to the density-dependent attacks of any other predator, it is not subject to the inaccuracies of sampling error, and so on. Data of this quality, however, are rarely if ever available for natural or even experimental populations. We return to the question of uncovering and integrating delayed density-dependent effects into an overall account of what determines abundance in Chapter 14. For now, though, this discussion highlights the relationship between 'regulation' and 'stability' in predator–prey interactions. Natural predator and prey populations tend to exhibit less violent and less regular fluctuations than those we have seen generated by the simplest models. Most of the rest of this chapter describes the search for explanations for these patterns and for the variations in dynamical pattern from case to case. A population that remains roughly constant in size provides evidence for the effects of both regulatory and stabilizing forces. The delayed density dependence of a predator–prey interaction 'regulates' in the sense of acting strongly on large populations and only weakly on small populations. But, as we have already seen, it can hardly be said, typically, to stabilize either population. What follows in this chapter is, therefore, in large part, a search for stabilizing forces that might complement the (delayed) regulatory forces that occur inherently in predator–prey interactions.

10.2.3 The Nicholson–Bailey model

Turning now to parasitoids, the basic model (Nicholson & Bailey, 1935) is again not so much realistic as a reasonable basis from which to start. Let H_t be the number of hosts, and P_t the number of parasitoids in generation t; r is the intrinsic rate of natural increase of the host. If H_a is the number of hosts attacked by

parasitoids (in generation t), then, assuming no intraspecific competition amongst the hosts (exponential growth – see Section 4.7.1), and that each host can support only one parasitoid (commonly the case):

$$H_{t+1} = e^{r}(H_t - H_a), \tag{10.9}$$

$$P_{t+1} = H_a. \tag{10.10}$$

In other words, hosts that are not attacked reproduce, and those that are attacked yield not hosts but parasitoids.

To derive a simple formulation for H_a, let E_t be the number of host–parasitoid encounters in generation t. Then, if A is the parasitoid's searching efficiency:

$$E_t = AH_tP_t \tag{10.11}$$

and:

$$E_t/H_t = AP_t. \tag{10.12}$$

Note the similarity to the formulation in Equation 10.2. Remember, though, that we are dealing with parasitoids, and hence a single host can be encountered several times, although only one encounter leads to successful parasitization (i.e. only one parasitoid develops). Predators, by contrast, would remove their prey and prevent re-encounters. Thus, Equation 10.2 dealt with instantaneous rates, rather than numbers.

a model based on random encounters . . .

If encounters are assumed to occur more or less at random, then the proportions of hosts that are encountered zero, one, two or more times are given by the successive terms in the appropriate 'Poisson distribution' (see any basic statistics textbook). The proportion not encountered at all, p_0, would be given by e^{-E_t/H_t}, and thus the proportion that *is* encountered (one or more times) is $1 - e^{-E_t/H_t}$. The number encountered (or attacked) is then:

$$H_a = H_t(1 - e^{-E_t/H_t}). \tag{10.13}$$

Using this and Equation 10.12 to substitute into Equations 10.9 and 10.10 gives us:

$$H_{t+1} = H_t e^{(r-AP_t)} \tag{10.14}$$

$$P_{t+1} = H_t(1 - e^{(-AP_t)}). \tag{10.15}$$

. . . giving rise to (unstable) coupled oscillations

This is the basic Nicholson–Bailey model of a host–parasitoid interaction. Its behavior is reminiscent of the Lotka–Volterra model but it is even less stable. An equilibrium combination of the two populations is a possibility, but even the slightest disturbance from this equilibrium leads to divergent coupled oscillations.

10.2.4 One-generation cycles

The coupled oscillations generated by the basic Lotka–Volterra and Nicholson–Bailey models are multigeneration cycles, i.e. there are several generations between successive peaks (or troughs), and such oscillations have lain at the heart of most attempts to understand cyclic predator–prey dynamics. However, other models of host–parasitoid (and host–pathogen) systems are able to generate coupled oscillations just *one* host generation in length (Knell, 1998; see, for example, Figure 10.1c). On the other hand, such 'generation cycles' can also occur in a population for reasons other than a predator–prey interaction – specifically as a result of competition between age classes within a population (Knell, 1998).

Predator–prey generation cycles occur essentially when the generation length of the consumer is roughly half that of its host – as it often is. Any small, chance peak in host abundance tends to generate a further peak in host abundance one host generation later. But any associated peak in consumer abundance occurs half a host generation length later, creating a trough in host abundance between the twin peaks. And this host trough creates a further host trough one generation later, but a consumer trough coinciding with the next host peak. Thus, the consumers have alternate 'feasts' and 'famines' that accentuate the originally small peaks and troughs in host abundance, and hence promote one-generation cycles (Figure 10.4).

10.2.5 Predator–prey cycles in nature: or are they?

The inherent tendency for predator–prey interactions to generate coupled oscillations in abundance might suggest an expectation of such oscillations in real populations. However, there are many important aspects of predator and prey ecology that have not been considered in the models derived so far; and as subsequent sections will show, these can greatly modify our expectations. Certainly, even if a population exhibits regular oscillations, this does not necessarily provide support for the Lotka–Volterra, Nicholson–Bailey or any other simple model. We saw cycles generated by intraspecific competition in Section 5.8, and we shall see several other routes to cycles in subsequent chapters (see also Kendall *et al.*, 1999). At this point, though, it is worth simply making the point that even when predators or prey exhibit regular cycles in abundance, it is never easy to demonstrate that these are *predator–prey* cycles.

hare and lynx: not the simple predator and prey they appear to be

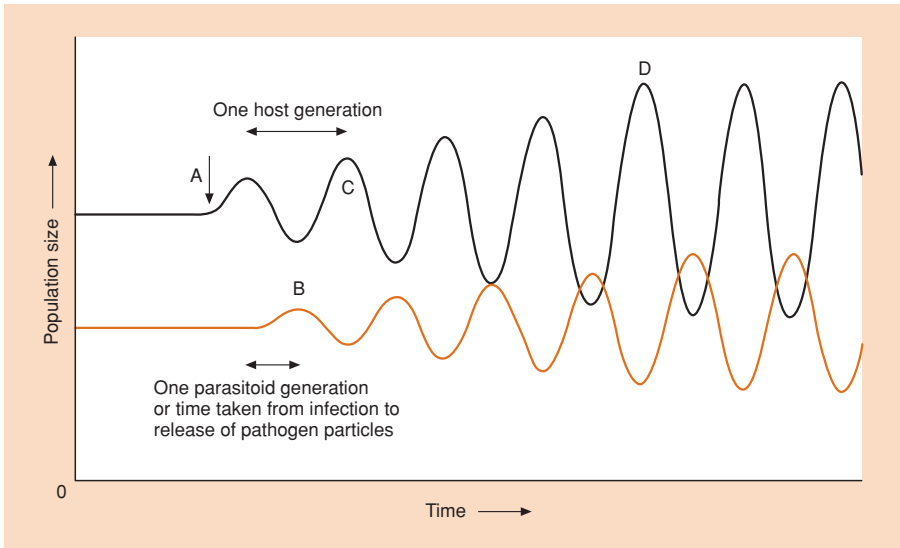

Figure 10.4 Schematic illustration of how a parasitoid or pathogen may generate coupled cycles in abundance in the host and itself that are approximately one host generation in length. For this, the parasitoid or pathogen must have a generation length approximately half that of the host. Any chance increase in host abundance (A) will first give rise to an increase in parasitoid abundance one parasitoid generation later (B), and also to an increase in host abundance one host generation later (C). But the parasitoid peak at B will also give rise to a coincident host trough, which will give rise to a parasitoid trough at C, reinforcing the host peak at that point. This mutual reinforcement will continue until by, say, D, persistent host generation-length cycles have become established. (After Knell, 1998; from Godfray & Hassell, 1989.)

The regular oscillations in the abundance of the snowshoe hare and the Canadian lynx shown in Figure 10.1a have often been held to epitomize predator–prey cycles. Recently, however, evidence has increasingly indicated that even this apparent exemplar is not as straightforward as it has seemed. Experimental manipulations carried out in the field are one powerful means of suggesting what forces are normally acting: if those forces are removed or exaggerated, is the cycle eliminated or enhanced? A whole series of coordinated field experiments has indicated that the cyclic hare is not simply a prey of the lynx (and other predators in the community), nor simply a predator of its plant food resources: the cycle can be understood only by taking account of its interactions both as a prey *and* as a predator (Krebs *et al.*, 2001). Furthermore, modern statistical analysis of the time series of abundances has tended to confirm this: the hare series carries a relatively complex 'signature', suggesting the influence of both its predators and its food, whereas the lynx series has a simpler signature, suggesting only the influence of its (hare) prey (Stenseth *et al.*, 1997; see also Section 14.5.2). What has so often been described as a predator-prey cycle seems rather to comprise one predator linked to a species that is both predator and prey.

moths and two natural enemies

Apparently coupled one-generation cycles linking a moth host (*Plodia interpunctella*) and its parasitoid *Venturia canescens* were shown in Figure 10.1c. In this case, the dangers of jumping too readily to the conclusion that these are predator–prey cycles are highlighted by the fact that the host also exhibits generation-length cycles when maintained alone, without any natural enemies, and also when maintained with another enemy, a granulovirus (Figure 10.5). It has been possible, however, to confirm that the cycles in Figure 10.1c are indeed coupled oscillations, using methods similar to those applied to the hare–lynx time series (Bjørnstad *et al.*, 2001). The host-alone cycles have within them the signature simply of intraspecific competition, and the virus seems to modulate this pattern but does not alter its basic structure (i.e. the patterns in Figure 10.5 are *not* predator–prey cycles). However, the host and parasitoid cycles in Figure 10.1c both carry the same, more complex signature that indicates a tightly coupled prey–predator interaction (see also Section 12.7.1).

We return to the question of cycles – indeed, some of the same cycles discussed above – in Section 14.6, as part of a more general exploration of how the whole range of biotic and abiotic factors come together to determine the level and pattern of a population's abundance.

10.3 Effects of crowding

The most obvious omission, perhaps, from the predator–prey interactions we have modeled so far has been any acknowledgement that prey abundance may be limited by other prey, and predator

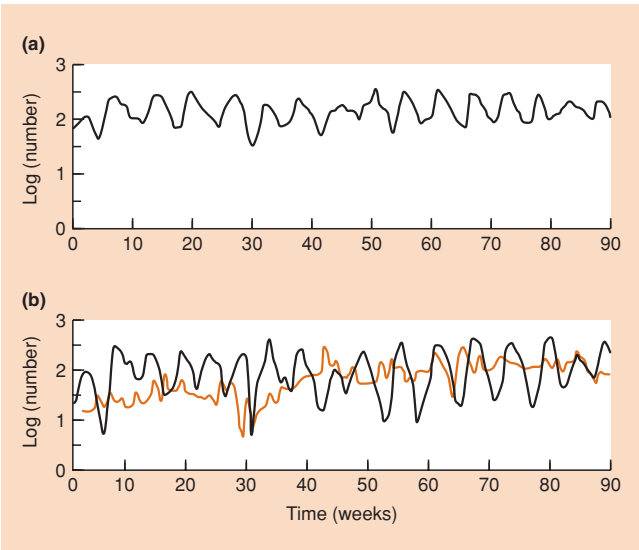

Figure 10.5 Host generation-length cycles in the moth *Plodia interpunctella* (a) alone (black line) and (b) with a granulovirus (colored line). These dynamics may be compared with those in Figure 10.1c. In spite of a superficial similarity in pattern, analysis indicates that those in (a) are generated by intraspecific competition; those in (b) are simply modulated versions of those in (a) and are therefore not predator–prey cycles. However, those in Figure 10.1c are predator–prey cycles. (After Bjornstad *et al.*, 2001.)

abundance by other predators. Prey are bound to be increasingly affected by intraspecific competition as their abundance increases; and predators, too, are likely to be limited at high densities by the availability of resting places, say, or safe refuges of their own, quite apart from their interaction with their most obvious resource, their prey.

mutual interference

More generally, predators have been assumed in the models discussed thus far to consume prey at a rate that depends only on prey abundance (in Equation 10.2, for example, the consumption rate per predator is simply aN). In reality, consumption rate will also often depend on the abundance of the predators themselves. Most obviously, food shortage – the abundance of prey *per predator* – will commonly result in a reduction in the consumption rate per individual as predator density increases. However, even when food is not limited, the consumption rate can be reduced by a number of processes known collectively as mutual interference (Hassell, 1978). For example, many consumers interact behaviorally with other members of their population, leaving less time for feeding and therefore depressing the overall feeding rate. For instance, humming-birds actively and aggressively defend rich sources of nectar. Alternatively, an increase in consumer density may lead to an increased rate of emigration, or of consumers stealing food from one another (as do many gulls), or the prey themselves may respond to the presence of consumers and become less available for capture. All of these mechanisms give rise to a decline in predator consumption rate with predator density. Figure 10.6a, for example, shows significant reductions in consumption rate with abundance even at low densities of the crab *Carcinus aestuarii* foraging for the mussel *Musculista senhousia*; while Figure 10.6b shows that the kill rate of wolves, *Canis lupus*, preying on moose, *Alces alces*, in Isle Royale National Park, Michigan, USA, was lowest when there were most wolves.

10.3.1 Crowding in the Lotka–Volterra model

The effects of intraspecific competition, and of a decline in predator consumption rate with predator density, can be investigated by modifying the Lotka–Volterra isoclines. The details of incorporating intraspecific competition into the prey zero isocline are described by Begon *et al.* (1990), but the end result (Figure 10.7a) can be understood without reference to these details. At low prey densities there is no intraspecific competition, and the prey isocline is horizontal as in the Lotka–Volterra model. But as density increases, it is increasingly the case that prey densities below the isocline (prey increase) must be placed above the isocline (prey decrease) because of the effects of intraspecific competition. Hence, the isocline is increasingly lowered until it reaches the prey axis at the carrying capacity, K_N; that is, the prey can only just maintain themselves even in the absence of predators.

crowding and the Lotka–Volterra isoclines

As we have seen, the predator isocline in the Lotka–Volterra model is vertical. This itself reflects the assumption that the ability of a predator population to increase in abundance is determined by the absolute abundance of prey, irrespective of the number of predators. If, however, mutual interference amongst the predators increases, then individual consumption rates will decline with predator abundance, and additional prey will be required to maintain a predator population of any given size. The predator zero isocline will depart increasingly from the vertical (Figure 10.7b). Moreover, at high densities, competition for other resources will put an upper limit on the predator population (a horizontal isocline) irrespective of prey numbers (Figure 10.7b).

ratio-dependent predation

An alternative modification is to abandon altogether the assumption that consumption rate depends only on the absolute availability of prey, and assume ratio-dependent predation instead (Arditi & Ginzburg, 1989), although this alternative has itself been criticized (see Abrams, 1997; Vucetich *et al.*, 2002). In this case, the consumption rate depends on the ratio of prey to predators, and a particular ratio needs to be exceeded for the predators to increase in abundance: a

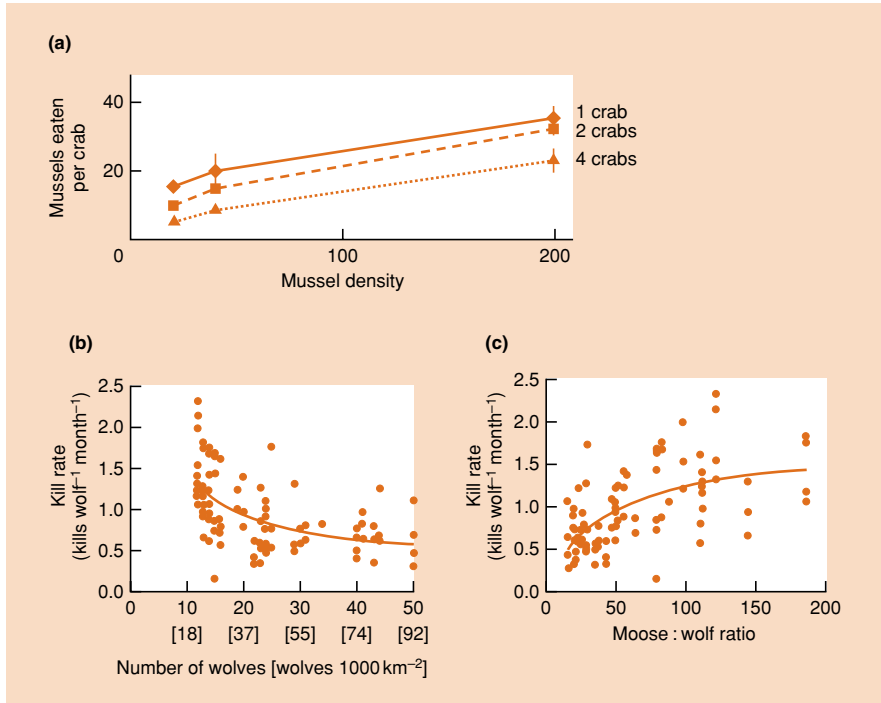

Figure 10.6 (a) Mutual interference amongst crabs, *Carcinus aestuarii*, feeding on mussels, *Musculista senhousia*. ◆ 1 crab; ■, 2 crabs; ▲, 4 crabs. The more crabs there were, the lower their per capita consumption rate. (After Mistri, 2003.) (b) Mutual interference amongst wolves, *Canis lupus*, preying on moose, *Alces alces*. (c) The same data but with wolf kill rate plotted against the moose : wolf ratio. The fitted curve assumes a dependence of kill rate on this ratio, but also that the wolves may become 'saturated' at high moose densities (see Section 10.4.2). This curve fits better than any for which kill rate depends on either predator density (e.g. (b)) or prey density. ((b, c) after Vucetich *et al.*, 2002.)

diagonal zero isocline passing through the origin (Figure 10.7c). Evidence of ratio-dependent predation is illustrated, for example, for the wolf–moose study in Figure 10.6c.

The likely effects of crowding in either population can now be deduced by combining the predator and prey isoclines (Figure 10.7d). Oscillations are still apparent for the most part, but these are no longer neutrally stable. Instead, they are damped so that they converge to a stable equilibrium. Predator–prey interactions in which either or both populations are substantially self-limited are likely, therefore, to exhibit patterns of abundance that are relatively stable, i.e. in which fluctuations in abundance are relatively slight.

crowding stabilizes dynamics

More particularly, when the predator is relatively inefficient, i.e. when many prey are needed to maintain a population of predators (curve (ii) in Figure 10.7d), the oscillations are damped quickly but the equilibrium prey abundance (N^*) is not much less than the equilibrium in the absence of predators (K_N). By contrast, when the predators are more efficient (curve (i)), N^* is lower and the equilibrium density of predators, P^*, is higher – but the interaction is less stable (the oscillations are more persistent). Moreover, if the predators are very strongly self-limited, then abundance may not oscillate at all (curve (iii)); but P^* will tend to be low, whilst N^* will tend to be not much less than K_N. Hence, for interactions where there is crowding, there appears to be a contrast between those in which predator density is low, prey abundance is little affected and the patterns of abundance are stable, and those in which predator density is higher and prey abundance is more drastically reduced, but the patterns of abundance are less stable. (Figure 10.7d does not use ratio-dependent predation, but a predator isocline with a steeper slope in a ratio-dependent model (more efficient predation) can be equated, for present purposes, with an isocline rising from closer to the origin in the figure – that is, curve (i) rather than curve (ii).)

Essentially similar conclusions emerge from modifications of the Nicholson–Bailey model that incorporate either simple (logistic) crowding effects amongst the hosts or mutual interference amongst the predators (Hassell, 1978).

To quote examples of data proving the stabilizing influence of self-limitation on predator–prey dynamics would be difficult, simply because it would be all but impossible to compare the dynamics of matched populations with and without such self-limitation. On the other hand, populations of predators and prey with relatively stable dynamics are commonplace, as are the stabilizing forces of self-limitation we have discussed here. To take a more specific example, there are two groups of primarily herbivorous rodents that are widespread in the Arctic: the microtine rodents (lemmings and voles) and the ground squirrels. The microtines are renowned for their dramatic, cyclic fluctuations in abundance (see Chapter 14), but the ground squirrels have populations that remain remarkably constant from year to year, especially in open meadow and tundra habitats. There, significantly, they appear to be strongly self-limited by food availability, suitable burrowing habitat and their own spacing behavior (Karels & Boonstra, 2000).

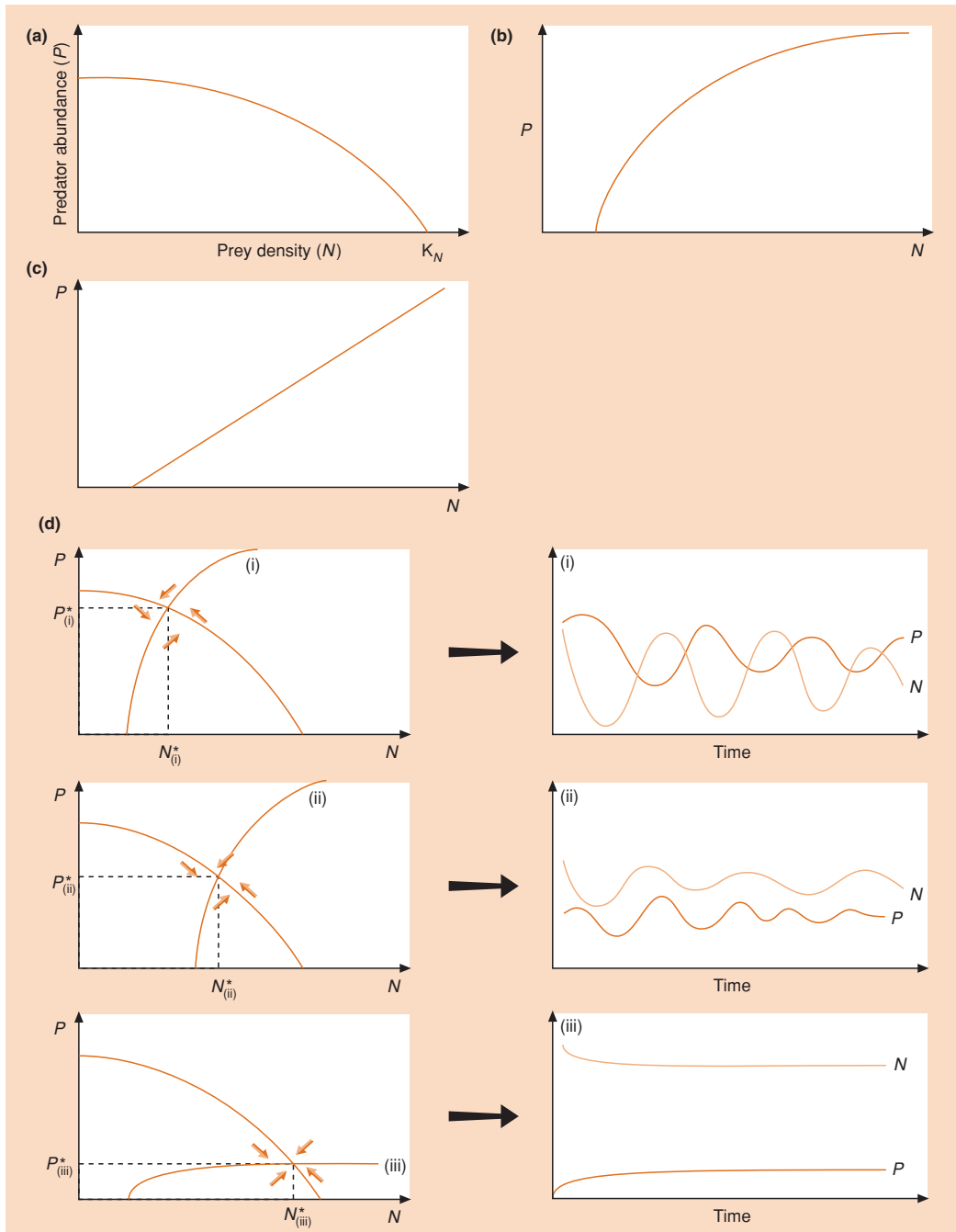

Figure 10.7 (a) A prey zero isocline subject to crowding. At the lowest prey densities this is the same as the Lotka–Volterra isocline, but when the density reaches the carrying capacity (K_N) the population can only just maintain itself even in the complete absence of predators. (b) A predator zero isocline subject to crowding (see text). (c) A predator zero isocline when there is prey : predator ratio dependent predation. (d) The prey zero isocline combined with the predator zero isoclines with increasing levels of crowding: (i), (ii) and (iii). P^* is the equilibrium abundance of predators, and N^* the equilibrium abundance of prey. Combination (i) is the least stable (most persistent oscillations) and has the most predators and least prey: the predators are relatively efficient. Less efficient predators, as in (ii), give rise to a lowered predator abundance, an increased prey abundance and less persistent oscillations. Strong predator self-limitation (iii) can eliminate oscillations altogether, but P^* is low and N^* is close to K_N.

how important is mutual interference in practice?

On a cautionary note, however, Umbanhowar *et al.* (2003), for example, failed to find evidence of mutual interference in a field study of the parasitoid *Tachinomyia similis* attacking its moth host *Orgyia vetusta*. The strength of mutual interference may often have been exaggerated by forcing predators to forage in artificial arenas at densities much higher than those they experience naturally. This is a useful reminder of the general point that an ecological force that is powerful in models or in the laboratory may none the less often be trivial, in practice, in natural populations. There can be little doubt, though, that self-limitation in its various forms frequently plays a key role in shaping predator–prey dynamics.

10.4 Functional responses

Having examined the relationship between a predator's consumption rate and the predator's own abundance, above, we turn now to the effect on this consumption rate of the prey's abundance, the so-called functional response (Solomon, 1949). Below we describe the three main types of functional response (Holling, 1959), before considering how they might modify predator–prey dynamics.

10.4.1 The type 1 functional response

The most basic, 'type 1' functional response is that assumed by the Lotka–Volterra equations: consumption rate rises linearly with prey density (indicated by the constant, a, in Equation 10.2). An example is illustrated in Figure 10.8. The rate at which *Daphnia magna* consumed yeast cells rose linearly when the density of cells

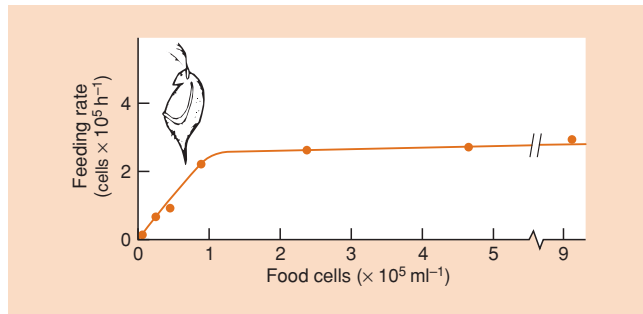

Figure 10.8 The type 1 functional response of *Daphnia magna* to different concentrations of the yeast *Saccharomyces cerevisiae*. (After Rigler, 1961.)

varied, because the yeast cells were extracted by the *Daphnia* from a constant volume of water washed over their filtering apparatus, and the amount extracted therefore rose in line with food concentration. Above 10^5 cells ml^{-1}, however, the *Daphnia* could filter more cells but were unable to swallow all the food they filtered. They therefore ingested food at a maximum (plateau) rate irrespective of its concentration.

10.4.2 The type 2 functional response

The most frequently observed functional response is the 'type 2' response, in which consumption rate rises with prey density, but gradually decelerates until a plateau is reached at which consumption rate remains constant irrespective of prey density. (Realistically, even a type 1 response must have a plateau, as in the example above. The distinction is between the deceleration of a type 2 response and the linearity of the type 1 response.) Type 2 responses are shown for a carnivore, a herbivore and a parasitoid in Figure 10.9.

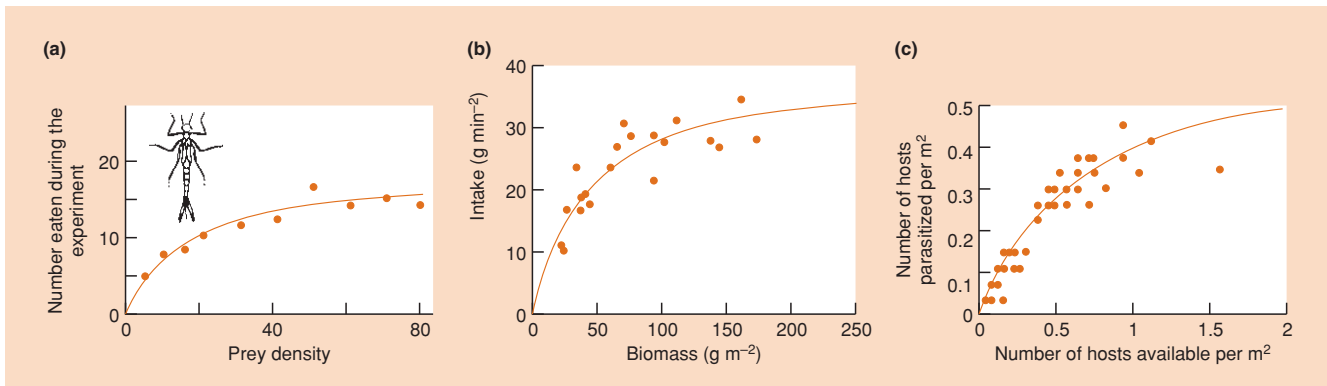

Figure 10.9 Type 2 functional responses. (a) Tenth-instar damselfly nymphs (*Ishnura elegans*) eating *Daphnia* of approximately constant size. (After Thompson, 1975.) (b) Wood bison (*Bison bison*) feeding on the sedge *Carex atherodes* presented at a range of sedge biomass densities. (After Bergman *et al.*, 2000.) (c) The parasitoid *Microplitis croceipes* attacking the tobacco budworm *Heliothis virescens*. (After Tillman, 1996.)

the type 2 response and handling time

The type 2 response can be explained by noting that a predator has to devote a certain handling time to each prey item it consumes (i.e. pursuing, subduing and consuming the prey item, and then preparing itself for further search). As prey density increases, finding prey becomes increasingly easy. Handling a prey item, however, still takes the same length of time, and handling overall therefore takes up an increasing proportion of the predator's time – until at high prey densities the predator is effectively spending all of its time handling prey. The consumption rate therefore approaches and then reaches a maximum (the plateau), determined by the maximum number of handling times that can be fitted into the total time available.

We can derive a relationship between P_e (the number of prey items eaten by a predator during a period of searching time, T_s) and N, the density of those prey items (Holling, 1959). P_e increases with the time available for searching, it increases with prey density, and it increases with the searching efficiency or attack rate of the predator, a. Thus:

Holling's type 2 response equation

$$P_e = aT_sN. \tag{10.16}$$

However, the time available for searching will be less than the total time, T, because of time spent handling prey. Hence, if T_h is the handling time of each prey item, then T_hP_e is the total time spent handling prey, and:

$$T_s = T - T_hP_e. \tag{10.17}$$

Substituting this into Equation 10.16 we have:

$$P_e = a(T - T_hP_e)N \tag{10.18}$$

or, rearranging:

$$P_e = aNT/1 + aT_hN. \tag{10.19}$$

Note that the equation describes the amount eaten during a specified period of time, T, and that the density of prey, N, is assumed to remain constant throughout that period. In experiments, this can sometimes be guaranteed by replacing any prey that are eaten, but more sophisticated models are required if prey density is depleted by the predator. Such models are described by Hassell (1978), who also discusses methods of estimating attack rates and handling times from a set of data. (Trexler *et al.*, 1988, discuss the general problem of fitting functional response curves to sets of data.)

other routes to a type 2 response

It would be wrong, however, to imagine that the existence of a handling time is the only or the complete explanation for all type 2 functional responses. For instance, if the prey are of variable profitability, then at high densities the diet may tend towards a decelerating number of highly profitable items (Krebs *et al.*, 1983); or a predator may become confused and less efficient at high prey densities.

10.4.3 The type 3 functional response

Type 3 functional responses are illustrated in Figure 10.10a–c. At high prey densities they are similar to a type 2 response, and the explanations for the two are the same. At low prey densities, however, the type 3 response has an accelerating phase where an increase in density leads to a more than linear increase in consumption rate. Overall, therefore, a type 3 response is 'S-shaped' or 'sigmoidal'.

switching

One important way in which a type 3 response can be generated is through switching by the predator (see Section 9.5.2). The similarities between Figures 9.15 and 10.10 are readily apparent. The difference is that discussions of switching focus on the density of a prey type relative to the densities of alternatives, whereas functional responses are based on only the absolute density of a single prey type. In practice, though, absolute and relative densities are likely to be closely correlated, and switching is therefore likely to lead frequently to a type 3 functional response.

variations in searching efficiency or handling time

More generally, a type 3 functional response will arise whenever an increase in food density leads to an increase in the consumer's searching efficiency, a, or a decrease in its handling time, T_h, since between them these two determine consumption rate (Equation 10.19). Thus, the small mammals in Figure 10.10a appear to develop a search image for sawfly cocoons as they become more abundant (increasing efficiency). The bluebottle fly, *Calliphora vomitoria* (Figure 10.10b), spends an increasing proportion of its time searching for 'prey' as prey density increases (Figure 10.10d), also increasing efficiency. Whilst the wasp *Aphelinus thomsoni* (Figure 10.10c) exhibits a reduction in mean handling time as the density of its sycamore aphid prey increases (Figure 10.10e). In each case, a type 3 functional response is the result.

10.4.4 Consequences for population dynamics of functional responses and the Allee effect

type 3 responses stabilize but may be unimportant in practice

Different types of functional response have different effects on population dynamics. A type 3 response means a low predation rate at low prey densities. In terms of isoclines, this means that

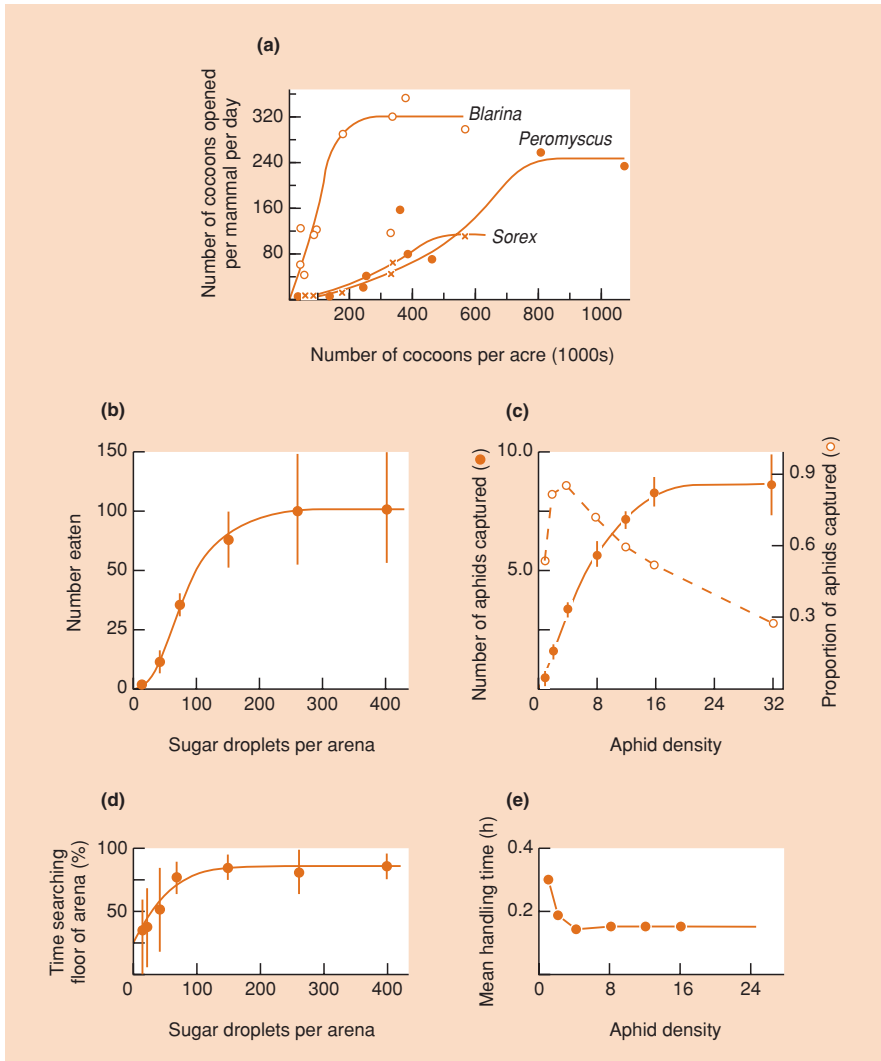

Figure 10.10 Type 3 (sigmoidal) functional responses. (a) The shrews *Sorex* and *Blarina* and the deer mouse *Peromyscus* responding to changing field densities of cocoons of the European pine sawfly, *Neodiprion sertifer*, in Ontario, Canada. (After Holling, 1959.) (b) The bluebottle fly, *Calliphora vomitoria*, feeding on sugar droplets. (After Murdie & Hassell, 1973.) (c) The wasp, *Aphelinus thomsoni*, attacking sycamore aphids, *Drepanosiphum platanoidis*: note the density-dependent increase in prey mortality rate at low prey densities (– – –) giving rise to the accelerating phase of the response curve (——). (After Collins *et al.*, 1981.) (d) The basis of the response in (b): searching efficiency of *C. vomitoria* increases with 'prey' (sugar droplet) density. (After Murdie & Hassell, 1973.) (e) The basis of the response in (c): handling time in *A. thomsoni* decreases with aphid density. (After Collins *et al.*, 1981.)

prey at low densities can increase in abundance virtually irrespective of predator density, and that the prey zero isocline will therefore rise vertically at low prey densities (Figure 10.11a). This could lend considerable stability to an interaction (Figure 10.11a, curve (i)), but for this the predator would have to be highly efficient at low prey densities (readily capable of maintaining itself), which contradicts the whole idea of a type 3 response (ignoring prey at low densities). Hence, curve (ii) in Figure 10.11a is likely to apply, and the stabilizing influence of the type 3 response may in practice be of little importance.

On the other hand, if a predator has a type 3 response to one particular type of prey because it switches its attacks amongst various prey types, then the population dynamics of the predator would be independent of the abundance of any particular prey type, and the vertical position of its zero isocline would therefore be the same at all prey densities. As Figure 10.11b shows, this can lead potentially to the predators regulating the prey at a low and stable level of abundance.

switching, stabilization and the voles of Fennoscandia

An apparent example of this is provided by studies of vole cycles in Europe (Hanski *et al.*, 1991; see also Section 14.6.4). In subarctic Finnish Lapland, there are regular 4- or 5-year cycles, with a ratio of maximum : minimum vole densities generally exceeding 100. In southern Sweden small rodents show no regular multiannual cycles. But between the two, moving north to south in Fennoscandia, there is a gradient of decreasing regularity, amplitude and length of the cycle. Hanski *et al.* argue that this gradient is itself correlated with a gradient of increasing densities of generalist predators that switch between alternative

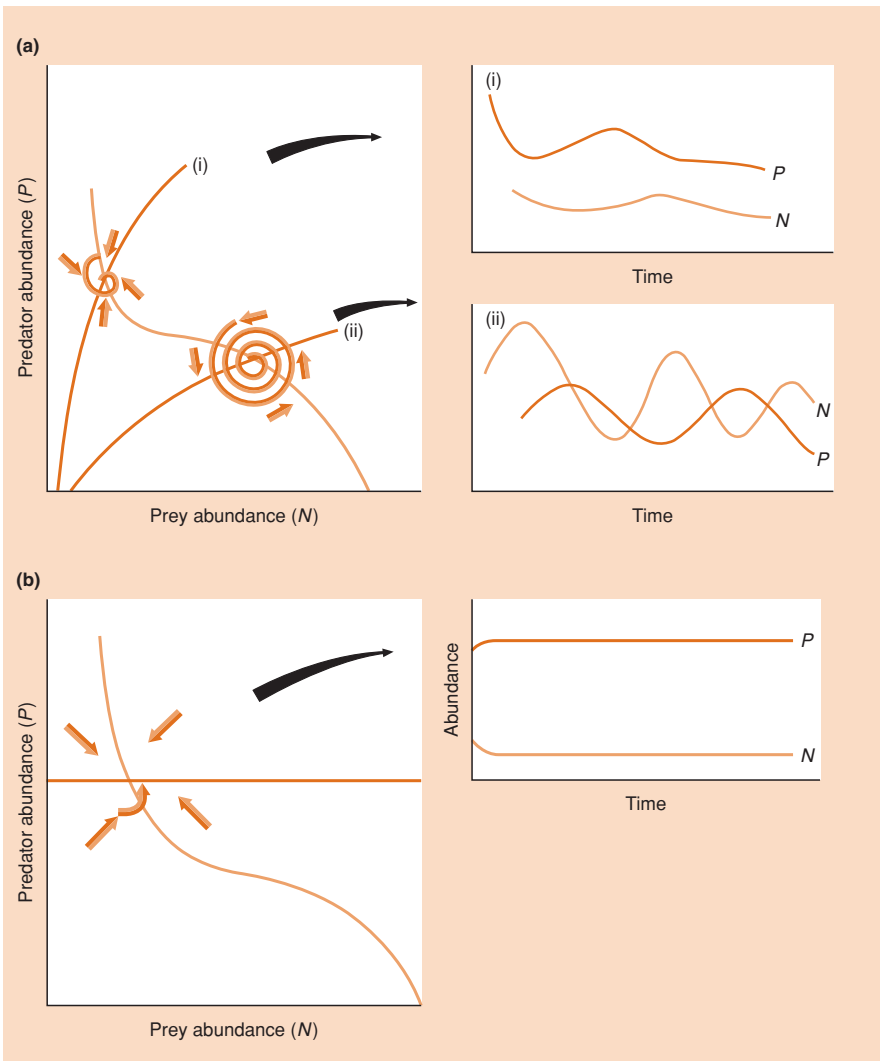

Figure 10.11 (a) The prey zero isocline is that which is appropriate when consumption rate is particularly low at low prey densities because of a type 3 functional response, an aggregative response (and partial refuge), an actual refuge or because of a reserve of plant material that is not palatable. With a relatively inefficient predator, predator zero isocline (ii) is appropriate and the outcome is not dissimilar from Figure 10.7. However, a relatively efficient predator will still be able to maintain itself at low prey densities. Predator zero isocline (i) will therefore be appropriate, leading to a stable pattern of abundance in which prey density is well below the carrying capacity and predator density is relatively high. (b) When a type 3 functional response arises because the predator exhibits switching behavior, the predator's abundance may be independent of the density of any particular prey type (main figure), and the predator zero isocline may therefore be horizontal (unchanging with prey density). This can lead to a stable pattern of abundance (inset) with prey density well below the carrying capacity.

prey as relative densities change (especially red foxes, badgers, domestic cats, buzzards, tawny owls and crows) and of specialist bird predators (especially other owl species and kestrels) that, being wide ranging in their activity, switch between alternative areas. In both cases, predator dynamics would be effectively independent of vole abundance, adding stability to the system in the manner of Figure 10.11b. In fact, Hanski *et al.* were able to go further in constructing a simple model of prey (voles) interacting with specialist predators (mustelids: stoats and weasels) and generalist (switching) predators. Their general contention was supported; as the number of generalist predators increased, oscillations in vole and mustelid abundance (which may or may not be the basis for the vole cycle) decreased in length and amplitude. Large enough densities of switching generalists stabilized the cycle entirely.

type 2 responses and the Allee effect destabilize – but not necessarily in practice

Turning to type 2 responses, if the predator has a response that reaches its plateau at relatively low prey densities (well below K_N), then the prey zero isocline has a hump, because there is a range of intermediate prey densities where the predators become less efficient with increasing prey density but the effects of competition amongst the prey are not intense. A hump will also arise here if the prey are subject to an 'Allee effect', where they have a disproportionately low rate of recruitment when their own density is low, perhaps because mates are difficult to find or because a 'critical number' must be exceeded before a resource can be properly exploited, i.e. there is inverse density dependence at low population densities (Courchamp *et al.*, 1999). If the predator

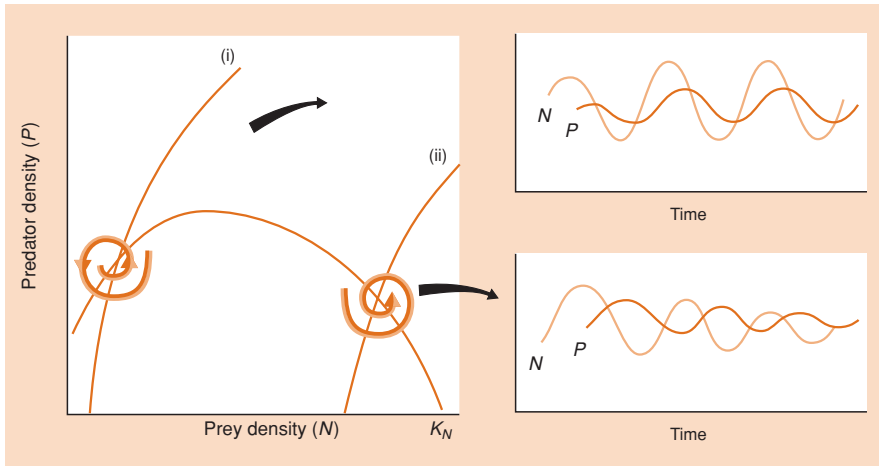

Figure 10.12 The possible effects of a prey isocline with a 'hump', either as a result of a type 2 functional response or an Allee effect. (i) If the predator is highly efficient, with its isocline crossing to the left of the hump, then the hump can be destabilizing, leading to the persistent oscillations of a limit cycle (inset). (ii) But if the predator is less efficient, crossing to the right of the hump, then the hump has little effect on the dynamics: the oscillations converge (inset).

isocline crosses to the right of the hump, then the population dynamics of the interaction will be little affected; but if the isocline crosses to the left of the hump, then the outcome will be persistent rather than convergent oscillations, i.e. the interaction will be *de*stabilized (Figure 10.12).

However, for a type 2 response to have this effect, predators would have to suffer serious reductions in their consumption rate at prey densities far below those at which the prey themselves suffer seriously from competition. This is unlikely. The potentially destabilizing effects of type 2 responses may also therefore be of little practical importance.

A destabilizing Allee effect has not apparently been established for any 'natural' predator–prey interaction. On the other hand, when we ourselves are the predator (for example, with exploited fisheries populations), we frequently have the ability (i.e. the technology) to maintain effective predation at low prey densities. If the prey population also exhibits an Allee effect, then the combination of this and persistent predation may all too readily drive a population towards extinction (Stephens & Sutherland, 1999; and see Section 15.3.5). That is, our isocline may cross that of the prey well to the left of their hump.

10.5 Heterogeneity, aggregation and spatial variation

Until now in this chapter, environmental heterogeneities, and the variable responses of predators and prey to such heterogeneities – all of which we saw in the previous chapter to be commonplace – have been ignored. We can ignore them no longer.

10.5.1 Aggregative responses to prey density

Because of the potential consequences for population dynamics, ecologists have been particularly interested in patch preferences where patches vary in the density of the food or prey items they contain (see Section 9.6). At one time it appeared, and was widely believed, that: (i) predators generally spent most time in patches containing high densities of prey (because these were the most profitable patches); (ii) most predators were therefore to be found in such patches; and (iii) prey in those patches were therefore most vulnerable to predation, whereas those in low-density patches were relatively protected and most likely to survive. Examples certainly exist to support the first two of these propositions (see Figure 9.20a–d), demonstrating an 'aggregative response' by the predators that is directly density dependent (predators spending most time in patches with high densities of prey such that prey and predator densities are positively correlated). However, this is not always the case. Furthermore, contrary to the third proposition, reviews of host–parasitoid interactions (e.g. Pacala & Hassell, 1991) have shown that prey (hosts) in high-density patches are not necessarily the most vulnerable to attack (direct density dependence): percentage parasitism may also be inversely density dependent or density independent between patches (see Figure 9.20e). Indeed, the reviews suggest that only around 50% of the studies examined show evidence of density dependence, and in only around 50% of these is the density dependence direct, as opposed to inverse. None the less, despite this variation in pattern, it remains true that the risk of predation often varies greatly between patches, and hence between individual prey.

do predators aggregate in high-density prey patches?

Many herbivores also display a marked tendency to aggregate, and many plants show marked variation in their risk of being attacked. The cabbage aphid (*Brevicoryne brassicae*) forms aggregates at two separate levels (Way & Cammell, 1970). Nymphs quickly form large groups when isolated on the surface of a single leaf, and populations on a single plant tend to be restricted to particular leaves. When aphids attack only one leaf

plants may be protected by the aggregative responses of herbivores

of a four-leaved cabbage plant (as they do naturally), the other three leaves survive; but if the same number of aphids are evenly spread over the four leaves, then all four leaves are destroyed (Way & Cammell, 1970). The aggregative behavior of the herbivores affords protection to the plant overall. But how might such heterogeneities influence the dynamics of predator–prey interactions?

10.5.2 Heterogeneity in the graphical model

refuges, partial refuges and vertical isoclines

We can start by incorporating into the Lotka–Volterra isoclines some relatively simple types of heterogeneity. Suppose that a portion of the prey population exists in a refuge: for example, shore snails packed into cracks in the cliff-face, away from marauding birds, or plants that maintain a reserve of material underground that cannot be grazed. In such cases, the prey zero isocline rises vertically at low prey densities (again, see Figure 10.11), since prey at low densities, hidden in their refuge, can increase in abundance irrespective of predator density.

Even if predators tend simply to ignore prey in low-density patches, as we have seen in some aggregative responses (see Section 9.6), this comes close to those prey being in a refuge, in the sense that the predators do not (rather than cannot) attack them. The prey may therefore be said to have a 'partial refuge', and this time the prey isocline can be expected to rise almost vertically at low prey abundances.

We saw above, when discussing type 3 functional responses, that such isoclines have a tendency to stabilize interactions. Early analyses of both the Lotka–Volterra and the Nicholson–Bailey systems (and early editions of this textbook) agreed with this conclusion: that spatial heterogeneities, and the responses of predators and prey to them, stabilize predator–prey dynamics, often at low prey densities (Beddington *et al.*, 1978). However, as we shall see next, subsequent developments have shown that the effects of heterogeneity are more complex than was previously supposed: the effects of heterogeneity vary depending on the type of predator, the type of heterogeneity, and so on.

10.5.3 Heterogeneity in the Nicholson–Bailey model

negative binomial encounters . . .

Most progress has been made in untangling these effects in host–parasitoid systems. A good starting point is the model constructed by May (1978), in which he ignored precise details and argued simply that the distribution of host–parasitoid encounters was not random but aggregated. In particular, he assumed that this distribution could be described by a particular statistical model, the negative binomial. In this case (in contrast to Section 10.2.3), the proportion of hosts not encountered at all is given by:

$$p_0 = \left[1 + \frac{AP_t}{k}\right]^{-k} \tag{10.20}$$

where k is a measure of the degree of aggregation; maximal aggregation at $k = 0$, but a random distribution (recovery of the Nicholson–Bailey model) at $k = \infty$. If this is incorporated into the Nicholson–Bailey model (Equations 10.14 and 10.15), then we have:

$$H_{t+1} = H_t e^r \left[1 + \frac{AP_t}{k}\right]^{-k} \tag{10.21}$$

$$P_{t+1} = H_t \left\{1 - \left[1 + \frac{AP_t}{k}\right]^{-k}\right\}. \tag{10.22}$$

. . . that stabilize dynamics

The behavior of a version of this model, which also includes a density-dependent host rate of increase, is illustrated in Figure 10.13, from which it is clear that the system is given a marked boost in stability by the incorporation of significant levels of aggregation ($k \leq 1$). Of particular importance is the existence of stable systems with low values of H^*/K; i.e. aggregation appears capable of generating stable host abundances well below the host's normal carrying capacity. This coincides with the conclusion drawn from Figure 10.11.

10.5.4 Aggregation of risk and spatial density dependence

pseudo-interference

How does this stability arise out of aggregation? The answer lies in what has been called 'pseudo-interference' (Free *et al.*, 1977). With mutual interference, as predator density increases, predators spend an increasing amount of time interacting with one another, and their attack rate therefore declines. With pseudo-interference, attack rate also declines with parasitoid density, but as a result of an increasing fraction of encounters being wasted on hosts that have already been attacked. The crucial point is that 'aggregation of risk' amongst hosts tends to increase the amount of pseudo-interference. At low parasitoid densities, a parasitoid is unlikely to have its attack rate reduced as a result of aggregation. But at higher parasitoid densities, parasitoids in aggregations (where most of them are) will increasingly be faced with host patches in which most or all of the hosts have already been parasitized. As parasitoid density increases, therefore, their effective attack rate (and hence their subsequent birth rate) declines rapidly – a *directly* density-dependent effect. This dampens both the natural oscillations in parasitoid density, and their impact on host mortality.

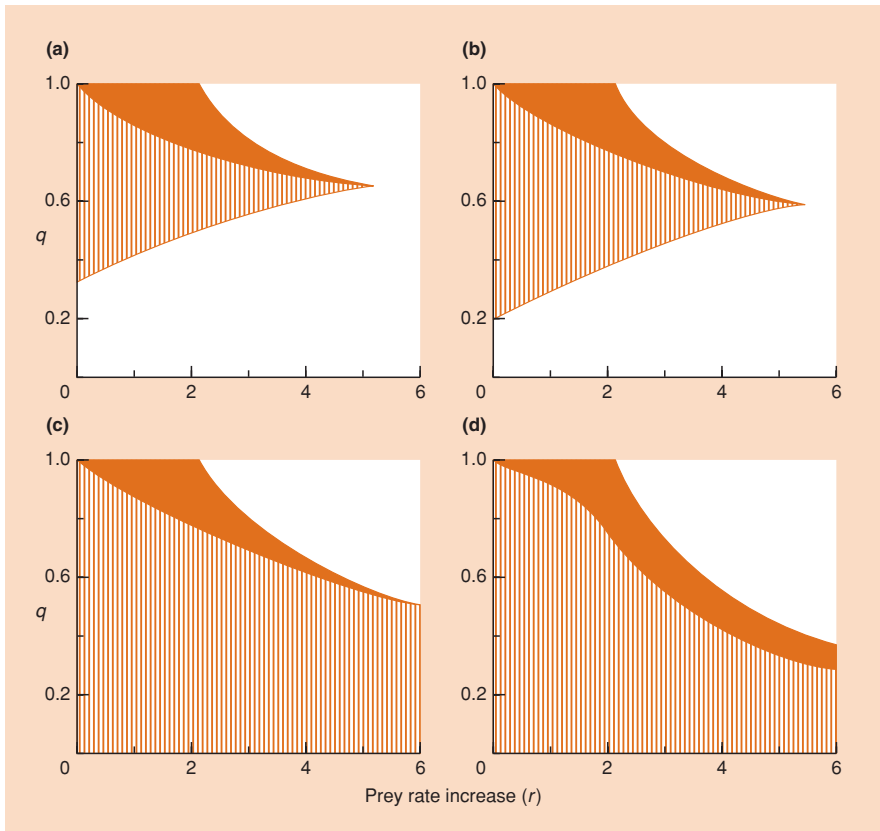

Figure 10.13 May's (1978) model of host–parasitoid aggregation, with host self-limitation incorporated, illustrates that aggregation can enhance stability and give rise to stability at low values of $q = H^*/K$. In the solid orange area there is an exponential approach to equilibrium; in the hatched area there is an oscillatory approach to equilibrium; outside these there is instability (the oscillations either diverge or are sustained). The four figures are for four values of k, the exponent of the negative binomial distribution in the model: (a) $k = \infty$: no aggregation, least stability; (b) $k = 2$; (c) $k = 1$; (d) $k = 0.1$: most aggregation. (After Hassell, 1978.)

aggregation of risk strengthens direct (temporal) density dependences

To summarize, aggregation of risk stabilizes host–parasitoid interactions by strengthening direct (not delayed) density dependencies that already exist (Taylor, 1993). The stabilizing powers of this spatial phenomenon, aggregation of risk, therefore arise not from any spatial density dependencies, but from its translation into direct, temporal density dependence.

But how does aggregation of risk relate to the aggregative responses of parasitoids? And do aggregative responses and aggregation of risk necessarily lead to enhanced stability? We can address these questions by examining Figure 10.14, bearing in mind from Section 9.6 that aggregated predators do not necessarily spend most time foraging in patches of high host density (spatial density dependence); foraging time can also be negatively correlated with host density (inverse density dependence) or independent of host density. Start with Figure 10.14a. The distribution of parasitoids over host patches follows a perfect, straight line density-dependent relationship. But, since the host : parasitoid ratio is therefore the same in each host patch, the risk is likely to be the same in each host patch, too. Thus, positive spatial density dependence does not necessarily lead to aggregation of risk and does not necessarily enhance stability. On the other hand, with a directly density-dependent relationship that accelerates (Figure 10.14b), there does appear to be aggregation of risk, and this might well enhance stability (Hassell & May, 1973); but it turns out that whether or not it does so depends on the parasitoids' functional response (Ives, 1992a). With a type 1 response, assumed by most analyses, stability is enhanced. But with a more realistic type 2 response, initial increases in density-dependent aggregation from zero aggregation actually decrease aggregation of risk and are destabilizing. Only high levels of density-dependent aggregation are stabilizing.

aggregative responses and aggregation of risk

Moreover, it is clear from Figures 10.14c and d that there can be considerable aggregation of risk with either inverse spatial density dependence or no spatial density dependence of any sort – and these would not be counteracted by a type 2 functional response. In partial answer to our two questions above, therefore, aggregative responses that are spatially density dependent are actually *least* likely to lead to aggregation of risk, and therefore least likely to enhance stability.

In practice, of course, with real sets of data (like those in Figure 9.20), aggregation of risk will often arise from a combination of spatially density-dependent (direct or inverse) and density-independent responses (Chesson & Murdoch, 1986; Pacala & Hassell, 1991). Pacala, Hassell and coworkers have

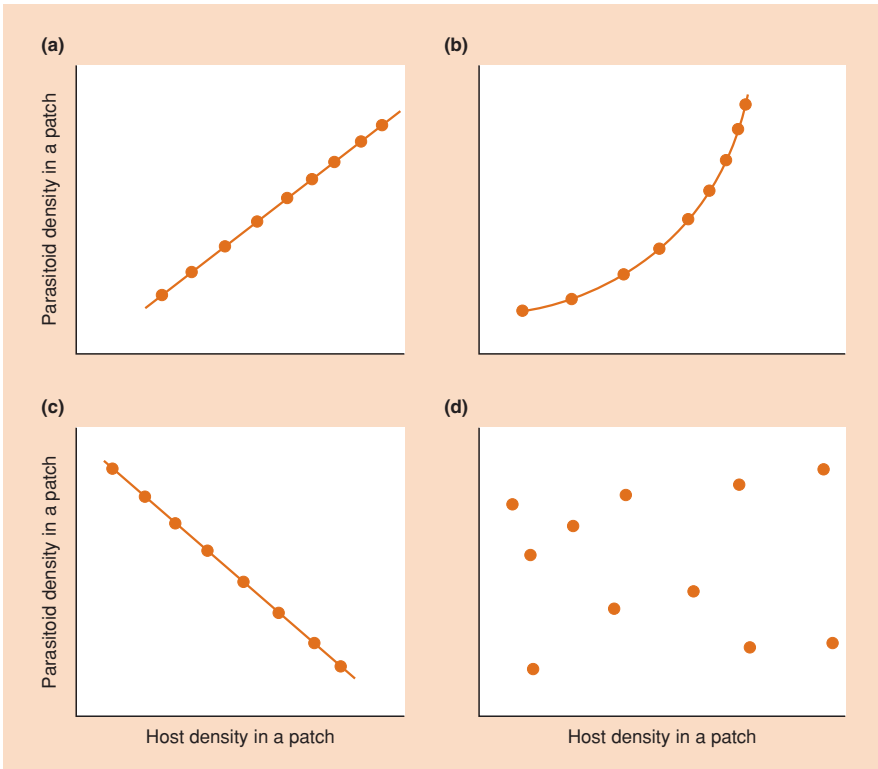

Figure 10.14 The aggregative responses of parasitoids and the aggregation of risk. (a) Parasitoids aggregate in high host-density patches, but the parasitoid : host ratio is the same in all patches (a perfect straight-line relationship), and hence the risk to hosts is apparently the same in all patches. (b) Parasitoid aggregation to high host-density patches now accelerates with increasing host density, and hosts in high-density patches are thus apparently at greater risk of parasitization: there is aggregation of risk. (c) With perfect inverse density dependence (i.e. parasitoid aggregation in *low* host-density patches) the hosts in the low-density patches are apparently at a much greater risk of parasitization: again there is aggregation of risk. (d) Even with no aggregative response (density independence) the hosts in some patches are apparently at a greater risk of parasitization (are subject to a higher parasitoid : host ratio) than others: here too there is aggregation of risk.

called the former the 'host density dependent' (HDD) component, and the latter the 'host density independent' (HDI) component, and have described methods by which, in real data sets like Figure 9.20, the aggregation of risk can be split between them. Interestingly, in an analysis of 65 data sets, representing 26 different host–parasitoid combinations (Pacala & Hassell, 1991), 18 appeared to have sufficient aggregation of risk to stabilize their interactions, but for 14 of these 18 cases, it was HDI variation that contributed most to the total, further weakening any imagined link between spatial density dependence and stability.

10.5.5 Heterogeneity in some continuous-time models

We have been pursuing parasitoids and hosts; and in doing so we have been retaining certain structural features in our analysis that we should now reconsider. In particular, our parasitoids have been assumed, in effect, to arrange themselves over host patches at the beginning of a generation (or whatever the time interval is between t and $t + 1$), and then to have to suffer the consequences of that arrangement until the beginning of the next generation. But suppose we move into continuous time – as appropriate for many parasitoids as it is for many other predators. Now, aggregation should be assumed to occur on a continuous basis, too. Predators in a depleted, or even a depleting, patch should leave and redistribute themselves (see Section 9.6.2). The whole basis of pseudo-interference and hence stability, namely wasted predator attacks in high predator density patches, tends to disappear.

continuous redistribution of predators and prey

Murdoch and Stewart-Oaten (1989) went to, perhaps, the opposite extreme to the one we have been considering, by constructing a continuous-time model in which prey moved instantly into patches to replace prey that had been consumed, and predators moved instantly into patches to maintain a consistent pattern of predator–prey covariation over space. The effects on their otherwise neutrally stable Lotka–Volterra model contrast strongly with those we have seen previously. First, predator aggregation that is independent of local prey density now has *no* effect on either stability or prey density. However, predator aggregation that is directly dependent on local prey density has an effect that depends on the strength of this dependence – although it always lowers prey density (because predator efficiency is increased). If such density dependence is relatively weak (as Murdoch and Stewart-Oaten argue it usually is in practice), then stability is *de*creased. Only if it is stronger than seems typical in nature is stability increased.

Other, less 'extreme' continuous-time formulations (Ives, 1992b), or those that combine discrete generations with redistribution within generations (Rohani *et al.*, 1994), produce results

that are themselves intermediate between the 'Nicholson–Bailey extreme' and the 'Murdoch–Stewart-Oaten extreme'. It seems certain, however, that a preoccupation with models lacking within-generation movement has, in the past, led to a serious overestimation of the significance of aggregation to patches of high host density in stabilizing host–parasitoid interactions.

10.5.6 The metapopulation perspective

The continuous- and discrete-time approaches clearly differ, but they share a common perspective in seeing predator–prey interactions occurring within single populations, albeit populations with inbuilt variability. An alternative is a 'metapopulation' perspective (see Section 6.9), in which environmental patches support subpopulations that have their own internal dynamics, but are linked to other subpopulations by movement between patches.

A number of studies have investigated predator–prey metapopulation models, usually with unstable dynamics within patches. Mathematical difficulties have often limited analysis to two-patch models, where, if the patches are the same, and dispersal is uniform, stability is unaffected: patchiness and dispersal have no effect in their own right (Murdoch *et al.*, 1992; Holt & Hassell, 1993).

patch differences stabilize through asynchrony

Differences between the patches, however, tend, in themselves, to stabilize the interaction (Ives, 1992b; Murdoch *et al.*, 1992; Holt & Hassell, 1993). The reason is that any difference in parameter values between patches leads to asynchrony in the fluctuations in the patches. Inevitably, therefore, a population at the peak of its cycle tends to lose more by dispersal than it gains, a population at a trough tends to gain more than it loses, and so on. Dispersal and asynchrony together, therefore, give rise to stabilizing temporal density dependence in net migration rates.

The situation becomes much more complex with the inclusion of aggregative behavior, since dispersal rates themselves become a much more complex function of both prey and predator densities. Aggregation appears to have two opposing effects (Murdoch *et al.*, 1992). It tends to increase the asynchrony between fluctuations in predator abundance (enhancing stability) but to reduce the asynchrony between prey fluctuations (decreasing stability). The balance between these forces appears to be sensitive to the strength of the aggregation, but perhaps even more sensitive to the assumptions built into the models (Godfray & Pacala, 1992; Ives, 1992b; Murdoch *et al.*, 1992). Aggregation may either stabilize or destabilize. In contrast to previous analyses, it has no clear effect on prey density since its stabilizing powers are not linked to predator efficiency.

an explicitly, and visually, spatial model

The treatment of a spatially heterogeneous predator–prey interaction as a problem in metapopulation dynamics was taken a stage further by Comins *et al.* (1992). They constructed computer models of an environment consisting of a patchwork of squares, which could actually be visualized as such (Figure 10.15). In each generation, two processes occurred in sequence. First, a fraction, μ_P, of predators, and a fraction, μ_N, of prey, dispersed from each square to the eight neighboring squares. At the same time, predators and prey from the eight neighboring squares were dispersing into the first square. Thus, for example, the dynamics for the density of prey, $N_{i,t+1}$, in square i in generation $t+1$, was given by:

$$N_{i,t+1} = N_{i,t}(1 - \mu_N) + \mu_N \bar{N}_{i,t} \qquad (10.23)$$

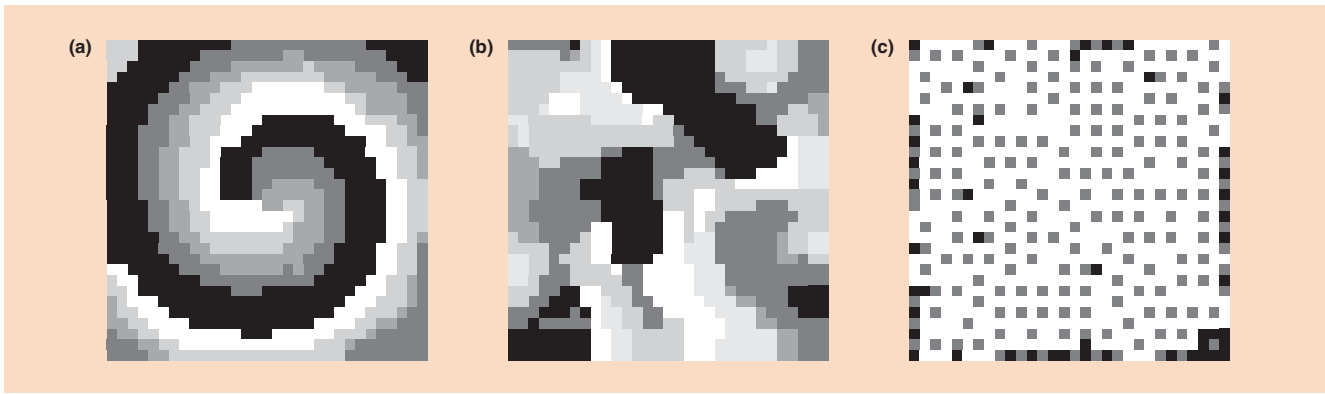

Figure 10.15 Instantaneous maps of population density for simulations of the dispersal model of Comins *et al.* (1992) with Nicholson–Bailey local dynamics. Different levels of shading represent different densities of hosts and parasitoids. Black squares represent empty patches; dark shades becoming paler represent patches with increasing host densities; light shades to white represent patches with hosts and increasing parasitoid densities. (a) Spirals: $\mu_N = 1$, $\mu_P = 0.89$; (b) spatial chaos: $\mu_i = 0.2$, $\mu_P = 0.89$; (c) a 'crystalline lattice': $\mu_N = 0.05$; $\mu_P = 1$. (After Comins *et al.*, 1992.)

or:

$$N_{i,t+1} = N_{i,t} + \mu_N (\bar{N}_{i,t} - N_{i,t}), \quad (10.24)$$

where $\bar{N}_{i,t}$ is the mean density in the eight squares neighboring square i in generation t. The second phase then consisted of one generation of standard predator–prey dynamics, either following the Nicholson–Bailey equations or a discrete-time version of the Lotka–Volterra equations (May, 1973). Simulations were started with random prey and predator populations in a single patch, with all the other patches empty.

We know that within individual squares, if they existed in isolation, the dynamics would be unstable. But within the patchwork of squares as a whole, stable or at least highly persistent patterns can readily be generated (Figure 10.15). The general message is similar to the results that we have already seen: that stability can be generated by dispersal in metapopulations in which different patches are fluctuating asynchronously. Note especially, in this case, that a patch experiences a net gain in migrants when its density is lower than the mean of the eight patches with which it connects (Equation 10.24) but experiences a net loss when its density is higher – a kind of density dependence. Note, too, that the asynchrony arises in the present case because the population has spread from a single initial patch (all patches are, in principle, the same) and that it is maintained by dispersal being limited to the neighboring patches (rather than being a powerful equalizing force over all patches).

emergent spatial patterns

Moreover, the explicitly spatial aspects of this model have, quite literally, added another dimension to the results. Depending on the dispersal fractions and the host reproductive rate, a number of quite different spatial structures can be generated (although they tend to blur into one another) (Figure 10.15a–c). 'Spatial chaos' can occur, in which a complex set of interacting wave fronts are established, each one persisting only briefly. With somewhat different parameter values, and especially when both predator and prey are highly mobile, the patterns are more structured than chaotic, with 'spiral waves' rotating around almost immobile focal points. The model, therefore, makes the point very graphically that persistence at the level of a whole population does not necessarily imply either uniformity across the population or stability in individual parts of it. Static 'crystalline lattices' can even occur within a narrow range of parameter values, with highly mobile predators and rather sedentary prey, emphasizing that pattern can be generated internally within a population even in an intrinsically homogeneous environment.

Is there one general message that can be taken from this body of theory? Certainly, we cannot say 'aggregation does *this* or *that* to predator–prey interactions'. Rather, aggregation can have a variety of effects, and knowing which of these is likely will require detailed knowledge of predator and prey biology for the interaction concerned. In particular, the effects of aggregation have been seen to depend on the predator's functional response, the extent of host self-regulation, and so on – other features that we have examined in isolation. It is necessary, as stressed at the beginning of this chapter, in seeking to understand complex processes, to isolate conceptually the different components. But it is also necessary, ultimately, to recombine those components.

10.5.7 Aggregation, heterogeneity and spatial variation in practice

What, then, can be said about the role of spatial variation in practice? The stabilizing effects of heterogeneity were demonstrated famously, long ago, by Huffaker (1958; Huffaker *et al.*, 1963), who studied a system in which a predatory mite fed on a herbivorous mite, which fed on oranges interspersed amongst rubber balls in a tray. In the absence of its predator, the prey maintained a fluctuating but persistent population (Figure 10.16a); but if the predator was added during the early stages of prey population growth, it rapidly increased its own population size, consumed all of its prey and then became extinct itself (Figure 10.16b). The interaction was altered, however, when Huffaker made his microcosm more 'patchy' (creating, effectively, a metapopulation, though the term had not been coined at the time). He spread the oranges further apart, and partially isolated each one by placing a complex arrangement of vaseline barriers in the tray, which the mites could not cross. But he facilitated the dispersal of the prey by inserting a number of upright sticks from which they could launch themselves on silken strands carried by air currents. Dispersal between patches was therefore much easier for the prey than it was for the predators. In a patch occupied by both predators and prey, the predators consumed all the prey and then either became extinct themselves or dispersed (with a low rate of success) to a new patch. But in patches occupied by prey alone, there was rapid, unhampered growth accompanied by successful dispersal to new patches. In a patch occupied by predators alone, there was usually death of the predators before their food arrived. Each patch was therefore ultimately doomed to the extinction of both predators and prey. But overall, at any one time, there was a mosaic of unoccupied patches, prey–predator patches heading for extinction, and thriving prey patches; and this mosaic was capable of maintaining persistent populations of both predators and prey (Figure 10.16c).

metapopulation effects in mites, beetles and ciliates

Subsequently, others, too, have demonstrated the power of a metapopulation structure in promoting the persistence of coupled predator and prey populations when their dynamics in individual subpopulations are unstable. Figure 10.17a, for example, shows this for a parasitoid attacking its beetle host. Figure 10.17b shows similar results for prey and predatory ciliates

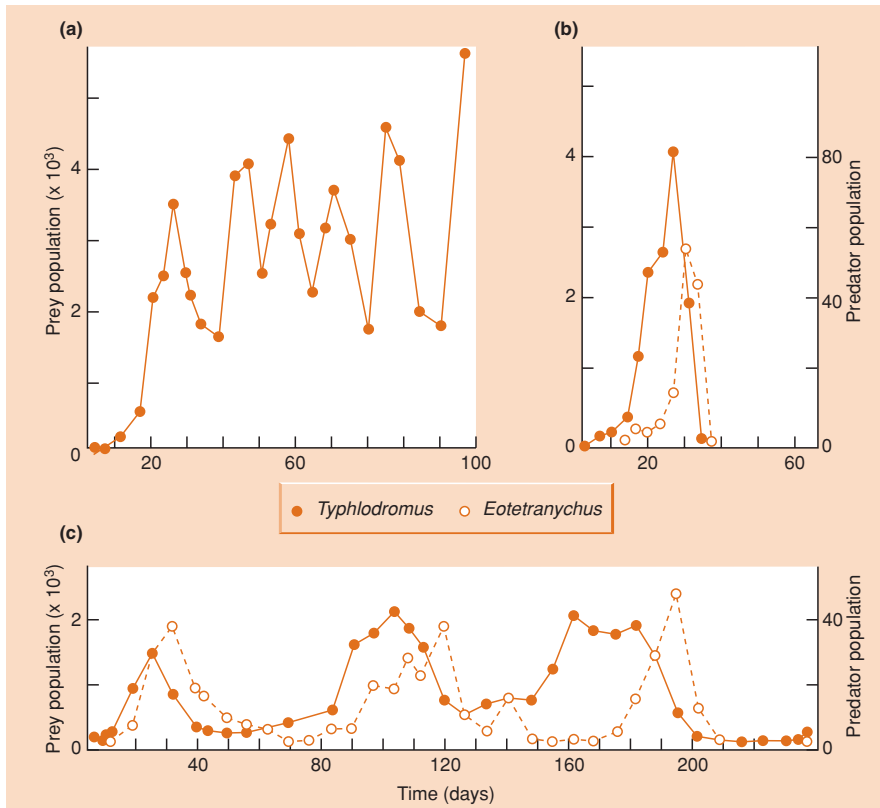

Figure 10.16 Hide and seek: predator–prey interactions between the mite *Eotetranychus sexmaculatus* and its predator, the mite *Typhlodromus occidentalis*. (a) Population fluctuations of *Eotetranychus* without its predator. (b) A single oscillation of the predator and prey in a simple system. (c) Sustained oscillations in a more complex system. (After Huffaker, 1958.)

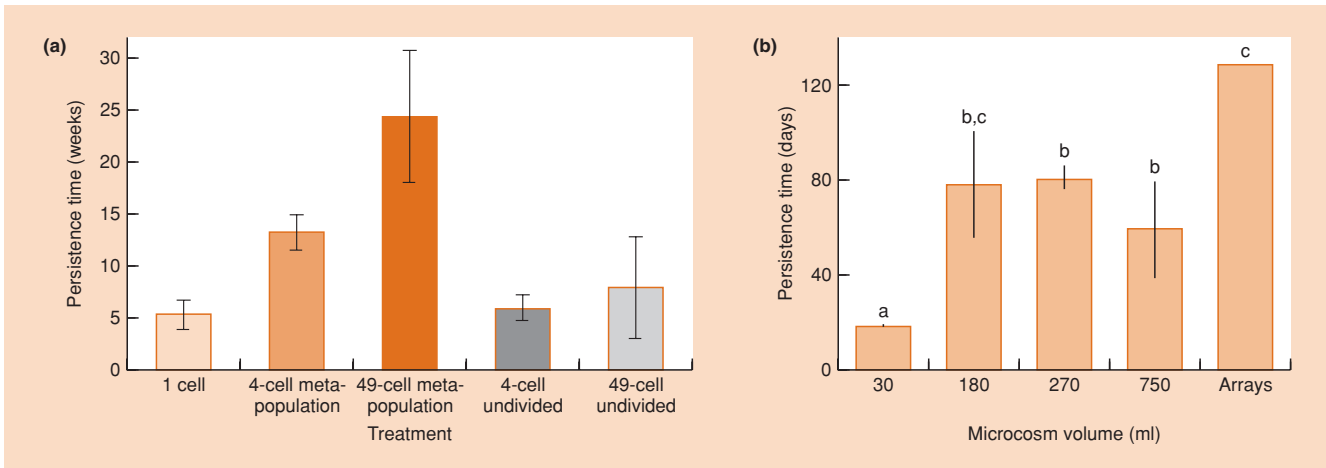

Figure 10.17 A metapopulation structure can increase the persistence of predator–prey interactions. (a) The parasitoid, *Anisopteromalus calandrae*, attacking its bruchid beetle host, *Callosobruchus chinensis*, living on beans either in small single 'cells' (short persistence time, left), or in combinations of cells (four or 49), which either had free access between them so that they effectively constituted a single population (persistence time not significantly increased, right), or had limited (infrequent) movement between cells so that they constituted a metapopulation of separate subpopulations (increased persistence time, center). Bars show standard errors. (After Bonsall *et al.*, 2002.) (b) The predatory ciliate, *Didinium nasutum*, feeding on the bacterivorous ciliate, *Colpidium striatum*, in bottles of various volumes, where persistence time varied little, except in the smallest populations (30 ml) where times were shorter, and also in 'arrays' of nine or 25 linked 30 ml bottles (metapopulations), where persistence was greatly prolonged: all populations persisted until the end of the experiment (130 days). Bars show standard errors; different letters above bars indicate treatments that were significantly different from one another ($P < 0.05$). (After Holyoak & Lawler, 1996.)

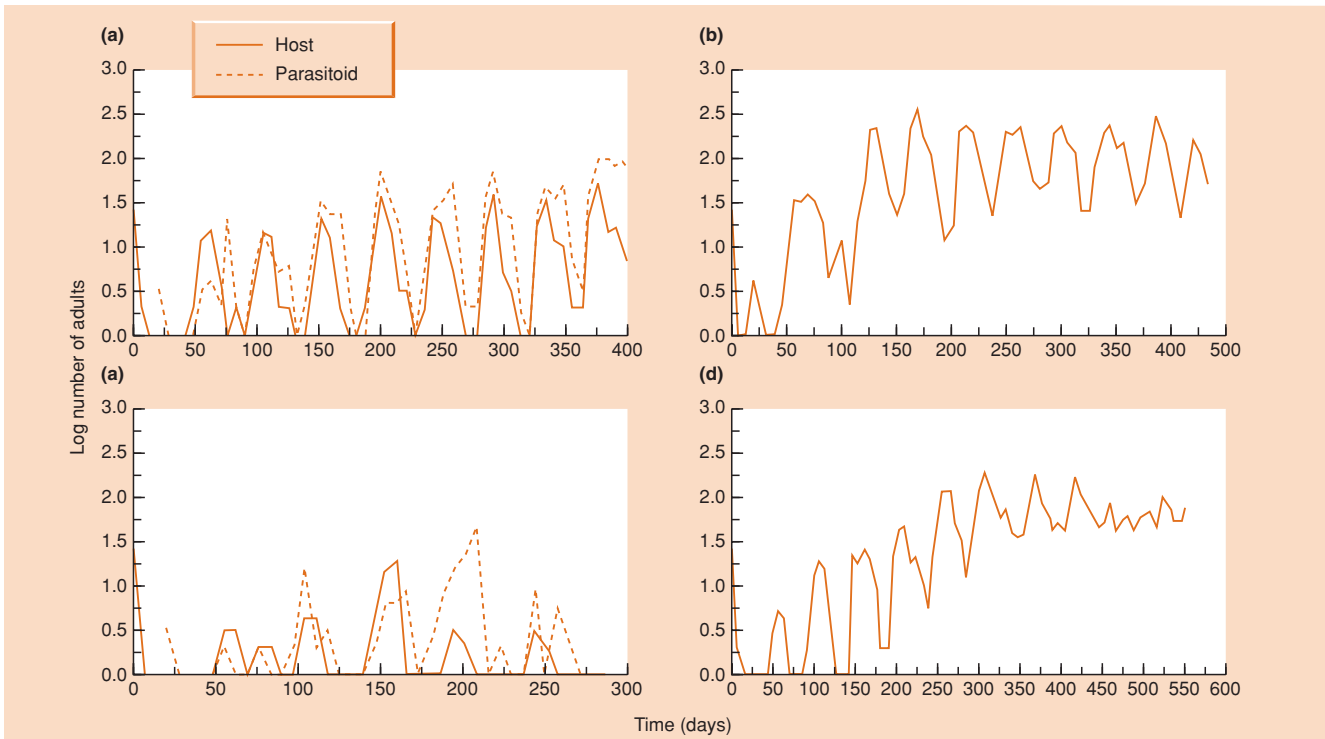

Figure 10.18 Long-term population dynamics in laboratory population cages of a host (*Plodia interpunctella*), with and without its parasitoid (*Venturia canescens*). (a) Host and parasitoid in deep medium, exhibiting coupled cycles in abundance, approximately one host generation in length. (b) The host alone in deep medium, exhibiting similar cycles. (c) Host and parasitoid in shallow medium, unable to persist. (d) The host alone in shallow medium, able to persist. The deep medium provides a refuge from attack for a proportion of the host population that is not present in the shallow medium (see Section 10.5.2). All data sets are selected from several replicates showing the same pattern. (After Begon *et al.*, 1995.)

(protists), where, in support of the role of a metapopulation structure, it was also possible to demonstrate asynchrony in the dynamics of individual subpopulations and frequent local prey extinctions and recolonizations (Holyoak & Lawler, 1996).

a refuge for a moth

A study providing support for the stabilizing powers of a physical refuge is illustrated in Figure 10.18, based on the same *Plodia–Venturia* host–parasitoid system as that in Figure 10.1c. In this case, hosts living deeper in their food are beyond the reach of the parasitoids attempting to lay their eggs in them. In the absence of this refuge, in a shallow food medium, this host–parasitoid interaction is unable to persist (Figure 10.18c), although the host alone does so readily (Figure 10.18d). With a refuge present, however, in a deeper food medium, the host and parasitoid can apparently persist together indefinitely (Figure 10.18a).

more mites: a metapopulation or a refuge?

In fact, though, the distinctions between different types of spatial heterogeneity may not be as clear cut in real systems as they are in mathematical models. Ellner *et al.* (2001), for example, examined a system of predatory mites, *Phytoseiulus persimilis*, feeding on herbivorous mites, *Tetranychus urticae*, feeding on bean plants, *Phaseolus lunatus*. On individual plants and on a single 'continent' of 90 plants (Figure 10.19a), the system had no long-term persistence (Figure 10.19c). However, when the Styrofoam sheet supporting the plants was split into eight islands of 10 plants, connected by bridges that limited the mites' powers of dispersal (Figure 10.19b), persistence was apparently indefinite (Figure 10.19d, e). It would be easy to jump to the conclusion that stability was increased by the eight-island metapopulation structure. But when Ellner *et al.* examined mathematical models of the system that allowed the various aspects of the altered layout to be investigated one by one, they could detect no significant effect of such a structure. Instead, they suggested that the enhanced stability arose from a different aspect: a reduction in the predators' ability to detect and respond to prey outbreaks on individual plants – a prey 'refuge' effect that could arise in the absence of any explicit spatial structure.

real data confirm the complexity of natural systems

One major problem in making pronouncements about the stabilizing role of aggregation of risk is that although, as we have seen, there have been wide-

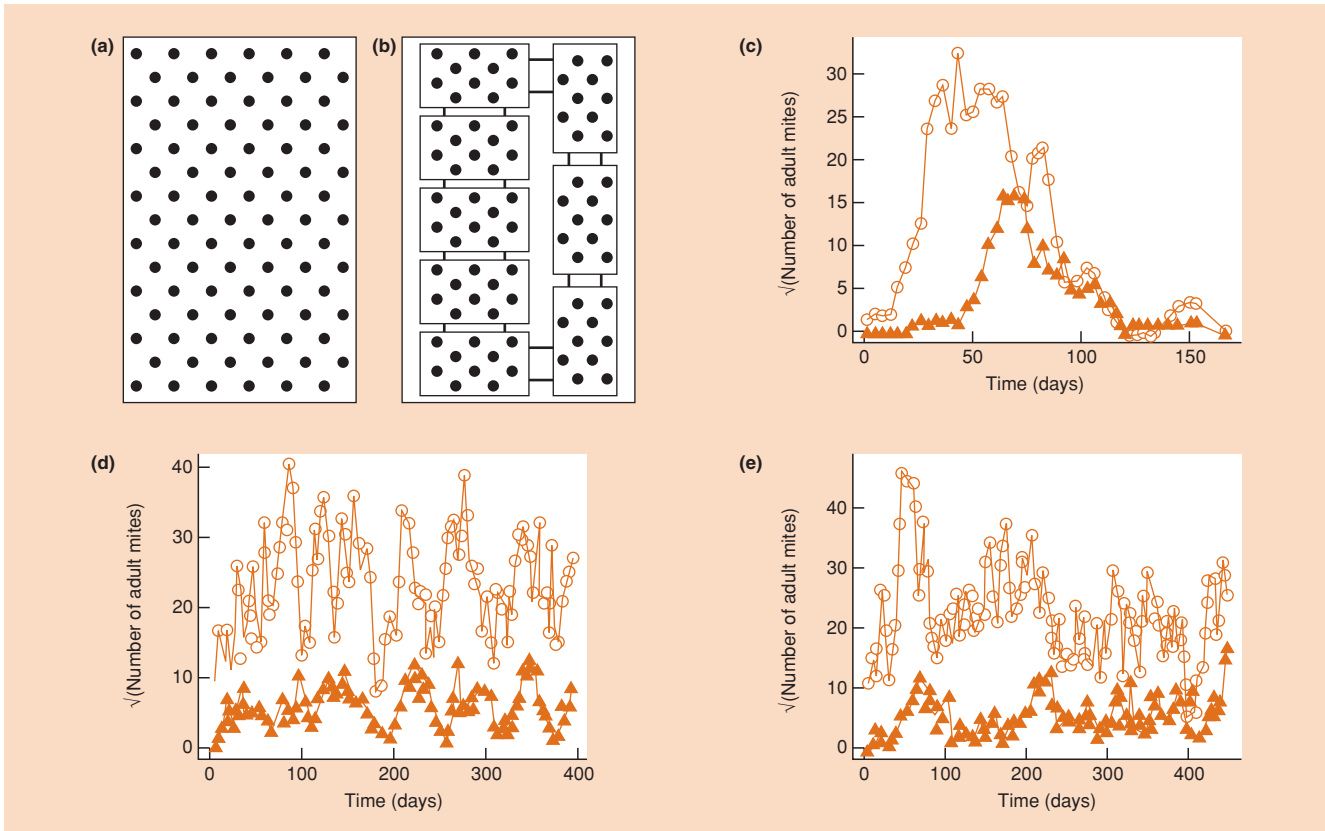

Figure 10.19 The population dynamics of the predatory mite, *Phytoseiulus persimilis*, and its herbivorous mite prey, *Tetranychus urticae*. They interacted either (a) on a single continent of 90 bean plants, the dynamics of which are shown in (c) (▲, predators; ○, prey), or (b) in a metapopulation of eight islands of 10 plants. For the latter, the dynamics of two replicates are shown in (d) and (e), where persistence (stability) is clearly enhanced. (After Ellner *et al.*, 2001.)

ranging surveys of the data on spatial distributions of attacks, these data generally come from studies of very short duration – often of only one generation. We do not know if the observed spatial patterns are typical for that interaction; nor do we know if the population dynamics show the degree of stability that the spatial patterns might seem to predict. One investigation that has examined population dynamics and spatial distributions over several generations is that of Redfern *et al.* (1992), who made a 7-year (seven-generation) study of two tephritid fly species that attack thistles and the guilds of parasitoids that attack the flies. For one host, *Terellia serratulae* (Figure 10.20a) there was evidence of year-to-year density dependence in the overall rate of parasitism (Figure 10.20b), but no strong evidence of significant levels of aggregation within generations, either overall (Figure 10.20c) or for parasitoid species individually. For the other species, *Urophora stylata* (Figure 10.20d), there was no apparent temporal density dependence but good evidence for the aggregation of risk (Figure 10.20e, f), and, to repeat a pattern we have seen before, most heterogeneity was contributed by the HDI component. It cannot be said, however, that the patterns of this study fit neatly, overall, to the theory we have outlined. First, both hosts were attacked by several parasitoid species – not one, as assumed by most models. Second, the levels of aggregation (and to a lesser extent the HDI or HDD contributions) varied considerably and apparently randomly from year to year (Figure 10.20c, f): no one year was typical, and no single 'snap-shot' could have captured either interaction. Finally, while the relatively stable dynamics of *Terellia* may have reflected the more demonstrable direct density dependence in parasitism, this appeared to be quite unconnected to any differences in the aggregation of risk.

spatial heterogeneity and the most effective biological control agents

The effects of spatial heterogeneities on the stability of predator–prey dynamics are not only of purely scientific interest. They have also been the subject of lively debate (Hawkins & Cornell, 1999) in considering the properties and nature of biological control agents: natural enemies of a pest that are imported into an area, or otherwise aided and abetted, in order to control the pest (see Section 15.2.5). What is required of a good biological control agent is the ability to

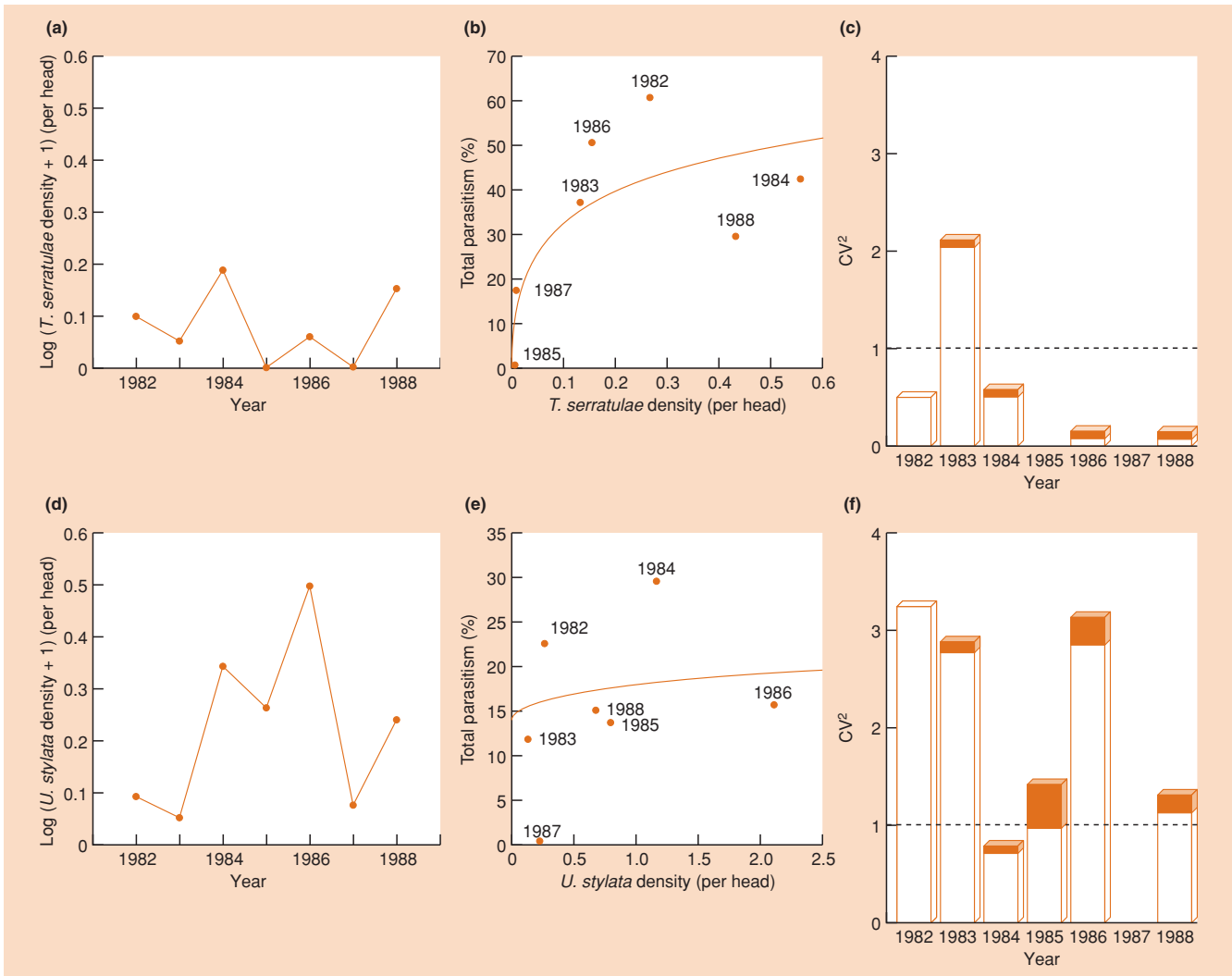

Figure 10.20 Attacks by parasitoids on tephrytid flies (*Terellia serratulae* and *Urophora stylata*) that attack thistle flower-heads. The dynamics of the populations are shown for *T. serratulae* in (a) and for *U. stylata* in (d). Temporal density dependence of parasitoid attacks on *T. serratulae* (b) is significant ($r^2 = 0.75$; $P < 0.05$), but for *U. stylata* (e) it is not ($r^2 = 0.44$; $P > 0.05$); both fitted lines take the form $y = a + b \log_{10} x$. However, whereas for *T. serratulae* (c) there is little aggregation of risk of parasitoid attack within years (measured as $CV^2 > 1$ for aggregation), with *U. stylata* (f) there is far more, most of which is HDI (no shading) rather than HDD (dark shading). (After Redfern *et al.*, 1992.)

reduce the prey (pest) to a stable abundance well below its normal, harmful level, and we have seen that some theoretical analyses suggest that this is precisely what aggregative responses help to generate. Establishing such a link in practice, however, has not proved easy. Murdoch *et al.* (1995), for example, noted that the California red scale, *Aonidiella aurantii*, an insect pest of citrus plants worldwide, appeared to be kept at low and remarkably stable densities in southern California by a parasitoid introduced to control it, *Aphytis mellitus*. The existence of a partial refuge from parasitization for the red scale seemed a plausible hypothesis for how this was achieved: on bark in the interior of the trees, rates of parasitism were very low and scale densities high, seemingly as a result of the activities of ants there that interfered with the searching parasitoids. Murdoch *et al.*, therefore, tested this hypothesis by a field experiment in which ants were removed from a number of trees. Parasitization rates in the refuge did increase, and scale abundance declined there (Figure 10.21), and there was some evidence that parasitization rates, and scale abundance, in the population as a whole were then more variable. But these effects were only slight and apparently short term, and there was certainly no evidence that scale abundance overall was increased by any diminution of the refuge effect.

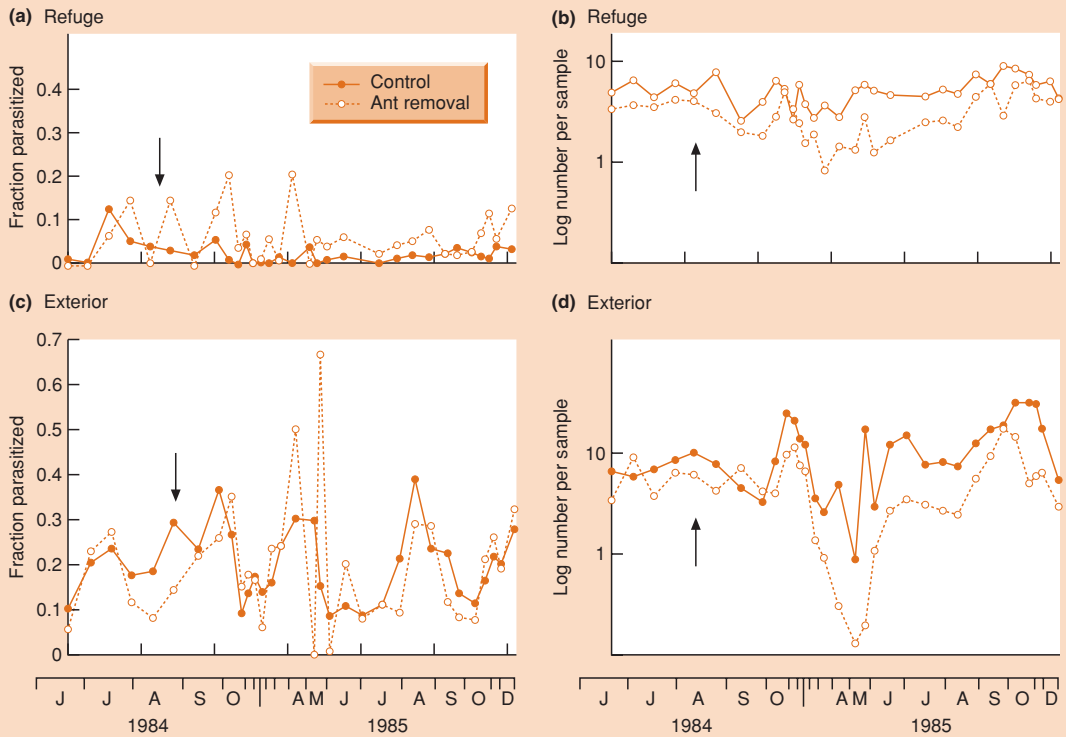

Figure 10.21 Results of a field experiment to test the hypothesis that the parasitoid *Aphytis mellitus* maintains the abundance of the California red scale, *Aonidiella aurantii*, at stable low levels because of a partial refuge from parasitization in the interior portions of citrus trees, where ants interfere with the parasitoids. When ants were removed from blocks of trees (time of removal indicated by the arrow), the fraction parasitized in the refuge tended to be higher (a), and scale abundance there was lower (b), but outside the refuge ('exterior') the fraction parasitized was only marginally more variable (c), and scale abundance was only more variable over one relatively brief period and tended to be lower than on control trees (d). (After Murdoch *et al.*, 1995.)

Moreover, Murdoch *et al.* (1985) had earlier argued that, in general, pest populations persist after successful biological control not as a result of aggregative responses, but because of the stochastic creation of host patches by colonization and their subsequent extinction when discovered by the agent: essentially, a metapopulation effect. Waage and Greathead (1988), however, suggested that a broader perspective could incorporate both aggregative responses and metapopulation effects. They proposed that scale insects and other homopterans, and mites (like Huffaker's), which may reproduce to have many generations within a patch, are often stabilized by asynchronies in the dynamics of different patches; whereas lepidopterans and hymenopterans, which typically occupy a patch for only part of a single generation, may often be stabilized by an aggregative response. In fact, though, with biological control, like predator–prey dynamics generally, building convincing links between patterns in population stability of natural populations and particular stabilizing mechanisms – or combinations of mechanisms – remains a challenge for the future.

10.6 Multiple equilibria: an explanation for outbreaks?

When predator and prey populations interact, there can sometimes be sudden changes in the abundance of one or both partners: outbreaks or crashes. Of course, this may reflect an equally sudden change in the environment, but ecologists working in a variety of fields have come to realize that there is not necessarily just one equilibrium combination of a predator and prey (about which there may or may not be oscillations). There can, instead, be 'multiple equilibria' or 'alternative stable states'.

a model with multiple equilibria

Figure 10.22 is a model with multiple equilibria. The prey zero isocline has both a vertical section at low densities and a hump. This could reflect a type 3 functional response of a predator that also has a long handling time, or perhaps the combination of an aggregative response and an Allee effect in the prey. As a consequence, the predator zero

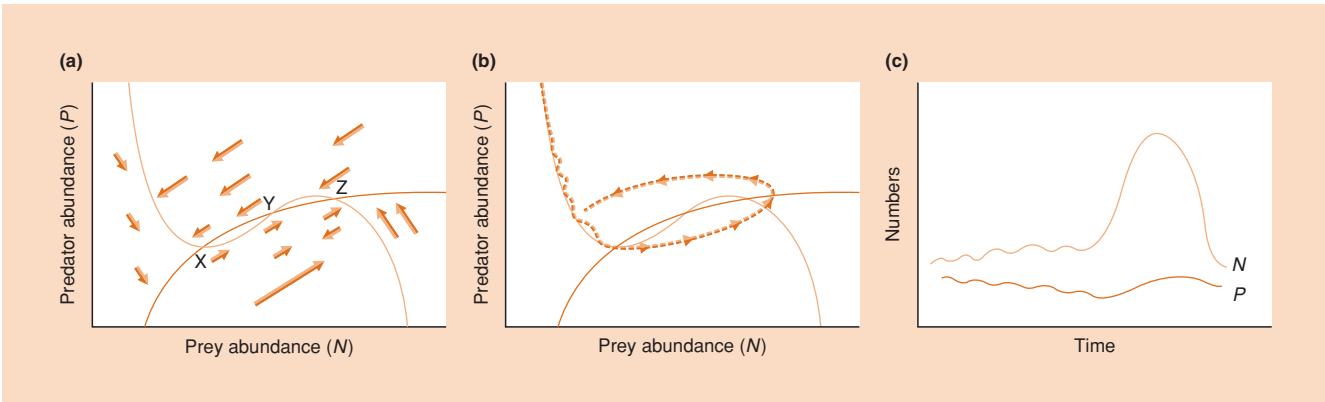

Figure 10.22 A predator–prey zero isocline model with multiple equilibria. (a) The prey zero isocline has a vertical section at low densities and a hump; the predator zero isocline can therefore cross it three times. Intersections X and Z are stable equilibria, but intersection Y is an unstable 'breakpoint' from which the joint abundances move towards either intersection X or intersection Z. (b) A feasible path that the joint abundances might take when subject to the forces shown in (a). (c) The same joint abundances plotted as numbers against time, showing that an interaction with characteristics that do not change can lead to apparent 'outbreaks' in abundance.

isocline crosses the prey zero isocline three times. The strengths and directions of the arrows in Figure 10.22a indicate that two of these points (X and Z) are fairly stable equilibria (although there are oscillations around each). The third point (Y), however, is unstable: populations near here will move towards either point X or point Z. Moreover, there are joint populations close to point X where the arrows lead to the zone around point Z, and joint populations close to point Z where the arrows lead back to the zone around point X. Even small environmental perturbations could put a population near point X on a path towards point Z, and vice versa.

The behavior of a hypothetical population, consistent with the arrows in Figure 10.22a, is plotted in Figure 10.22b on a joint abundance diagram, and in Figure 10.22c as a graph of numbers against time. The prey population, in particular, displays an 'eruption' in abundance, as it moves from a low-density equilibrium to a high-density equilibrium and back again. This eruption is in no sense a reflection of an equally marked change in the environment. It is, on the contrary, a pattern of abundance generated by the interaction itself (plus a small amount of environmental 'noise'), and in particular it reflects the existence of multiple equilibria. Similar explanations may be invoked to explain apparently complicated patterns of abundance in nature.

There are certainly examples of natural populations exhibiting outbreaks of abundance from levels that are otherwise low and apparently stable (Figure 10.23a), and there are other examples in which populations appear to alternate between two stable densities (Figure 10.23b). But it does not follow that each of these examples is necessarily an interaction with multiple equilibria.

sudden changes in abundance: multiple equilibria – or sudden changes in the environment

In some cases, a plausible argument for multiple equilibria can be put forward. This is true, for instance, of Clark's (1964) work in Australia on the eucalyptus psyllid (*Cardiaspina albitextura*), a homopteran bug (Figure 10.23a). These insects appear to have a low-density equilibrium maintained by their natural predators (especially birds), and a much less stable high-density equilibrium reflecting intraspecific competition (the destruction of host tree foliage leading to reductions in fecundity and survivorship). Outbreaks from one to the other can occur when there is just a short-term failure of the predators to react to an increase in the density of adult psyllids. Similarly, the observation of two alternative equilibria in Figure 10.23b for the viburnum whitefly, *Aleurotrachelus jelinekii*, is reinforced by a model for that population which predicts the same pattern (Southwood *et al.*, 1989).

Alternative stable states have also been proposed for a number of plant–herbivore interactions, often where increased grazing pressure seems to have led to the 'collapse' of the vegetation from a high biomass to a much lower one, which is then stable in the sense that there is no return to the high biomass state even when grazing pressure is severely reduced (van de Koppel *et al.*, 1997). The grasslands of the Sahel region of Africa, grazed by livestock, and the arctic plants along the coast of Hudson Bay in Canada, grazed by geese, are both examples. The conventional explanation (Noy-Meir, 1975) has essentially been that depicted in Figure 10.22: when driven to a low biomass, plants may have very little material above ground and hence very limited powers of immediate regrowth. This is a classic 'Allee effect' – the prey

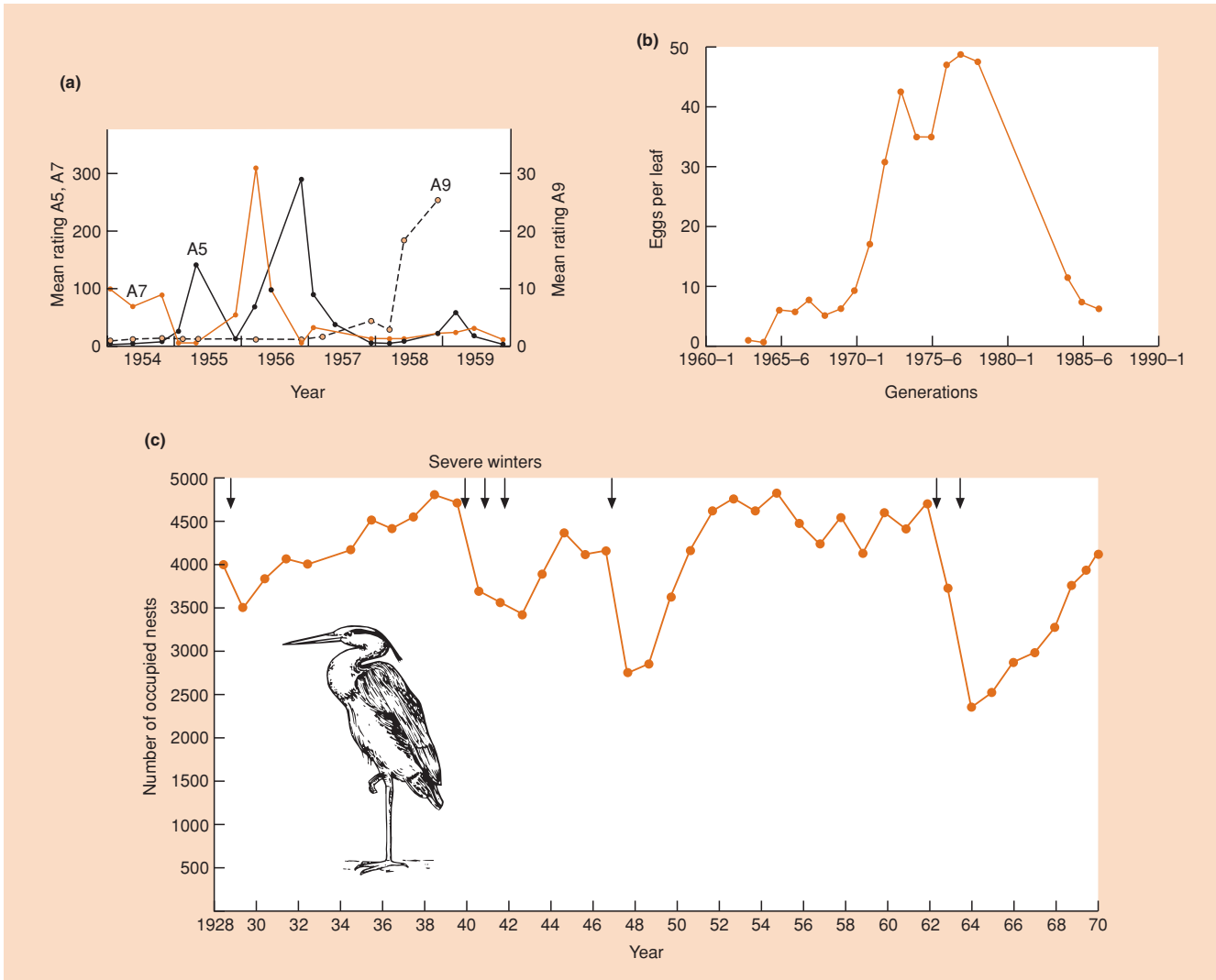

Figure 10.23 Possible examples of outbreaks and multiple equilibria. (a) Mean ratings of relative abundance of the eucalyptus psyllid, *Cardiaspina albitextura*, in three study areas in Australia (A5, A7 and A9). (After Clark, 1962.) (b) The mean number of eggs per leaf of the viburnum whitefly, *Aleurotrachelus jelinekii*, on a viburnum bush in Silwood Park, Berkshire, UK. No samples were taken between 1978 and 1979, and 1984 and 1985. (After Southwood *et al.*, 1989.) (c) Changes in the abundance of herons (*Ardea cinerea*) in England and Wales (measured by the number of nests occupied) are readily attributable to changes in environmental conditions (particularly severe winters). (After Stafford, 1971.)

suffering from too *low* an abundance – leading to a marked 'hump' in their isocline. It may also be, however, that the plants' problems at low biomass are compounded by soil deterioration – erosion, for example – introducing further positive feedback into the system: high grazing leading to low plant biomass, leading to poorer growing conditions, leading to lower plant biomass, leading to even poorer growing conditions, and so on (van de Koppel *et al.*, 1997).

On the other hand, there are many cases in which sudden changes in abundance are fairly accurate reflections of sudden changes in the environment or a food source. For instance, the number of herons nesting in England and Wales normally fluctuates around 4000–4500 pairs, but the population declines markedly after particularly severe winters (Figure 10.23c). This fish-eating bird is unable to find sufficient food when inland waters become frozen for long periods, but there is no suggestion that the lower population levels (2000–3000 pairs) are an alternative equilibrium. The population crashes are simply the result of density-independent mortality from which the herons rapidly recover.

10.7 Beyond predator–prey

The simplest mathematical models of predator–prey interactions produce coupled oscillations that are highly unstable. However, by adding various elements of realism to these models it is possible to reveal the features of real predator–prey relationships that are likely to contribute to their stability. A further insight provided by models is that predator–prey systems may exist in more than one stable state. We have seen that a variety of patterns in the abundance of predators and prey, both in nature and in the laboratory, are consistent with the conclusions derived from models. Unfortunately, we are rarely in a position to apply specific explanations to particular sets of data, because the critical experiments and observations to test the models have rarely been made. Natural populations are affected not just by their predators or their prey, but also by many other environmental factors that serve to 'muddy the waters' when direct comparisons are made with simple models.

Moreover, the attention of both modelers and data gatherers (not that the two need be different) is increasingly being directed away from single- or two-species systems, towards those in which three species interact. For example, a pathogen attacking a predator that attacks a prey, or a parasitoid and a pathogen both attacking a prey/host. Interestingly, in several of these systems, unexpected dynamical properties emerge that are not just the expected blend of the component two-species interactions (Begon *et al.*, 1996; Holt, 1997). We return to the problems of 'abundance' in a broader context in Chapter 14.

Summary

Predator and prey populations display a variety of dynamic patterns. It is a major task for ecologists to account for the differences from one example to the next.

A number of mathematical models illustrate an underlying tendency for predator and prey populations to undergo coupled oscillations (cycles) in abundance. We explain the Lotka–Volterra model, which is the simplest differential equation predator–prey model, and using zero isoclines we show that the coupled oscillations are structurally unstable in this case. The model also illustrates the role of delayed density-dependent numerical responses in generating the cycles. We explain, too, the Nicholson–Bailey host–parasitoid model, which also displays unstable oscillations.

In both these models, cycles are several prey (host) generations in length, but other models of host–parasitoid (and host–pathogen) systems are able to generate coupled oscillations just one host generation in length.

We ask whether there is good evidence for predator–prey cycles in nature, focusing especially on a hare–lynx system and a moth attacked by two natural enemies. Even when predators or prey exhibit regular cycles in abundance, it is never easy to demonstrate that these are predator–prey cycles.

We begin an examination of the effects on dynamics of factors missing from the simplest models by looking at crowding. For predators, the most important expression of this is mutual interference. We look at the effects of crowding in the Lotka–Volterra model, including ratio-dependent predation: crowding stabilizes the dynamics, although this effect is strongest when the predators are least efficient. Essentially similar conclusions emerge from modifications of the Nicholson–Bailey model. There is, though, little direct evidence for these effects in nature.

The functional response describes the effect of prey abundance on predator consumption rate. The three types of functional response are explained, including the role of handling time in generating type 2 responses, and of variations in handling time and searching efficiency in generating type 3 responses. We explain the consequences for predator–prey dynamics of the different types of functional responses and of the 'Allee effect' (lowered recruitment at low abundance). Type 2 responses tend to destabilize, and type 3 responses to stabilize, but these are not necessarily important in practice.

Predators often (but not always) exhibit an aggregative response. We examine the effects of refuges and partial refuges in the Lotka–Volterra model, suggesting that spatial heterogeneities, and the responses to them, stabilize predator–prey dynamics, often at low prey densities. However, further work, especially with host–parasitoid systems and the Nicholson–Bailey model, shows that the effects of heterogeneity are complex. Stability arises through 'aggregation of risk', strengthening direct density dependencies that already exist. But aggregative responses that are spatially density dependent are least likely to

lead to aggregation of risk and least likely to enhance stability. Models with within-generation movement further undermine the significance of aggregative responses in stabilizing host–parasitoid interactions. A metapopulation perspective emphasizes that patch differences may stabilize through asynchrony, and also that predator–prey interactions may generate spatial as well as temporal patterns.

In practice, the stabilizing effects of metapopulation structure and of refuges have been demonstrated, and the general importance of responses to spatial heterogeneity in the choice of biocontrol agents has been the subject of lively debate.

Finally, predator–prey systems with more than one equilibrium combination of predators and prey are examined as a possible basis for prey (or predator) outbreaks.