POPULATION DYNAMICS 7

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7.1 INTRODUCTION

The reasons for studying the population dynamics of insect natural enemies are basically twofold. Firstly, predators and parasitoids are an important component of terrestrial communities (LaSalle and Gauld, 1994), so therefore are of central interest to the ecologist who attempts to unravel the complexity of factors driving the dynamics of species interactions. Secondly, the knowledge gained from studies of predator and parasitoid populations may be of immense practical value in insect pest management (Hassell, 1978, 2000b; DeBach and Rosen, 1991; Van Driesche and Bellows, 1996).

In this chapter, we aim to demonstrate how ecologists and biological control researchers can assess the role of natural enemies in insect population dynamics, and how the information obtained (together with that gained from other biological studies) can be put to use in terms of biological control practice. We begin by reviewing methods for demonstrating and quantifying predation and parasitism (section 7.2). We then examine the different techniques for determining the effects of natural enemies on insect population dynamics (section 7.3). Finally, we examine ways in which this and other information can be used in choosing appropriate biological control agents for introduction (section 7.4). The reader should note that we make no attempt at providing a comprehensive review of insect natural enemy population dynamics – that would require a book in itself (a very large one at that!). Thus, there are several topics to which we make only brief reference, or do not mention at all. In most cases, the reasons for omission are simply either that the topic has received adequate coverage in other texts, or that it is not specifically related to insect natural enemy population dynamics. Readers mainly concerned with the theoretical background to parasitoid and predator population dynamics should also consult the books by Hassell (1978, 2000), Hawkins and Cornell (1999) and Hochberg and Ives (2000).

7.2 DEMONSTRATING AND QUANTIFYING PREDATION AND PARASITISM

7.2.1 INTRODUCTION

In this section we are concerned with a variety of techniques that can be applied to field, and in some cases laboratory, populations of natural enemies and their prey for the following purposes:

1. To demonstrate that natural enemies have a significant impact upon host and prey populations (subsections 7.2.3 to 7.2.9);
2. To measure rates of predation and parasitism in the field and/or the laboratory, and to provide indices of predation and parasitism (subsections 7.2.10 to 7.2.15).

The techniques used for 1. and 2. provide respectively: (a) a preliminary assessment of the impact of parasitoids and predators upon host and prey populations, and (b) quantitative information which can be used to further our understanding of the role of natural enemies in insect population dynamics. In the latter case, additional methodologies are required to achieve the objective; these are discussed in detail in section 7.3.

We begin by discussing the introduction of natural enemies in classical biological control. Strictly speaking, this is not a technique *per se* for demonstrating that natural enemies have a significant impact on host/prey populations. However, we include it because: (a) it can provide dramatic evidence of the impact of natural enemies upon insect populations, and (b) it can be simulated in the laboratory.

7.2.2 NATURAL ENEMY INTRODUCTIONS IN CLASSICAL BIOLOGICAL CONTROL

Some of the best demonstrations of the effectiveness of natural enemies are provided by cases of so-called **'classical biological control'**. Classical biological control may be defined as the importation of a natural enemy into a geographic region to control an insect species. Typically, the latter is an exotic species that has become established without its adapted natural enemy complex, and because the local natural enemies are ineffective, it has become a pest. For a classical biological control programme to be completely successful, the natural enemy has to reduce the pest populations to a level where the latter no longer inflict economic damage. In ecological terms, the natural enemy is required both to depress the pest population below a certain level and to prevent it from again reaching that level by promoting stability (Figure 7.1 and Figure 7.2b) (for a dissenting view regarding the necessity for stability in natural enemy-pest population interactions, see Murdoch *et al.*, 1985; Kidd and Jervis, 1997; and section 7.4). Successful biological control resulting from parasitoid introduction can be simulated in the laboratory, as shown in Figure 7.2b (also Figure 7.3).

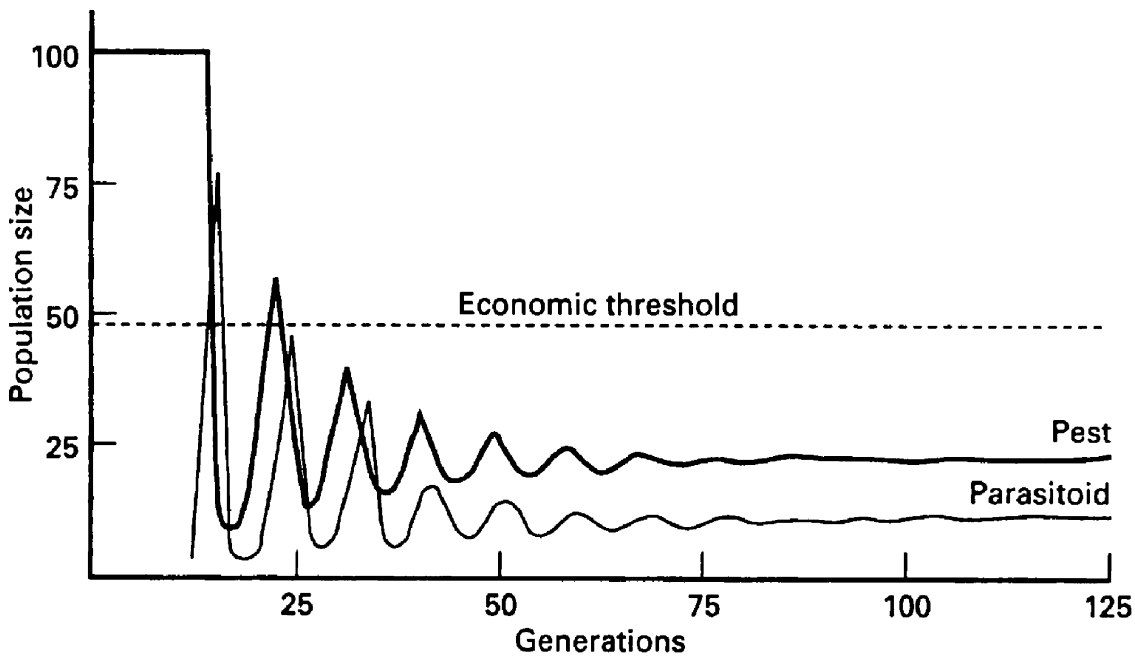

Figure 7.1 Hypothetical example of successful control of a pest population by an introduced parasitoid or predator. In this case, the pest population in the first generation is at an outbreak level (100), well above the economic threshold. The parasitoid or predator is introduced after 10 generations. As its numbers increase, pest numbers decrease. Initial oscillations in both populations decrease with time, and a stable equilibrium is attained at which the pest population remains suppressed to well below the economic threshold. Source: Greathead and Waage (1983), reproduced by permission of The World Bank.

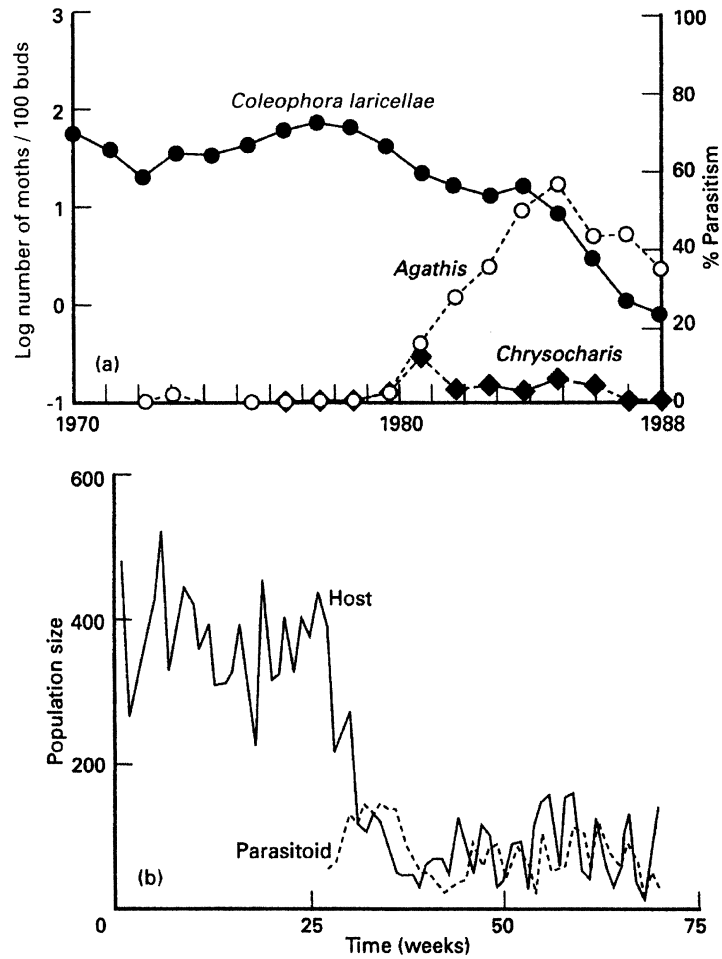

Figure 7.2 Examples, one from the field, the other from the laboratory, of the successful biological control of an insect pest following introduction of parasitoids: (a) Two parasitoid species, *Agathis pumila* (Braconidae) and *Chrysocharis laricinella* (Eulophidae), were introduced into Oregon, U.S.A., against the larch case-bearer, *Coleophora laricellae* (Lepidoptera). The figure shows the combined data for 13 plots over 18 years (source: Ryan, 1990). Reproduced by permission of the Entomological Society of America. (b) The pteromalid *Anisopteromalus calandrae* was introduced into a laboratory culture of the bruchid beetle *Callosobruchus chinesis* 26 weeks after the beetle culture was started (source: May and Hassell, 1988). Reproduced by permission of the Royal Society.

Classical biological control has served as a paradigm for the role of predators and parasitoids in insect herbivore population dynamics. Natural enemy introductions have been viewed as ecological experiments on a grand scale, allowing comparison, under field conditions, of the population dynamics of insect species in the presence and absence of natural enemies (Strong *et al.*, 1984). Many biocontrol practitioners (e.g. DeBach and Rosen, 1991; Waage and Hassell, 1982) consider there to be no fundamental difference between the successes achieved using exotic natural enemies in classical biological control and the action of indigenous species ('natural control' *sensu* Solomon, 1949, and DeBach, 1964). Classical biological control is thus seen as simply isolating a process that is taking place around us all the time. Using

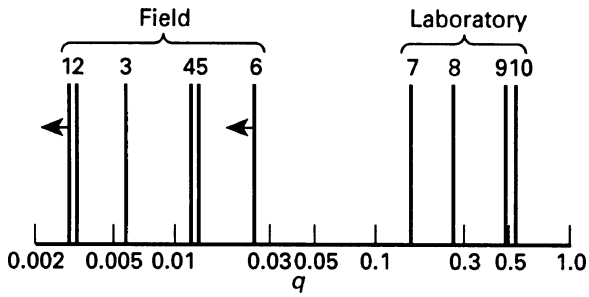

Figure 7.3 The degree to which a pest population may be suppressed by an introduced parasitoid, in six field and four laboratory parasitoid-host systems. The degree of depression in each case is expressed as a q-value, q being defined as the average abundance of the host in the presence of the parasitoid (i.e. post-introduction: N^*) divided by the host's average abundance in the absence of the parasitoid (i.e. pre-introduction: K). Arrows imply minimum estimates of the degree of depression. Note that doubts have been cast upon the role of *Cyzenis albicans* (5) in the direct control of the pest (the winter moth, in Canada) (subsection 7.3.4). Source: Beddington *et al.* (1978) (see that paper for the identity of all the species involved), reproduced by permission of Macmillan Magazines Ltd.

a database analysis of the results of key factor analyses (see subsection 7.3.4) and of historical data from natural enemy introductions, Hawkins *et al.* (1999) showed that classical biological control is, in fact, not strictly a 'natural' phenomenon, because it: (a) overestimates the extent to which parasitoids exert top-down control (subsection 7.3.10) on insect populations, and (b) results most often from the formation of a single, strong link in simplified food webs, in contrast to the 'natural control' that results from multiple links in more complex webs. With these caveats in mind, studies of classical biological control introductions can nevertheless shed considerable light on 'natural control' by predators and parasitoids.

The degree to which a host population may be reduced in abundance by an introduced parasitoid was examined by Beddington *et al.* (1978), who used a simple measure, $q = N^*/K$, where N^* is the average abundance of the host in the presence of the parasitoid (i.e. post-introduction) and K is the average abundance of the host prior to introduction of the parasitoid. Beddington *et al.* calculated q-values for six different field parasitoid-host systems (cases of successful biological control) and four laboratory systems. Figure 7.3 shows the calculated q-values to be of the order of 0.01; that is, the host populations were depressed to about one hundredth of their former abundance. [With the arrowhead scale (not included in Beddington *et al*,'s analysis), abundance was reduced by one hundredth but then rose again, settling at under one sixtieth. (Itioka *et al.*, 1997).] Note that the degree of depression of pest abundance required for successful biological control will vary from case to case, because economic thresholds are determined not only by pest density, but also by pest impact, crop value and other factors (Figure 7.4) (Waage

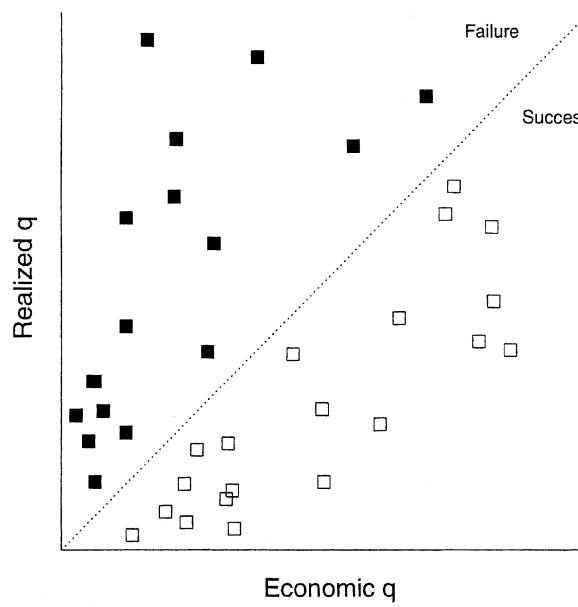

Figure 7.4 Hypothetical plot of outcomes of biological control programmes in terms of realised level of pest suppression and the threshold level required for the programme to be an economic success. q refers to the Beddington *et al.* (1975) index of equilibrium pest density after a classical biological control introduction, divided by equlibrium pest density prior to introduction (see text and Figure 7.3). From Hochberg and Holt (1999). Reproduced by permission of Cambridge University Press.

and Mills, 1992; Hochberg and Holt, 1999, see also section 7.4).

In the past, the value of detailed and precise quantitative data on the effects of natural enemy introductions on pest populations was not fully appreciated (Waage and Greathead, 1988; May and Hassell, 1988), with the result that often only anecdotal evidence on the effects of introductions has been available for some programmes (section 7.4). Notable exceptions are the introduction of the tachinid parasitoid *Cyzenis albicans*, introduced to control the winter moth in Canada (Embree, 1966), the release of parasitoid wasps against the larch casebearer in the USA (Ryan, 1990, 1997) (Figure 7.2a), the release of *Encarsia partenopea* against the whitefly *Siphoninus phillyreae* in the USA (Gould *et al*., 1992a,b) (Figure 7.5), and the release of two parasitoid wasps against the arrowhead scale (*Unaspis yanonensis*) in Japan (Itioka *et al*., 1997).

Another criticism that can be aimed at some programmes is that depression of the pest population cannot necessarily be attributed to the introduced natural enemy. That is, introduction and depression may be merely coincidental. For example, in programmes involving whitefly pests, reductions in pest density following parasitoid release were reported but the workers concerned failed to provide proper controls to demonstrate that the introduced natural enemies were indeed responsible for the depression (Gould *et al*., 1992a). Even where experimental controls are employed in biological control programmes, it is likely that, due to the rapid spread of the natural enemy, comparisons of test and control plots are possible for a brief period only. This problem arose with the monitoring of releases of *Encarsia partenopea* against the whitefly *Siphoninus phillyreae* in California (Figure 7.5). Parasitoids were released in May, and by midsummer had appeared at all control sites (4–11 km away from the nearest release sites) (Gould *et al*., 1992a). A problem of this type could be difficult to overcome, since too wide a separation of control and test sites reduces the validity of comparisons.

Nowadays there is an increasing awareness of the need for detailed and precise quantitative data on the effects of introductions, and classical biological control programmes are tending to be much more carefully documented through the routine collection of population data. However, this usually applies only to biological control programmes with good funding and well-trained staff. Another constraint upon the gathering of pre- and post-release population data is the great rapidity with which many pest problems arise. Examples of insects having very rapidly become serious pests are the mealybug *Rastrococcus invadens* in west Africa, and the psyllid *Heteropsylla cubana* in the Pacific region, Asia and elsewhere.

Traditionally, most quantitative studies measure population densities over a number of seasons before and after release (section 7.3). Nowadays, however, there is an awareness of the usefulness of undertaking detailed studies of within-season changes in population density (Gould *et al*., 1992b; Itioka *et al*., 1997) and also of recording changes in pest age-structure immediately following parasitoid introduction (Gould *et al*., 1992a, see Figure 7.5).

At present, there is no standard protocol (at least not a sufficiently detailed one; see Neuenschwander and Gutierrez, 1989; Waterhouse, 1991; Van Driesche and Bellows, 1996) for the quantification of the impact of natural enemies in classical biological control programmes. Such a protocol, if developed, would probably be restrictive, given the diversity in ecology that exists among insect pests.

7.2.3 EXCLUSION OF NATURAL ENEMIES

Exclusion methods have been widely employed in assessing the impact of insect natural enemies on host and prey populations under field conditions. The principle behind their use is that prey populations in plots (any habitat unit, from part of a plant to a whole plant or a group of plants) from which natural enemies have been eradicated and subsequently excluded will, compared with populations in plots to which natural enemies are allowed access: (a) suffer lower predator-induced mortality or parasitism, and (b) if the experiment is continued for a long

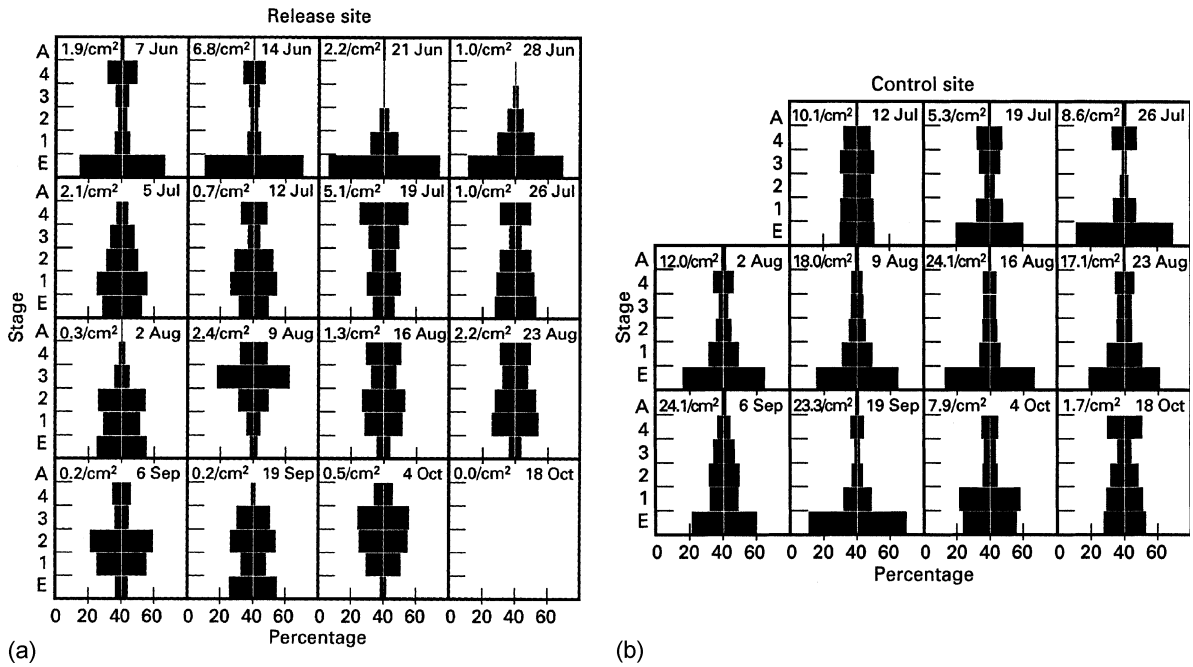

Figure 7.5 An example of classical biological control where post-introduction changes in pest age-structure (histograms) were monitored. The parasitoid wasp *Encarsia partenopea* (Aphelinidae) was introduced into California as an attempt to control the whitefly *Siphoninus phillyreae*. The whitefly, a pest of ornamental shrubs and fruit trees, was first recorded in the USA in 1988, and since then spread rapidly within California and into neighbouring states . Gould *et al*. (1992a) used several study sites, which were divided into release (test) and non-release (control) sites. In the release sites, parasitoids were released in large numbers over a period of several weeks, commencing 10th May. In all sites, densities of the pest's immature stages (eggs, nymphs) and adult stages were monitored, while in the release sites parasitism by *E. partenopea* was also recorded. Densities of the pest (numbers/cm^2, in the top left-hand corner of each graph) remained at low levels at the release sites, whereas at the control sites they were increasing by the beginning of summer. Shown here are changes in age-structure for (a) one release site and (b) one control site. After the parasitoid became abundant at a site (the parasitoid eventually dispersed to, and became established in, control sites), the pest population contained a decreasing proportion of young stages, as result of the parasitoid killing (through parasitism) fourth instars, so reducing recruitment of eggs to the whitefly population. Observe that in (a) the decline in the proportion of immature stages was more marked, and occurred much earlier than in (b). The initial increase in density of whiteflies in (a) is attributable to oviposition by female whiteflies that were already present at the time of parasitoid release. Source: Gould *et al*. (1992a). Reproduced by permission of Blackwell Publishing.

enough period, increase more rapidly and reach higher levels. The results of some exclusion experiments are shown in Figure 7.6.

Usually, the starting densities of prey are made equal in both the test and the control plots (for consistency's sake, we refer here to the exclusion plots as 'test' plots, and the non-exclusion plots as 'control' plots, since what is being tested is the effect of excluding natural enemies, not of including them; note that not all authors use the same nomenclature). Exclusion experiments may be conducted for periods of several days to several weeks. A long experimental period will be required if test-control differences in prey equilibrium densities (section 7.3) are to be compared.

Various exclusion devices have been employed; they include mesh cages placed over individual plants or groups of plants (Figure 7.7), mesh cages in the form of sleeves

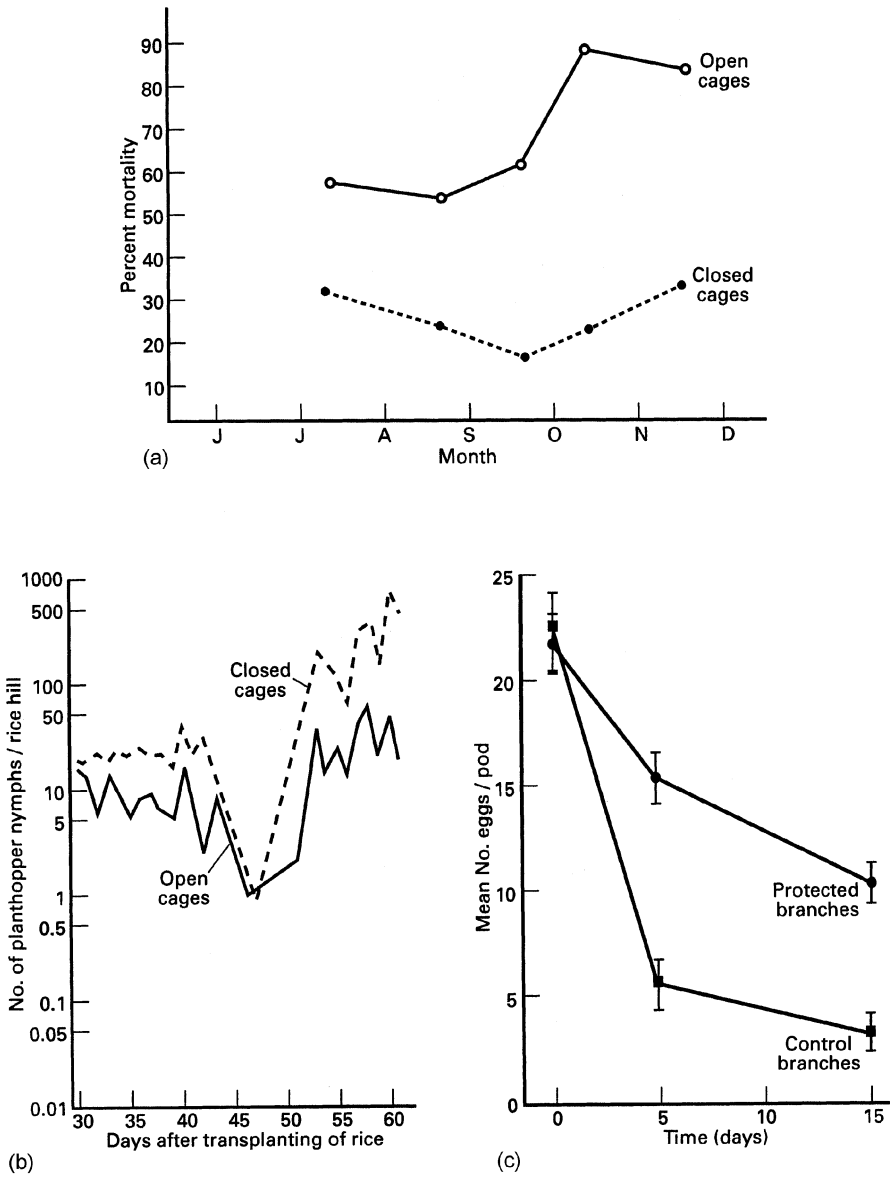

Figure 7.6 Effects of excluding predators from prey: (a) percentage mortality of California red scale (*Aonidiella aurantii*) on *Hedera helix*, in open clip cages that permitted the entry of parasitoids, and in closed cages that excluded the parasitoids (source: DeBach and Huffaker, 1971). (b) Population changes in the rice brown planthopper (*Nilaparvata lugens*) in closed and open field cages after all the arthropods had been removed and replaced with 25 first-instar planthopper nymphs per rice plant. Exclusion was not perfect: large numbers of predators were recorded in the 'closed' cages towards the end of the experiment, so the experiment ought to be considered as an 'interference' experiment (source: Kenmore *et al.*, 1985). (c) The mean number of bruchid beetle eggs per pod of *Acacia farnesiana* on protected and unprotected branches at days 0, 5 and 15 of the experiment. Protection of the branches was achieved by wrapping a 10 cm-wide band of tape around the base, and applying a sticky resin to the tape (source: Traveset, 1990). (a) reproduced by permission of Plenum Publishing Corporation, (b) by permission of The Malaysia Plant Protection Society, (c) by permission of Blackwell Publishing.

Figure 7.7 Exclusion cages in use in a rice paddy in Indonesia. Reproduced by kind permission of Anja Steenkiste.

placed over branches or leaves, clip cages attached to leaves, greased plastic bands tied around tree branches and trunks, and vertical barriers (walls constructed of polythene, wood or hardboard) around plants. For details of construction, consult the references cited below. The precise type of device used will depend upon the natural enemies being investigated, and whether the aim is either to exclude all natural enemies (so-called **total exclusion)** or to exclude particular species or groups of species (so-called **partial exclusion)**. For example, a terylene mesh/gauze cage placed over a plant ought, if the mesh size is sufficiently small, to exclude all aerial and surface-dwelling insect natural enemies, from the largest to the smallest. By increasing the size slightly, small parasitoid wasps may be allowed in, while increasing the mesh size further will allow larger types natural enemy to enter also, and so on. By first examining the ability of tiny *Anagrus* wasps (Mymaridae) to pass through terylene meshes of different mesh sizes, colleagues at Cardiff were able to decide on the appropriate size of mesh for excluding all natural enemies of rice brown planthopper other than the egg parasitoids (Mymaridae and Trichogrammatidae) whose impact was being investigated (Claridge *et al*., 2002). By having a cage with its sides raised slightly above the ground, predators such as carabid beetles and ants may be allowed access to insect prey such as aphids on cereals, whereas adult hover-flies and many types of parasitoid will be denied access. Conversely, a trench or a wall may prevent access to prey by ground-dwelling predators but allow access by aerial predators and parasitoids.

The exclusion devices can be placed around or over already existing populations of prey, in which case the density of the prey at the start of the experiment will need to be recorded. Preferably, prey-free individual plants, plant parts or plots of several plants (any prey already present are cleared by hand removal or by using low-persistence insecticides) can be loaded with set numbers of prey. The latter approach has the advantage that equivalent starting densities of prey/hosts in test and control plots can be more easily ensured, and also any parasitoid immature stages present within hosts can be eliminated from within the test plots. It may also be necessary to employ a systemic insecticide

when eradicating prey such as leafhoppers or planthoppers from a plot, in order that any prey eggs present within plant tissues are killed; of course, loading with prey cannot take place until one can be sure that the plant is free of the insecticide.

In some cases, simply comparing prey densities on caged plants with those on uncaged plants may produce misleading results, because:

1. Prey within the cages may be protected to some extent from the mortality or other deleterious effects of weather factors such as as rainfall or wind;
2. In the two treatments microclimatic conditions may be very different. Cages, even ones constructed largely of nylon or terylene mesh, may alter the microenvironment (light intensity, humidity, wind speed, temperature) surrounding the plant (Hand and Keaster, 1967) to such a degree as to influence the impact of natural enemies, either: (a) directly by affecting the physiology, the behaviour and consequently the searching efficiency of the predators or parasitoids, or (b) indirectly by affecting the behaviour, e.g. spatial distribution, and physiology, e.g. rate of development, longevity, fecundity, of the prey. Changes in prey behaviour and physiology can be brought about by microclimate-induced alterations in plant physiology.

In order to determine whether microclimatic effects on prey are likely to confound the results of an exclusion experiment, the effects of caging on prey population parameters such as fecundity and survival should be investigated. Frazer *et al*. (1981b), for example, investigated whether the observed increase in densities of pea aphid (*Acyrthosiphon pisum*) in exclusion cages (to as much as five times the levels recorded in uncaged plots) was due to an effect of caging upon aphid fecundity. No significant differences in the fecundity of aphids were found between caged and uncaged insects. Furthermore, a simulation model (subsection 7.3.8) showed that for a change in fecundity alone to be responsible for the difference in densities of prey between test and control plots, fecundity would have to have been three times the maximum rate ever observed.

In order to separate the effects of microclimate and natural enemy exclusion upon prey populations, exclusion devices that are either: (a) as similar as possible in construction, or (b) very different in construction but which nevertheless provide similar microclimatic conditions in their interiors, may have to be employed in both test and control treatments, with the obvious proviso that predators need to be allowed adequate access to prey in the control treatment. For example, in assessing the impact of the egg parasitoids *Anagrus* spp. (Mymaridae) and *Oligosita* (Trichogrammatidae) upon planthopper populations, exclusion cages can be constructed that have a very small mesh size to prevent such tiny parasitoids from entering (see above), while almost identical cages with a slightly larger mesh size can be constructed to allow the parasitoids to enter but prevent entry of larger types of natural enemy (Fowler, 1988; Claridge *et al*., 2002) (see above). In assessing the impact of parasitoids on insect herbivores on trees, gauze sleeve cages can be used on tree branches, the test cages being tied at both ends to exclude parasitoids, and the control cages being left open at both ends to allow parasitoids to enter (DeBach and Huffaker, 1971). However, insects such as hover-flies are deterred from ovipositing on branches in open-ended sleeves (Way and Banks, 1968). Way and Banks used rather dissimilar test and control cages in controlling for the effects of microclimate. The test cages had walls of terylene mesh, whereas the control cages had walls of wooden slats. Despite the major difference in construction, microclimate was similar in the two cage types.

One solution to the problem of achieving a closely similar cage design in the different treatments is not to bother providing natural enemies with access routes to the interior of the control cages, but to carry out an **exclusion/inclusion experiment**. Such an experiment would involve the use of identical cages in the two treatments and the caging of

a known number of predators and parasitoids with prey/hosts in the 'control' treatment (Lingren *et al*., 1968). This type of experiment has the added advantage that the densities of natural enemies will be more precisely known. *Per capita* predation and parasitism rates can be calculated (Dennis and Wratten, 1991) and, provided the densities used reflect those normally recorded in the field (this includes taking account of aggregative responses; Dennis and Wratten, 1991), useful estimates of *per population* rates of predation and parasitism can be obtained. A major disadvantage of exclusion/inclusion experiments is that in the control cages the dispersal of natural enemies is likely to be severely restricted or prevented. Long-distance approach behaviour of foraging predators and parasitoids to prey and hosts e.g. in response to kairomones (subsection 1.5.1), may also be interfered with;

3. If the prey are mobile, both immigration and emigration of prey/hosts may be different between the test and the control treatments (restricted or prevented altogether in the test treatment, normal in the control). In order to rule out the possibility that aphid population numbers in fully-caged cereal plots were augmented as a result of emigrant alatae re-infesting the plots, Chambers *et al*. (1983) removed all alate (winged) aphids that settled on the insides of some of the test cages whilst allowing the aphids to remain in another. Removal of alatae was found not to alter the pattern of population change in the cages. Therefore, re-infestation of shoots inside cages was unlikely to have been a cause of the cage/open plot differences in population numbers observed by Chambers *et al*. in their study (Figure 7.8a).

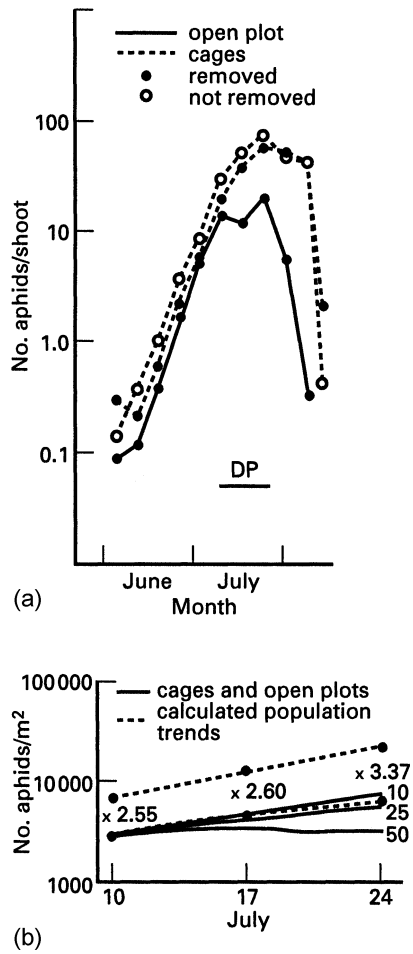

Figure 7.8 (a) Total numbers of cereal aphids in cages where alate adult aphids were removed from the inner walls and roof, in cages where they were not removed, and in the adjacent open plot. DP denotes the period of divergence between treatments. (b) Aphid populations in cages and open plots, and calculated population trends for different *per capita* predation rates. Also shown is the difference, expressed as a multiple, between populations in cages (containing aphids but not predators) and in open plots during the DP. Note log scales. Source: Chambers *et al*. (1983). Reproduced by permission of Blackwell Publishing.

Exclusion methods have a number of other important potential limitations:

4. Even where the microclimate is the same in the different treatments, it may be so different from ambient conditions that prey/host populations are severely affected, and any results obtained bear little relation to natural processes. The effects of caging upon microclimate can be assessed using instrumentation of various kinds (see Unwin and

Corbet, 1991, for a review). If caging is found to affect microclimate significantly, then it may be possible to provide some means of ventilation, e.g. an electrical fan, to maintain ambient temperature and humidity. Effects on light intensity may be minimised by choosing the appropriate type of screening material;

5. Whilst it is often possible to establish whether a particular **guild** (a group of species attacking the same host or prey stage/stages) of natural enemies has a significant impact upon prey populations, it may not be possible to determine which particular species of that guild is mainly responsible for the effect. Unless direct observations shed light on which species is responsible, information will be required on the relative abundance of different natural enemy species within a locality. Where the immature stages of parasitoids can be identified to genera or species, dissections of hosts (subsection 6.2.9) in the control plots may allow determination of the parasitoid species that usually contributes most to parasitism and help show whether an increase in host numbers in the test plots is due to the exclusion of that species. The problem of attribution of predatory or parasitic impact is a minor one where the natural enemy complex is known to comprise only one or two species in a locality;
6. If, in the test plots, prey numbers (e.g. of aphids) increase, they may do so to such an extent that predator species (e.g. coccinellids, hover-flies) other than the ones that are excluded (e.g. carabid beetles) are attracted preferentially into the test plots, i.e. through an aggregative response (subsection 1.14.7) by the predator or parasitoid. The impact of the excluded natural enemy species may thus be underestimated. This limitation also applies to the use of barriers and trenches, where the enclosed plants are exposed to invasion by a variety of aerial predators;
7. Whilst exclusion methods can reveal that natural enemies have a significant impact upon prey populations, other methods generally need to be applied before the predator-prey interaction can be properly quantified. The results need to be related to the density of predators present in the habitat, if realistic estimates of predation rates are to be obtained. Exclusion experiments provide minimal information, if any, on the *dynamics* of the predator-prey or parasitoid-host interaction, a limitation that applies also to several of the methods described below. This problem can be at least partly overcome by the construction of paired life-tables for the insects in test and control plots (Van Driesche and Bellows, 1996; Itioka *et al*., 1997).
8. One hundred per cent exclusion of natural enemies is sometimes difficult to achieve, with the result that zero predation or parasitism in test plots is not recorded (e.g. see Kenmore *et al*., 1985). Either during or at the end of an exclusion experiment, it is important to check for the presence of natural enemies in the test plots (see caption to Figure 7.6b), and to count the numbers of any such insects that have succeeded in gaining entry to the latter. Exclusion methods employing devices that are far from 100% effective in excluding natural enemies are, strictly speaking, interference methods (see below).

Other serious problems that may be encountered by experimenters include: (a) plants outgrowing their cages; expanding cages can be devised to counter this problem (Nicholls and Bérubé, 1965); (b) plants in test cages deteriorating very rapidly due to the abnormally high prey densities reached; little can be done to remedy this problem, which can severely limit the duration of the experiment.

Exclusion methods have been used to assess the impact of predators and parasitoids on populations of a wide variety of prey and host insects including: (a) *aphids* (Way and Banks, 1968; Campbell, 1978; Edwards *et al*., 1979; Aveling, 1981; Frazer *et al*., 1981b; Chambers *et al*., 1983; Carroll and Hoyt, 1984; de Clercq, 1985; Kring *et al*., 1985; Hance, 1986; Dennis and Wratten, 1991; Hopper *et al*., 1995;

Bishop and Bristow, 2001); (b) *mealybugs* (Neuenschwander and Herren, 1988; Boavida *et al*., 1995), (c) *armoured scale insects* (DeBach and Huffaker, 1971; Itioka *et al*., 1997); (d) *soft scale insects* (Smith and DeBach, 1942; Bishop and Bristow, 2001); (e) *planthoppers* (Kenmore *et al*., 1985; Rubia and Shepard, 1987; Fowler, 1988; Rubia *et al*., 1990; Claridge *et al*., 2002); (f) *pond-skaters* (water-striders) (Spence, 1986); (g) *aquatic stoneflies and chironomids* (Lancaster *et al*., 1991); (h) *beetles* (Sotherton, 1982; Sotherton *et al*., 1985; Traveset, 1990); (i) *flies* (Burn, 1982); (j) *moths* (Sparks *et al*., 1966; Lingren *et al*., 1968; van den Bosch *et al*., 1969; Irwin *et al*., 1974; Rubia and Shepard, 1987; Steward *et al*., 1988; Rubia *et al*., 1990); (k) *butterflies* (Ashby, 1974).

The results of exclusion experiments can be quite dramatic. For example, the numbers of brown planthopper nymphs on rice plants in test cages reached twelve times the level attained in control cages, even though exclusion of predators proved to be imperfect (Kenmore *et al*., 1985; Figure 7.6b). In Campbell's (1978) study of the hop aphid *(Phorodon humuli)*, aphid numbers reached around $1000/0.1\,m^2$ in test cages, whereas in uncaged control plots they declined virtually to zero. In exclusion/inclusion experiments carried out by Lingren *et al*. (1968), adult bollworm moths were introduced into test and control cages, and in the control cages different types of predator were subsequently introduced. The number of moth eggs in the test cages reached a level ten times higher than that recorded in the control cages containing the predators *Geocoris punctipes* (Lygaeidae) and *Chrysoperla* spp. (Chrysopidae).

Even where a marked difference in prey numbers is observed between test and control treatments, and the possible confounding effects of factors other than predation can be discounted, it is important to establish whether the predators in question really do have the potential to produce the test versus control plot difference observed. This requirement was appreciated by Chambers *et al*. (1983). As well as testing for the effects of aphid emigration, parasitism, fungal disease and cage microclimate, they sought to determine whether the *per capita* daily predation rates of aphid-specific predators were sufficiently high to have accounted for the differences in aphid numbers they recorded between fully-caged and open plots (Figure 7.8a and above). Using information on: (a) aphid rate of increase in the absence of predators (i.e. data were obtained from aphids in the cages); (b) predator densities in the open (control) plots; and (c) *per capita* daily predation rates of predators (published values), Chambers *et al*. were able to calculate population trends for aphid populations exposed to predation (Figure 7.8b, see Chambers *et al*. for method of calculation). They established that the predation rate that would be required to bring about the observed cage/open plot difference lay within published values.

7.2.4 INSECTICIDAL INTERFERENCE

The phenomenon of pest resurgence brought about by the application of insecticides, and inadvertent elimination of a pest's natural enemies reveals dramatically the impact the latter normally have (DeBach and Rosen, 1991; Shepard and Ooi, 1991). With this effect in mind, insecticides have been used as a method of assessing the effectiveness of natural enemies.

With the insecticidal interference method, the test plots are treated with an insecticide, so as to eliminate the natural enemies, and the control plots are untreated. The insecticide used is either a selective one, or a broad-spectrum one that is applied in such a way as to be selective, affecting only the natural enemies. Depending on the duration of the experiment, repeated applications of the insecticide may be required, to prevent immigrating natural enemies from exerting an impact upon prey in the test plots. Drift of insecticides onto control plots also needs to be carefully avoided. The results of some insecticidal interference experiments are shown in Figure 7.9.

Some limitations of the method are that:

1. In the test plots not only the natural enemies but also the prey may be affected by the insecticides, so confounding the results of the experiment. The numbers of prey may

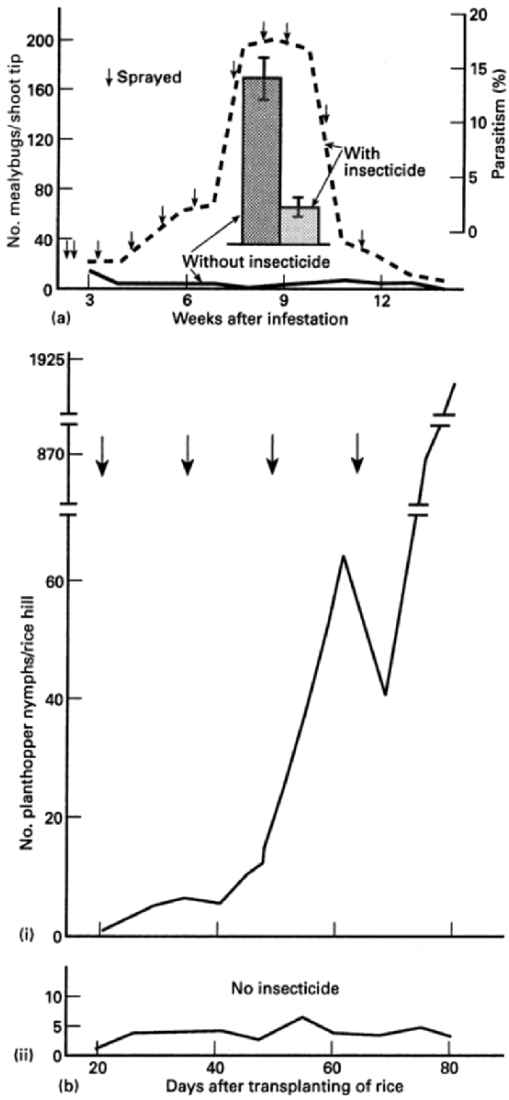

Figure 7.9 (a) Cassava mealybug (*Phenacoccus manihoti*) population development in insecticide-treated and untreated plots, together with mean levels of parasitism (histograms) (source: Neuenschwander and Herren, 1988). (b) Population changes in rice brown planthopper (*Nilaparvata lugens*) in: (i) a plot treated with four sprays (arrowed) of the insecticide decamethrin, and (ii) an untreated plot. Planthoppers were sampled from 40 rice hills per plot, using a vacuum net (subsection 6.2.2) (source: Heinrichs *et al.*, 1982). (a) reproduced by permission of The Royal Society, (b) by permission of The Entomological Society of America.

be inadvertently reduced due to the toxic effects of the insecticide (i.e. either the insecticide turns out not to be selective in action, or drift of a broad-spectrum insecticide has occurred) or they may be inadvertently increased due to some stimulatory, sublethal, effect of the insecticide upon prey reproduction (e.g. prey fecundity may be increased). Insecticides can be tested in the laboratory for their possible sublethal effects upon prey reproduction (Meuke *et al.*, 1978; Kenmore *et al.*, 1985).

2. In the test plots, 100% elimination of natural enemies is often not achieved (e.g. see Kfir, 2002), and so the full potential of natural enemies to reduce prey numbers is underestimated;
3. Limited information is provided on the dynamics of the predator-prey interaction, even where densities of natural enemies are known (see **Exclusion Methods**).

The main advantages of the method are that the possibly confounding effects of microclimate can be ruled out, and very large experimental plots can be used.

As an alternative to blanket spraying of test plots, an **insecticide trap method** can be used. Ropes of plaited straw treated with insecticide, trenches dug in the soil and containing formalin solution or insecticide-soaked straw, or some other insecticide-impregnated barrier, can severely reduce the numbers of natural enemies entering test plots. One treatment used by Wright *et al.* (1960) and Coaker (1965) in studying beetle predators of the cabbage root-fly *Delia radicum*, involved the placing of insecticide-soaked straw ropes along the perimeters of test plots. Whilst it was not 100% efficient, the latter treatment had a dramatic effect upon predator numbers, and also significantly affected prey numbers in test plots.

The insecticide interference method has been used to assess the impact of parasitoids and predators upon populations of: (a) *aphids* (Bartlett, 1968); (b) *armoured scale insects* (DeBach, 1946; 1955; Huffaker *et al.*, 1962; Huffaker and Kennett, 1966); (c) *leafhoppers and planthoppers*

(Kenmore *et al*., 1985; Ooi, 1986); (d) *flies* (Wright *et al*., 1960; Coaker, 1968); *moths* (Ehler *et al*., 1973; Eveleens *et al*., 1973; Kfir, 2002); (e) *thrips* (Nagai, 1990) and (f) *spider mites* (Plaut, 1965; Readshaw, 1973; Braun *et al*., 1989).

7.2.5 PHYSICAL REMOVAL OF NATURAL ENEMIES

As its name suggests, physical removal involves just that: predators are removed either by hand or with a hand-operated device, each day, from test plots. The method is a variant of exclusion, described above. Large, relatively slow-moving predators can simply be picked off plants by hand, while small, very active predators and parasitoids can be removed using an aspirator. This method has advantages in that microclimatic confounding effects can be ruled out (since cages are not used), and the contribution of particular natural enemy species to parasitism and predation can be relatively easily assessed. However, the method also has disadvantages in that:

1. Removal of natural enemies is very labour-intensive; for the method to provide more than just a crude measure of natural enemy effectiveness, a 24 hour per day watch needs to be kept on plants, and several observers need to be involved in removing insects;
2. Removal of natural enemies may involve disturbance to prey and thereby increase prey emigration;
3. Predators and parasitoids, before they are detected and removed, may have the opportunity to kill or parasitise hosts;
4. Like exclusion, the method provides limited information on the dynamics of the predator-prey interaction, even where densities of natural enemies are known (see **Exclusion Methods)**.

Hand removal has been used to evaluate the effectiveness of aphid predators (Way and Banks, 1968 (Figure 7.10); Pollard, 1971). Luck *et al*. (1988) suggest it can be used as a calibration method for interference and exclusion methods.

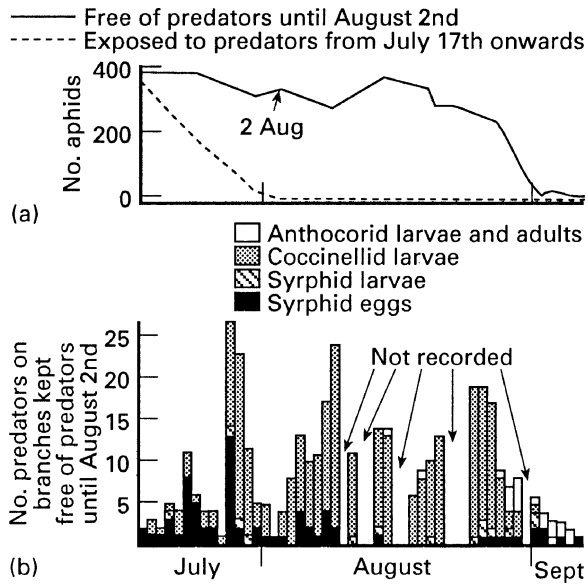

Figure 7.10 Effect of hand-removal of predators from colonies of *Aphis fabae*: (a) numbers of aphids on six branches of *Euonymus europaeus* kept free of predators until August 2nd, compared with numbers of aphids on branches exposed to predators from July 17th onwards; (b) numbers of different predators found on, and subsequently removed from, branches up to l 2nd August, and numbers that were found on, and allowed to remain, after 2nd August. In this experiment, crawling predators were excluded from the branches by a grease band. Source: Way and Banks (1968), reproduced by permission of The Association of Applied Biologists.

7.2.6 BIOLOGICAL 'CHECK' METHOD

This interference method exploits the fact that honeydew-feeding ant species, when foraging for honeydew sources and tending homopteran prey, interfere with non-ant predators and parasitoids, either causing them to disperse or killing them. In one set of plots, ants are allowed to forage over plants, whereas they are excluded from the other set. Natural enemies have access to both types of plot, but they are subject to interference by ants in the former. The method can

be used with prey that do not produce honeydew, provided either natural or artificial honeydew is made available to the ants. This method has several of the disadvantages of other interference methods and exclusion methods.

7.2.7 EGRESS BOUNDARIES

Egress boundaries are simple devices which allow predators to move out of, but not into, plots and thereby reduce predator numbers (see caption to Figure 7.11). These devices were used by Wratten and Pearson (1982) in assessing the effectiveness of predators of sugar beet aphids. Predator numbers within the plots were monitored using pitfall traps (subsection 6.2.1). A many-fold difference in aphid numbers was eventually recorded between test and control plots (40 aphids per plant and no more than 0.3 aphids per plant, respectively).

With egress boundaries (and ingress boundaries, see subsection 7.2.8 below), it is difficult to attribute differences in prey mortality between test and control plots to particular densities of predators, as the densities vary continuously over time. Therefore, it is difficult to calculate predation rates.

7.2.8 PREDATOR ENRICHMENT

With this method, the numbers of predators in the test plots are artificially boosted whereas the numbers in the control plots are not. Predator numbers in the test plots are enhanced by means of **ingress boundaries**, devices which allow predators to move into but not out of the plots. This method was employed by Wright *et al.* (1960) and Coaker (1965) in assessing the effectiveness of predatory beetles attacking cabbage root-fly (Figure 7.11), and by Wratten and Pearson (1982) in assessing the effectiveness of various predators of sugar beet aphids. Wratten and Pearson found that, by using their ingress boundaries, total numbers of harvestmen (Opiliones) captured (by pitfall-trapping) in test plots were 45% higher than in control plots, whereas the numbers of staphylinids, coccinellids and lycosids were increased by a maximum of 14%. However, the numbers of aphids eventually recorded in the test and control plots did not differ greatly.

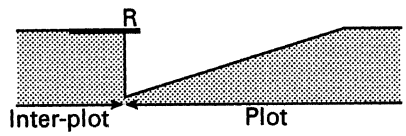

Figure 7.11 Cross-section through boundary used to allow predators to move into, but not out of, experimental plots (i.e. ***ingress* boundary**), in a study of predation of cabbage root-fly (*Delia radicum*). R = roofing felt. The device can be easily converted into an ***egress* boundary** if the roofing felt is suspended from the plot margin, and the sloping part of the trench is on the margin of the interplot. Source: Wright *et al.* (1960), by permission of The Association of Applied Biologists.

7.2.9 PREDATION AND PARASITISM OF PLACED-OUT PREY

With this method, known densities of prey are placed out in the field for a set period of time, and the numbers of dead or parasitised individuals recorded. The main conditions applying to the method are that prey ought to be placed out in as natural a fashion as possible, using natural densities, locations and spatial arrangements, so that they are neither more nor less susceptible to predation than usual. Also, an alteration in the overall density of prey in the field habitat (and therefore a perturbation to the system) ought to be avoided by having the artificially placed prey replace an equivalent number of prey simultaneously removed from the habitat. To enable the prey to be identified at the end of the experimental period, they may need to be either marked in some way or, if they are mobile, tethered. The marking or tethering technique ought not to affect (increase or decrease) the acceptability of prey to predators. Burn (1982) placed out eggs (stained with Bengal Rose) of the carrot fly (*Psila rosae*) to measure predation by beetles, and Weseloh (1990) placed out larvae (tethered with long thread) of the gypsy moth (*Lymantria dispar*) to measure predation by a complex of predators. Burn

(1982) determined beforehand whether staining of eggs affected the readiness of predators to eat treated eggs. Weseloh (1990), using a type of cage that allowed ants to enter but prevented moth larvae from escaping, compared the degree of predation recorded for tethered larvae with that recorded for untethered larvae. He found the tethered larvae to be more susceptible to predation by ants than untethered ones, and so he used a correction factor to apply to the mortality rates he recorded for tethered larvae placed out in open sites.

Ôtake (1967, 1970) devised a method, involving the use of artificially infested plants containing eggs of known age, to measure field parasitism of planthopper eggs by *Anagrus* (Mymaridae) wasps. The plants were exposed in the field for a set time period, and were then returned to the laboratory and dissected to determine the numbers of parasitised and unparasitised eggs. This **'trap plant' method'** was used by Fowler *et al*. (1991) and Claridge *et al*. (1999) to investigate various aspects of egg parasitism of rice-associated planthoppers and leafhoppers.

Provided the conditions set out above are satisfied, or some correction for bias in results can be applied, this method can provide useful data on the effectiveness of natural enemies. Weseloh (1990) concluded that the estimates of daily *per population* predation rates that he obtained by placing out tethered larvae were, if suitably corrected for bias, comparable with estimates obtained by other methods.

The main usefulness of the method, however, lies in providing comparative data, especially indices of predation and parasitism. For example, it can shed light on the relative effectiveness of different natural enemy species within a habitat, or on the effectiveness of a particular natural enemy species in different parts of a habitat (Fowler *et al*., 1991; Speight and Lawton, 1976). Speight and Lawton (1976) used the method to examine the influence of weed cover on predation by carabid beetles within a habitat. Their study is also interesting in that artificial prey, *Drosophila* pupae killed by deep-freezing, were used.

The term **'prey enrichment'** has been used to describe experiments involving the placing out of prey without the removal of existing prey.

Paired life-tables can be constructed for test (hosts or prey added) and control plots (Van Dreische and Bellows, 1996).

7.2.10 LABELLING OF PREY

With this method, prey are labelled with a dye, a radioactive isotope or a rare element (subsection 6.3.10 describes labelling methods) and released into the field to expose them to natural predation. After an appropriate period of time has elapsed, predators are collected from the field, screened for the label, and the amount of label present quantified. The *per capita* consumption rates of predators are calculated by measuring the label 'burdens' of the insects, and if the field density of predators is known, *per population* estimates of predation can also be estimated. For details, see McDaniel and Sterling (1979).

The technique has little to recommend it, in view of the following:

1. Radioactive labels can be hazardous to health;
2. It is difficult to ensure that all prey carry the same amount of label – there is normally considerable variation;
3. The same level of radioactivity can result from consumption of different numbers of prey;
4. The rate of excretion of the label from an individual predator appears to depend upon the quantity of food subsequently eaten;
5. Labelling can affect the susceptibility of prey to predation. Earwigs (*Forficula auricularia*), for example, prefer to feed on undyed as opposed to dyed eggs of the cinnabar moth (*Tyria jacobaeae*) (Hawkes, 1972);
6. The label can easily and rapidly spread through the insect community by various routes, including excretion, honeydew production, trophallaxis (i.e. by ants), moulting, scavenging on dead prey, and secondary predation;

7. The protocol can be very labour-intensive and, where rare elements and radioisotopes are used, specialised equipment is required;
8. Field populations of prey are disturbed.

Prey labelling has been used to quantify predation by natural enemies of aphids (Pendleton and Grundmann (1954), moths (Buschman *et al*., 1977; McDaniel and Sterling, 1979; Gravena and Sterling, 1983) and isopods (Paris and Sikora, 1967).

7.2.11 FIELD OBSERVATIONS

With this method, predation is quantified by making field observations, either directly or using video recording techniques, of predators *in situ* (subsections 6.2.6 and 6.3.2 describe methods). Kiritani *et al*. (1972) estimated the number (n) of rice leafhoppers killed by spiders per rice hill per day as follows:

$$n = FC/P \qquad (7.1)$$

where F is the number of predators seen feeding per rice hill during the observation period, C is the total amount of feeding activity in 24 hours expressed in terms of the specified period of observation, and P is the probability of observing predation (the average amount of time, in hours, taken to eat a prey individual, divided by 24 hours). A series of values of n were plotted against time and the area under the curve taken as the total number of prey killed. As noted by Southwood (1978), this method relies upon a high degree of accuracy in observing all instances of predation at a given moment and on values of C and the time taken to eat prey being fairly constant.

Edgar (1970) measured predation by wolf spiders (Lycosidae) in a similar manner to Kiritani *et al*. (1972), while Sunderland *et al*. (1986b) quantified predation of web-spinning money spiders (Linyphiidae) differently, as follows:

$$n = prk \qquad (7.2)$$

where n is the number of aphids killed/m^2/day, p is the proportion of ground covered by webs, r is the rate of aphid falling/m^2, and k is the proportion of aphids entering webs that are killed or die (determined from field observations and laboratory experiments). Using this approach, it was shown that aphid populations could be reduced by spider predation by up to approx. 40%.

Waage's (1983) work on foraging by ichneumonid parasitoids (subsection 6.2.6) shows direct observation to have considerable potential as a method for measuring rates of parasitism, at least for some medium– to large-bodied parasitoid wasp species.

As noted in subsection 6.3.3 the prey 'booty' collected by ants can be taken from the insects upon their return to the nest. A mechanical or photoelectric counter, as suggested by Sunderland (1988), or video recording equipment can enable predation rates to be calculated. The particular prey population being exploited by ants can easily be located by following the insects' trails, so that the prey's population density can be measured.

Video recording of predation and parasitism is likely to prove most fruitful if either the prey are relatively sedentary (e.g. some predators of aphids) or the predators are sedentary (e.g. ant-lion larvae, tiger-beetle larvae).

7.2.12 GUT DISSECTION

Gut dissection (subsection 6.3.8), is one of the simplest techniques for measuring ingestion and predation rates. Also, being a **'post-mortem method'** like serology and electrophoresis discussed below, it has the advantage over methods involving experimental manipulations of the predator-prey system that the results apply directly to an undisturbed, natural system (Sunderland, 1988).

The proportion of dissected predators containing remains of a particular prey in their guts can provide a crude index of *per population* predation rates. More meaningful measures can be obtained by counting the number prey remains, corresponding to prey individuals, present within the guts of predators (e.g. number of prey head capsules), and recording also the throughput time of prey in the gut.

Sunderland and Vickerman (1980) used gut dissection in evaluating the relative effectiveness of different predators of cereal aphids, by multiplying the proportion of such insects that contained aphid remains during the aphid population increase phase by the mean density of predators (ground examination samples) at this time. The species with the highest indices were considered to be the most valuable in constraining the build-up of aphids in cereal fields.

For other studies employing the technique, see Andow (1992) and Cook *et al*. (1994).

7.2.13 SEROLOGY

Field predation rates

The serological methods discussed in subsection 6.3.9 in relation to the determination of dietary range in field-collected predators have mainly been aimed at quantifying field predation. Various models are available (Table 7.1); these are based, to varying extents, upon the following variables: predator density, the proportion of predators testing positive for the prey, the detection period of the prey remains in the predator's gut (= the 'prey antigen half-life'), the prey biomass recovered, the mean proportion of the meal remaining, the *per capita* predation rate as measured in the laboratory or outdoor insectary, and the *per capita* predation rate measured as a function of prey density. For reviews of the models, see Sopp *et al*. (1992) and Naranjo and Hagler (2001).

Although shown by Sopp *et al*. (1992) to provide more accurate estimates of predation rate than its predecessors, their model (5 in Table 7.1) still involves several assumptions (some of which are common to other models) which, when violated, will reduce the accuracy of the predation rate estimates:

1. The detection periods measured in the laboratory are realistic estimates of those in the field;
2. The term ft_{DP} relates to the mean time since ingestion of prey materials for the population under study;
3. The predator takes discrete meals which are digested and voided before another meal is taken. If the meal comprises several prey individuals or, in the case of those predators with long detection periods, several meals are taken in rapid succession, they are regarded as a single meal, and so predation rate will be underestimated;
4. There is not a variable degree of partial prey consumption;

Table 7.1 Predation rate models employed with serological methods.

1. pd/t_{DP}	Dempster (1960, 1967)
2. pr_id	Rothschild (1966)
3. pr_id/t_{DP}	Kuperstein (1974, 1979)
4. $[\log_e (1 - p)]d/t_{DP}$	Nakamura and Nakamura (1977)
5. Q_od/ft_{DP}	Sopp *et al*. (1992)
6. $pdr_i(N)/t_{DP}(\theta)$	Naranjo and Hagler (2001)

r = *per population* field ingestion or predation rate (biomass or numbers of prey; to convert the former to the latter, the mean weight of individual prey in the field needs to be known).
r_i = *per capita* ingestion or predation rate measured in the laboratory or an outdoor insectary.
p = the proportion of field-collected predators found to contain prey remains.
d = predator population density.
t_{DP} = detection period for prey in the predator's gut (a function of temperature, see below)
Q_o = the quantity of prey recorded in the predator's gut (note that the immunodot assay technique (subsection 6.3.9) cannot be used to record this, see Greenstone, 1995).
f = the proportion of food remaining in the predator's gut.
N = prey density
θ = temperature

5. The predator density in the field is accurately known (this is rarely the case);
6. Both the predator sample and the amount of prey biomass present in predator guts are representative of the predator population as a whole. This is related to sample size and the sampling regime adopted;
7. There is no cross-reactivity between the prey species and non-target prey species (if cross-reactivity is a problem, it can be overcome by the use of monoclonal antibodies, subsection 6.3.9)
8. The presence of prey remains is the result of predation and not of scavenging, secondary predation or feeding on alternative prey.

The Naranjo and Hagler (2001) model (6 in Table 7.1) incorporates more biological realism by using the predators' functional response. Comparisons were made with the Dempster (1960) and the Nakamura and Nakamura (1977) models; these were found to either overestimate (model 1) or to underestimate (model 4), predation rates. Comparisons were not made with the Rothschild (1966), Kuperstein (1979) and Sopp *et al*. (1992) models. However, Naranjo and Hagler argue that the first two of these (models 2 and 3) would have greatly overestimated prey attack rates in their study system, and that the last (5, i.e. Sopp *et al*.'s) has limited applicability, due mainly to the problems inherent in using Q_0 (it remains to be seen whether Naranjo and Hagler are correct on this point).

It is important to know how the detection period (t_{DP}) can vary with: (a) meal size, (b) temperature, and (c) the presence of non-target prey items in the gut. Sopp and Sunderland (1989) demonstrated the effects of (a) and (b) on the detection period and antigen decay rate (the rate of disappearance of detectable food) in the beetles *Bembidion lampros* (Carabidae) and *Tachyporus hypnorum* (Staphylinidae) and the spider *Erigone atra* (Linyphiidae). Previously starved predators were fed freshly-killed aphids and were then kept at one of a range of temperatures for varying periods. The proportion of prey remaining in the gut at intervals after feeding was measured and plotted (Figure 7.12a). Curves were fitted to the (transformed) data (Figure 7.12b) and the detection period estimated (this is just one method of detection period measurement; Symondson and Liddell, 1993e, provide a review).

Sopp and Sunderland (1989) concluded the following from their study and other studies:

1. Usually, within a predator species, the detection period declines with increasing temperature; larger species tend to have longer detection periods, possibly because of the larger meal sizes, but within a species meal size appears to have little effect upon the detection period. Spiders have very long detection periods, even at high temperatures, perhaps because of their ability to store partially digested food in gut diverticula;
2. In most predators the antigen decay rate follows a negative exponential form; the majority of the detectable antigens disappear within one-third of the detection period. Agustí *et al*. (1999a) found that, whereas an exponential decay model was appropriate for predatory heteropteran bugs (*Dicyphus tamaninii*) that had eaten one *Helicoverpa armigera* egg, a linear model gave a better fit for individuals that had consumed ten eggs.

Symondson and Liddell (1993e) point out that the delectability of invertebrate remains in the crops of predators such as carabid beetles is influenced not only by the antigen decay rate but also the residence time of a meal in the crop and gizzard (i.e. the fore gut). If the rate of through-put of prey material happens to be less than the antigen decay rate, then there will be a discrepancy between the true proportion of prey material present and the amount estimated from an ELISA. Quantification of this discrepancy would provide a means of estimating original meal size, when the time since feeding can be estimated; such quantification requires crop weight loss and antigen decay rate to be measured as separate variables (for protocol, see Symondson and Liddell's paper).

Harwood *et al*. (2001b) have shown that it is possible to test whether secondary predation is

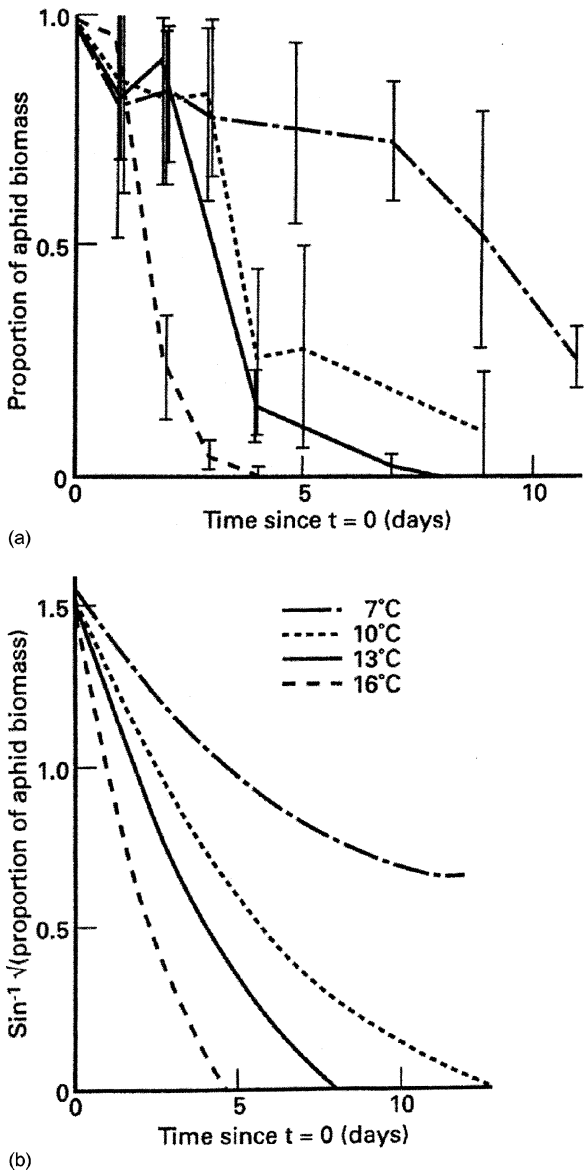

Figure 7.12 (a) The proportion of aphid biomass present in the gut, immediately after feeding, that is subsequently detected at various time intervals in the carabid beetle *Bembidion lampros*. (b) Antigen decay rate curve. Symondson and Liddell (1993e) expressed the antigen decay rate differently, and also took account of the loss in weight of the predator's crop in estimating meal size (see their paper for details). Source: Sopp and Sunderland (1989).

likely to be a significant confounding factor in a particular study, if a serological technique is applied in such a way as to maximise the possibility of such detection.

Predation Indices

Sunderland *et al*. (1987a) compared different polyphagous predators in terms of their probable value as cereal aphid predators, by calculating, for each predator species, the following index:

$$P_g d/D_{max} \qquad (7.3)$$

where P_g is the percentage of predators testing positive using ELISA, D_{max} is the maximum period over which prey antigens can be detected in any individual of a given species, and d is the mean predator density. Spiders tended to have the highest indices.

7.2.14 ELECTROPHORESIS

Electrophoresis, like serology, has been used in quantifying predation by fluid-feeding arthropod predators, albeit less commonly (for a review, see Solomon *et al*., 1996). As with serology and gut dissection, the proportion of predators testing positive for prey contents can easily be determined (subsection 6.3.11), but to obtain meaningful quantitative information on predation, the quantity and detection period of prey materials ingested also need to be known. We have little further to say about electrophoresis, as it has been superseded by ELISA. The latter is not only a far more sensitive method for determining the quantity of prey proteins in the guts of predators, but also it requires less time to test gut contents.

With the aforementioned indirect and post-mortem methods (electrophoresis, serology, gut dissection, labelling of prey) converting ingestion rate to predation rate can generate serious errors. For example, if scavenging occurs, the true predation rate will be overestimated. Sunderland (1996), in discussing sources of potential error in estimating predation rates, points out that the latter (predation being defined loosely) can also be *underestimated*, because predators may kill or

wound prey without ingestion occurring (e.g. 'wasteful killing' by satiated predators, see Johnson *et al.*, 1975). For a discussion of the multiplicity of factors that can lead to inaccurate estimates of predation rates, see Sunderland's (1996) review.

7.3 THE ROLE OF NATURAL ENEMIES IN INSECT POPULATION DYNAMICS

7.3.1 INTRODUCTION

Having reviewed some of the methods by which insect mortality due to natural enemies can be quantified, we now turn our attention to the more difficult task of assessing its dynamic significance. Mortality factors acting on an insect population can cause three possible dynamic changes. They can:

1. Affect the average population density;
2. Induce fluctuations in numbers;
3. Contribute to the regulation of population numbers.

Of the three, it is undoubtedly the contribution that natural enemies make to population regulation which has most occupied the minds of ecologists over the years.

Factors which regulate population numbers can act either by:

1. Returning populations towards a notional **equilibrium** number after some perturbation (i.e. **stabilising** population numbers);
2. Restricting population numbers within certain limits, but allowing fluctuations in numbers (e.g. cycles) within those limits (Murdoch and Walde, 1989).

For a factor such as parasitism or predation to regulate, the strength of its action must be dependent on the density of the population affected. That is, it needs to be **density-dependent**, its *proportional* effect being greater at high population densities and smaller at low densities (Figure 7.13; cf. **density-independent** factors). Density-dependence operates through **negative feedback** on population numbers, which may involve changes in the rates of reproduction, dispersal and immigration as well as changes in mortality. If the proportion of hosts parasitised varies with changing host density, either temporally or spatially (subsection 7.3.10), this can profoundly affect the dynamics of the interaction. As we shall see (subsections 7.3.4, 7.3.7), density-dependent factors can also affect average population levels and can, under certain conditions, induce perturbations too (subsection 7.3.4).

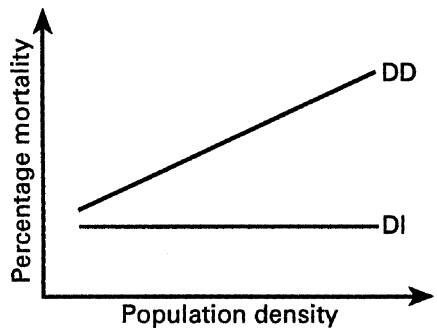

Figure 7.13 The negative feedback effect of a density-dependent mortality factor (DD) in which proportional mortality increases with population density (*cf.* density-independent factors (DI) in which proportional mortality is unrelated to population density).

We should make it clear at this stage that our discussion of population dynamics and population regulation is specifically aimed at issues and techniques relating to the actions of natural enemies. In recent years a number of issues which do not focus *directly* on natural enemies have received a great deal of attention in the literature, for example, the detection of density-dependence from time-series data (Godfray and Hassell, 1992; Holyoak, 1994; Rothery *et al.*, 1997; Hunter and Price, 1998; Turchin and Berryman, 2000; Berryman and Turchin, 2001), and the nature and significance of deterministic chaos (May, 1974a; Gleick, 1987; Berryman, 1991; Logan and Allen, 1992; Godfray and Grenfell, 1993; Hastings *et al.*, 1993; Cavalieri and Kocak, 1995; Desharnais *et al.*, 2001). We make no attempt to cover these important topics, but simply refer the reader to the references given above.

We begin by addressing the problems associated with using percentage parasitism estimates

to assess the impact of parasitoids on host populations (subsection 7.3.2). We then discuss what is perhaps the simplest (but least insightful) technique of assessing the impact of natural enemies, that of comparing their numbers with those of the prey or host populations (subsection 7.3.3). We then review the more conventional methods of life-table analysis (subsection 7.3.4) and show how simple population models can be derived from the information obtained. The limitations of the life-table approach are discussed, showing the need for supplementary field experiments (e.g. convergence and factorial experiments) (subsection 7.3.5). Next, we discuss how the important methodology of experimental component analysis can be applied using both analytical and simulation models (subsections 7.3.6, 7.3.7, 7.3.8, 7.3.9), and go on to examine some of the more contentious issues which have developed out of this approach (subsection 7.3.10).

7.3.2 THE PROBLEM OF 'PERCENT PARASITISM'

A point which is perhaps worth stressing at this stage is that the importance of natural enemies in host or prey population dynamics may have little to do with the degree of mortality which they cause *per se*, a fact which is often misunderstood by researchers in pest management. Many publications, for example, have reported high 'percent parasitism' in insect pest populations, the clear implication being that this mortality is likely to contribute, in a major way, to reducing average population levels and/or to regulating populations. Unfortunately, such inferences may not be justified, for reasons which will become apparent later on in this chapter.

'Percent parasitism' may also be a poor measure of the impact of parasitoids on host population dynamics for a number of other reasons. First, as Van Driesche (1983) pointed out, the number and timing of samples taken are usually inadequate for the task. To assess a parasitoid's contribution to host population mortality, it is the percentage attacked for the *generation* which must be determined and this may best be done within the context of a complete life-table study of the host population (subsection 7.3.4). Furthermore, percent parasitism does not take account of other forms of parasitoid-induced mortality, such as host-feeding (Jervis and Kidd, 1986; Jervis *et al.*, 1992a), which may sometimes outweigh parasitism in their contribution to host mortality. The degree of temporal synchrony of parasitoid and host population may also be an important factor in determining how well sampling estimates generational levels of parasitism. Using a series of simple theoretical models Van Driesche (1983) was able to establish that:

1. Where susceptible hosts are all present before parasitoids begin ovipositing, and the parasitoid oviposition period does not overlap with the start of parasitoid emergence (Figure 7.14a), then the peak percent parasitism sampled can give a good estimate of generational percent parasitism;
2. Where the situation in (1.) prevails, but hosts begin to develop to the next (unsusceptible) stage before all parasitoids have emerged, this will cause the peak percent parasitism to overestimate generational parasitism (Figure 7.14b);
3. Where the situation in (1.) prevails, but parasitoids begin to emerge before all parasitoid oviposition is complete, then peak percent parasitism will underestimate generational parasitism (Figure 7.14c);
4. If hosts enter the susceptible stage gradually and concurrently with parasitoid oviposition, and if host entry and exit do not overlap appreciably and parasitoid oviposition and emergence do not overlap appreciably (point X in Figure 7.14d), then a sample of percent parasitism at this point can accurately estimate generational parasitism;
5. If hosts enter the susceptible stage gradually and concurrently with parasitoid oviposition, but if both hosts and parasitoids enter and leave the system at rates other than in (4.), then samples of percent parasitism will bear little relation to generational percentage parasitism.

All of the above conclusions are based on the assumption that host mortality is caused solely

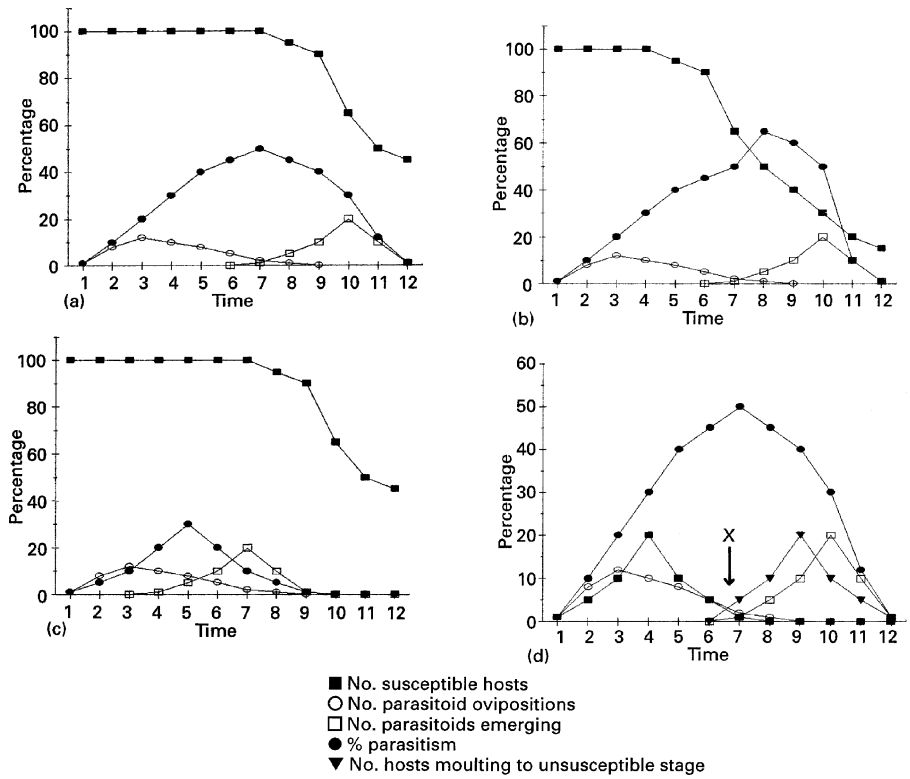

Figure 7.14 Synchrony of parasitoid and host populations may affect the accuracy of estimates of generational percent parasitism (see text for an explanation). Adapted from Van Driesche, 1983)

by parasitism. Where this restriction does not apply (possibly most cases!), correspondence between samples and generational parasitism levels will be even harder to determine. Also, if the sampling method is in any way selective towards either parasitised or unparasitised hosts (subsection 6.2.9), this will introduce a further error into the estimate (Van Driesche, 1983). Van Driesche *et al*. (1991) suggested some ways of circumventing the above problems. One is to measure recruitment to both the host and the parasitoid (parasitised hosts) populations continuously, total recruitment to both populations being found by summing the recruitment values for all intervals. The ratio of total parasitoid recruitment to total host recruitment provides an unbiased estimate of total losses to parasitism. Another method uses death rate measurements from field samples. If individuals are collected at frequent intervals, reared under field temperatures, and the proportion dying from each cause recorded from one sample to the next, then the original percentage of the sample that was parasitised can be estimated. Gould *et al*. (1990) and Buonaccorsi and Elkinton (1990) provide equations for the calculations. The method requires that all hosts have entered the susceptible stage before the first sample and that no host recruitment occurs during the sampling period. Details and examples of these and other techniques can be found in Van Driesche and Bellows (1988), Bellows *et al*., (1989a) Van Driesche *et al*. (1991) and Ruiz-Narvaez and Castro-Webb (2003). Ruiz-Narvaez and Castro-Webb (2003) devised a statistical method for estimating percentage parasitism when host and parasitoid phenologies are unknown.

7.3.3 CORRELATION METHODS

In field populations a useful preliminary indication of the impact of natural enemies can often be obtained by statistically correlating their numbers against those of their prey or hosts. Significant positive or negative correlations may imply some causative association, which can then be tested by further investigation. Correlation alone, of course, should not be taken as proof of causation. A high positive correlation may indicate a degree of prey specificity on the part of the predator (Kuno and Dyck, 1985), which might be expected to show a rapid numerical response to variations in prey density (subsection 7.3.7 gives a definition). Heong *et al*. (1991), for example, found that the numbers of heteropteran bugs and spiders, which are major predators of Homoptera Auchenorrhyncha in rice, correlated positively with the numbers of Delphacidae and Cicadellidae. A positive correlation would also be accentuated by a low predator attack rate and/or a prey species with a relatively slow rate of population growth (Figure 7.15a).

Negative correlations, on the other hand, may indicate a slow or delayed numerical response by the predator to changing prey density. These responses are commonly shown by highly polyphagous predators which may 'switch' to feeding on a prey type only after it has increased in relative abundance in the environment (section 1.11). Negative correlations are also more likely to be associated with prey species which tend to show rapid changes in abundance, or with predators having a high attack rate (Figure 7.15b). For example, negative correlations between aphids and coccinellid beetles are frequently found on lime trees during the summer, and can be explained by the rapid rate of increase in the aphid population in the spring, coupled with the slow rate of response by the coccinellids (Dixon and Barlow, 1979). Later in the season predator numbers increase, forcing the already declining aphid population to crash (Figure 7.16a). Syrphid predators, on the other hand, can show a rapid numerical response to increasing cereal aphid populations, producing a positive within-season correlation (Chambers and Adams, 1986) (Figure 7.16b).

The tentative conclusions afforded by correlation techniques should only be drawn with extreme caution, and then only with a detailed appreciation of the biologies of the species involved. In particular, it must be remembered that correlations can be created just as easily by predator populations *tracking* changes in prey numbers, as by *bringing about* those changes. Also, absence of any correlation should not be taken to imply that predators do not have any

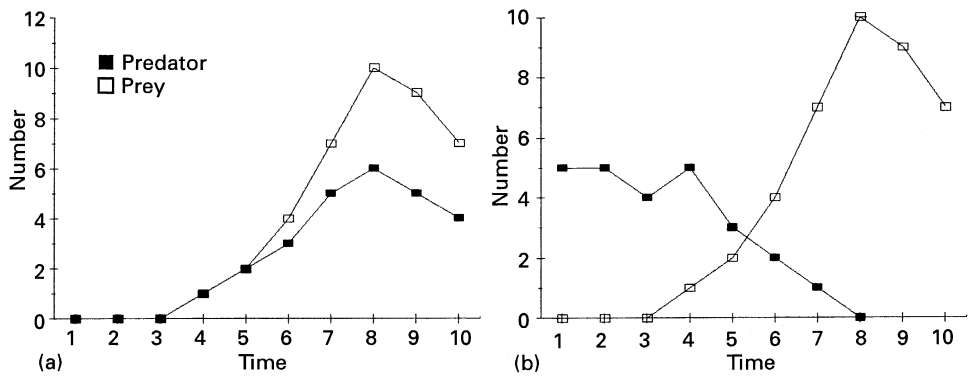

Figure 7.15 Relationships between predator and prey population numbers which produce either positive or negative correlations: (a) a positive correlation between predator and prey numbers produced by a slow rate of prey increase coupled with a relatively low predator attack rate, such that prey numbers are not reduced, while predator numbers are still rising; (b) a negative correlation between predator and prey numbers caused by predators depressing prey numbers, which only increase after predator numbers have declined.

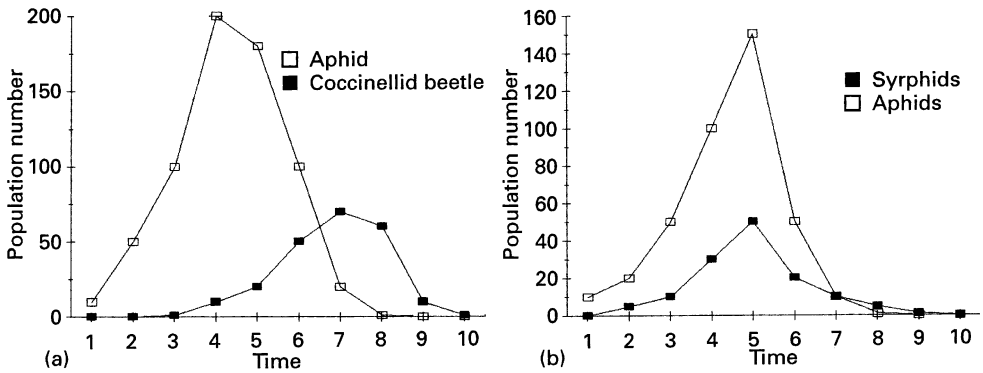

Figure 7.16 (a) A negative relationship between aphid and coccinellid beetle numbers on lime trees. Predator numbers increase slowly in response to aphid numbers and only reach their highest densities after aphid numbers have already declined; (Schematic representation based on information given in Barlow and Dixon, 1979). (b) Syrphids show a rapid numerical response to increasing cereal aphid numbers, declining as aphid numbers decline. This produces a positive correlation between predator and prey numbers. Schematic representation based on Chambers and Adams (1986).

impact. We might expect a lack of any correlation in cases where predators have features intermediate to the aforementioned extremes.

7.3.4 LIFE-TABLE ANALYSIS

Introduction

The concept of the life-table has already been introduced in section 2.11.2, in relation to the calculation of intrinsic rates of increase. Here, we are concerned with using life-tables of a somewhat different nature to determine how specific mortality factors (e.g. a particular natural enemy species) affect prey or host population dynamics. For example, is the mortality density-dependent or density-independent? Is there evidence for delayed or over-compensating density-dependence? In short, does mortality from this source tend to regulate numbers at, or disturb numbers from, a certain level? To answer these and related questions, we need to take life-tables apart and analyse the specific mortalities separately. Because some insect populations (e.g. aphids) tend to have generations which overlap in time, while others do not, two quite different approaches have been developed for each category, respectively the time-specific life-table and the age-specific life-table.

Age-specific Life-Tables

The life-table approach pioneered by Pearl and Parker (1921), Pearl and Miner (1935) and Deevey (1947) was extended to insects with discrete generations by the single-factor analysis of Morris (1959) and the **key factor analysis** of Varley and Gradwell (1960) (the latter sometimes incorrectly referred to as *k*-factor analysis). Of the two methods, the latter has been most widely used in population ecology (Podoler and Rogers, 1975) and will be the one concentrated upon here. For those readers interested in the Morris method, details are provided by Southwood and Henderson (2000). Varley and Gradwell's method is given a very detailed treatment suitable for the beginner in Varley *et al*. (1973). As the latter book is now, alas, out of print, we feel it is worthwhile discussing the procedures in detail, especially since there have been subsequent developments.

The usefulness of the Varley and Gradwell approach depends on the availability of sequential life tables for a number of generations of a univoltine population. In temperate regions, for example, it is commonly the case that insect populations overwinter as eggs and develop through a number of discrete stages in the spring and summer (Figure 7.17). The adults

then mature in the autumn to lay a new generation of overwintering eggs before dying. In this situation, generations remain completely separate. By obtaining population density estimates for the numbers entering each stage in the life cycle, it is then possible to construct a **composite life-table**, consisting of a sequence of independent life-tables for each generation (Table 7.2). The numbers entering each stage can be estimated in two different ways: (a) by direct assessment of recruitment (for example, by measuring fecundity or fertility, section 2.7), or (b) by indirect calculation from counts of stage densities. Several techniques are available which provide an estimate by the second route, and these are reviewed by Southwood and Henderson (2000). The graphical method of Southwood and Jepson (1962), for example, involves plotting the density of a stage against time and dividing the area under the plot by the average duration of the stage (mean development time). This yields an unbiased estimate of the number entering the stage if there is either no mortality, or the mortality occurs only at the end of the stage. Any mortality during the stage will result in underestimation. Bellows *et al*. (1989b) provide an extension to this method which can be used for interacting host and parasitoid populations. A number of other methods are discussed by Manly (1990).

It should be noted from Table 7.2 that the actual density estimates of numbers entering each stage are retained in the life-table, rather than corrected to a common starting number (*cf*. Table 2.2). The reason for this will become clear. Where stage mortalities can be partitioned into a number of definable causes, these are quantified separately in the table. In this way it may be possible to build similar life-tables for particular natural enemies. Varley *et al*. (1973) provide a number of rules to follow in the construction of the table. These are:

1. Where mortalities are reasonably well separated in time, they are treated as if they are entirely separated with no overlap;
2. Where events overlap significantly in time, they can be considered as if they are exactly contemporaneous;
3. All insects must be considered either as live and healthy or, alternatively, as dead or cer-

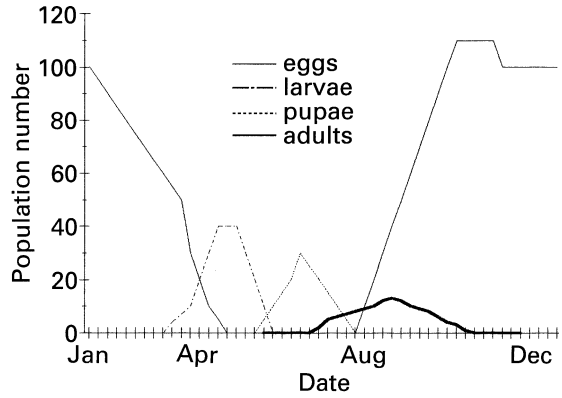

Figure 7.17 Schematic life-cycle of a typical temperate-zone univoltine insect population.

Table 7.2 Composite life-tables for six generations of a hypothetical insect population with discrete generations. Each *k*-value is calculated as $k = \log_{10}$ before mortality $-\log_{10}$ after mortality. $K = k_1 + k_2 + k_3$ (Note: whilst such life-tables have traditionally been presented in columns, putting them in rows (as is done here) makes spreadsheet regression calculations easier.)

Year	Eggs	k_1	Larvae	k_2	Pupae	k_3	Adults	K
1	1000	0.824	150	0.398	60	1.080	5	2.302
2	800	0.426	300	0.685	62	1.190	4	2.301
3	1200	0.681	250	0.455	50	0.824	12	1.960
4	700	0.942	50	0.204	50	0.699	10	1.845
5	500	0.553	140	0.301	70	0.766	12	1.620
6	1200	1.000	120	0.150	85	1.230	5	2.380

tain to die from some cause. For example, parasitised larvae are scored as certain to die, with the parasitoid recorded as the cause of death;

4. No insect can be killed more than once. Where hosts are attacked by two parasitoid species, death of the host is credited to the first parasitoid. If the second parasitoid emerges as the victor, it is taken as the cause of death of the first parasitoid. The second attack is thus entered in the life-table of the first parasitoid but not in that of the host.

Although somewhat arbitrary, rules such as these are necessary to balance the budget. However, as we explain below, conclusions from the analysis may unfortunately be sensitive to the rules adopted.

By converting the data in Table 7.2 to logarithms ($\log_{10}$), we can calculate for each successive mortality, in any generation:

$k = \log_{10}$ number before mortality - $\log_{10}$ number after mortality

where k is a measure of the proportion dying from the action of the mortality factor. In practice these calculations are easily carried out using a spreadsheet programme (Table 7.2 caption), which can also be used for the regression analyses (see below). Within each generation, we can thus determine a sequence of k-values, k_1, k_2, k_3, ... k_n, corresponding to each successively acting defined mortality up to the adult stage (Table 7.2). Strictly speaking, this should be up to the stage before reproduction begins, any pre-reproductive mortality being counted as separate k-factors. Mortality during the adult stage can be counted as one or more k-factors acting on the adults, or alternatively as a k-mortality acting on the next generation of eggs (Varley *et al.*, 1973). The final post-reproductive mortality to act on a generation, i.e. that which brings generation numbers to zero, contributes nothing to between-generation variation in numbers and is not included in the analysis. To do so would cause two problems. First, we are dealing here with the $\log_{10}$, of numbers, so how would we treat zero values? Second, the final reduction in adult numbers to zero, is by its nature density-dependent. In a sense, the ultimate extreme of regulation is to return a population to an equilibrium of zero! We illustrate the point by including this spurious density-dependence in our analysis (Figure 7.20). The sum of all the k-values up to, but not including this last mortality, provides us with a measure of total generation mortality K, i.e.

$$k_1 + k_2 + k_3 \ldots\ldots\ldots k_{n-1} = k \qquad (7.4)$$

The advantages of using k-values instead of percentage mortalities lie in the ease of calculation and the fact that k-values can be added to give a measure for total generation mortality (K) (adding percentages would have no meaning).

Two basic questions can be answered from an analysis of the table at this stage:

1. Which factor or factors contribute most to variations in mortality from generation to generation, i.e. the so-called **key factor(s)** causing population change?
2. Which factors contribute to regulation of population numbers?

Key Factors

The answer to the first question can often be obtained from a graphical representation of the data. Plotting the k-values against generation may be enough to reveal the key factor(s) causing population change (Figure 7.18). Here, variations in k_3 between generations most closely follow variations in overall mortality (K), indicating that k_3, is the key factor. Note that the key factor is not necessarily the factor causing greatest mortality (k_1 in this case).

Sometimes, a simple graphical inspection may not be enough to reveal the key factor, in which case the statistical method of Podoler and Rogers (1975) can be employed. This involves regressing each k-value against total generation mortality (K), the mortality with the greatest slope (b) being the key factor. In our example k_3 is confirmed as the only significant key factor (Figure 7.19). Where more than one factor is found to contribute, a hierarchy of significance can be constructed.

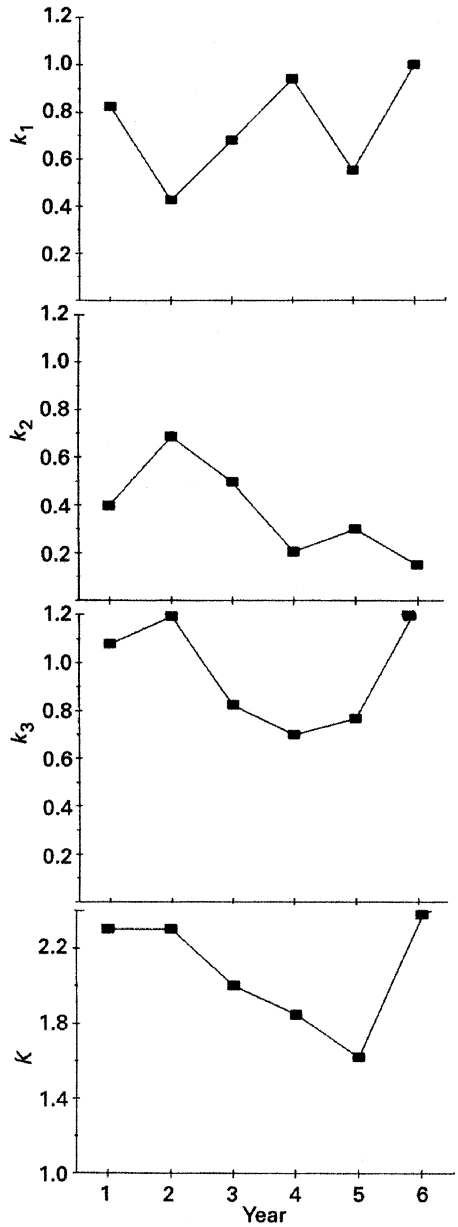

Figure 7.18 Key factor analysis of the mortalities acting on a hypothetical insect population (see Table 7.2 for data).

Strictly speaking, the Podoler and Rogers' procedure for identifying key factors is not statistically valid, in that it contravenes the basic rules of regression. These are that the axes should be independent of each other and the independent

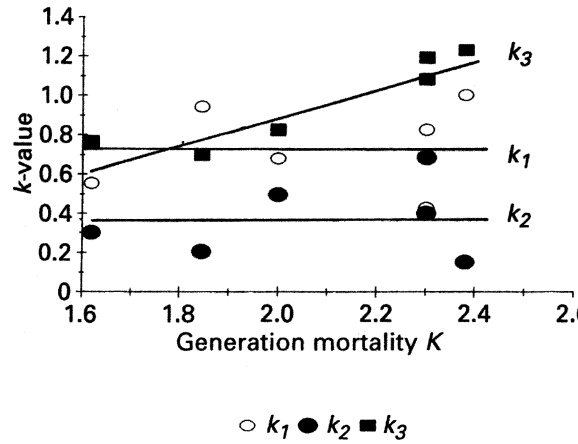

Figure 7.19 Podoler and Rogers' method for identifying key factors. The factor with the greatest slope (k_3 in this case) is the key factor causing population change ($k_3 = 0.68K - 0.46$; $R^2 = 0.84$).

variable should be error-free. Clearly, K consists of the k-values against which it is being regressed, and it is also subject to sampling error. Where the regression relationship of the putative key factor is not clear cut, a simpler expedient may be to use the correlation coefficients, which are not subject to the same restrictions. In this case, the key factor would be the one with the highest correlation between k and K, (maximum $r = 1$). Manly (1977) devised an alternative method based on multiple regression analysis, whilst the problems of sampling error have also been considered by Kuno (1971). As we shall now see, a similar problem with regression is confronted in the detection of density-dependence from life-table data.

Detecting Density-dependence

Assessing which factors contribute to regulation of the population again involves plotting each k-value, this time against the $\log_{10}$ density on which it acts (i.e. *before* the mortality). In our example (Figure 7.20) the plot of k_1 against log density of eggs contains six data points, corresponding to each generation. Similarly, k_2 is plotted against $\log_{10}$ density of new larvae, again with six data points, and so

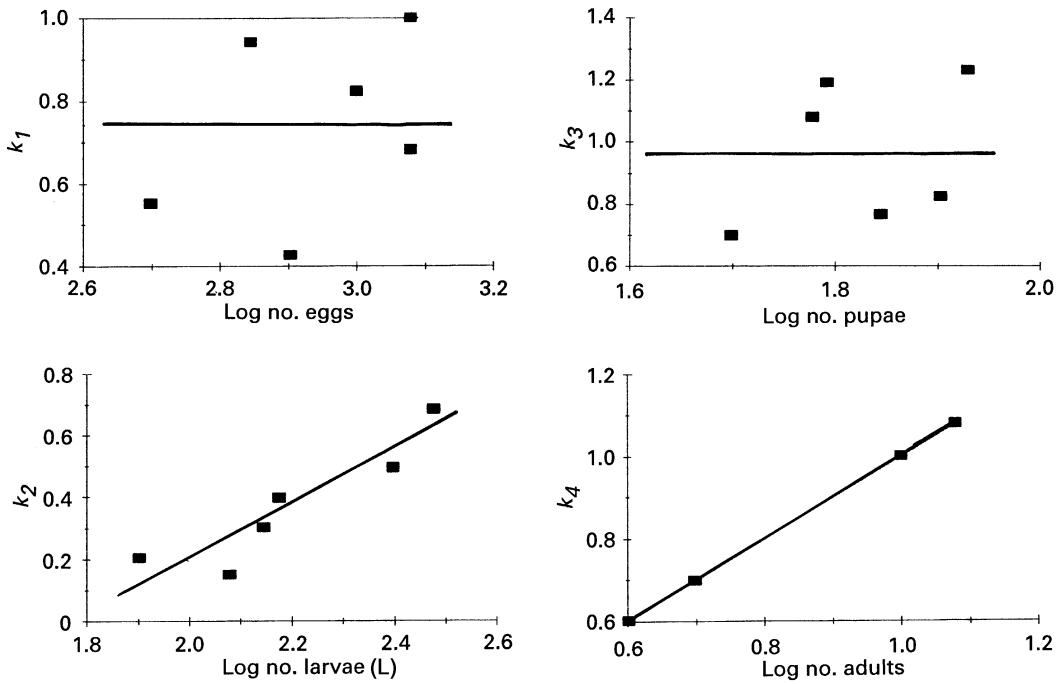

Figure 7.20 The identification of density-dependent factors from life table data. *k*-values for the different mortalities are plotted against the population densities on which they acted. In this case, only k_2 is significantly density-dependent. ($k_2 = 0.86$L–1.52; $R^2 = 0.84$; $k_1 = 0.74$; $k_3, = 0.96$.) k_4 is the last mortality to act, bringing numbers down to 0 (or in this case 1, which was used to make the log calculations workable). This mortality is, by its nature, always density-dependent (see text), but is not included in the analysis, as it contributes nothing to population variation or regulation.

on. Remembering that each *k*-value is a measure of *proportional* mortality; positive relationships for any of these plots would indicate that mortality is acting in a density-dependent fashion. A horizontal slope would indicate density-independence, while a negative slope would indicate inverse density-dependence. Regression analysis is generally employed to calculate the significance of the slopes. Here, the only significant density-dependence is found in k_2. However, the problem of statistical validity (mentioned above in relation to Podoler and Roger's method) again arises. As *k*-values are calculated in the first place from $\log_{10}$ densities, the two axes are not independent. Moreover, the independent variable ($\log_{10}$ density), estimated from population samples, is not error-free. To overcome the problem, Varley and Gradwell (1968) suggest a 'two-way regression' test, which involves both the regression of $\log_{10} N_t$ (initial density) on $\log_{10} N_{t+1}$ (final density) and $\log_{10} N_{t+1}$ on $\log_{10} N_t$. If both regressions yield slopes significantly different from $b = 1$ and are on the same side of the line, then the density-dependence can be taken as real. This method may be unnecessarily stringent (Hassell *et al.*, 1987; Southwood *et al.*, 1989), requiring that density-dependence remains apparent when all sampling errors are assumed to lie firstly in the estimates of N_t, then in N_{t+1}. Bartlett (1949) provided an alternative regression method in which sampling errors are distributed between both axes.

If density-dependence is accepted, then the regression coefficients can be taken as a measure of the *strength* of the density-dependence. The

closer b is to 1, the greater the stabilising effect of the mortality. A slope of $b = 1$ will compensate perfectly for any changes in density at this stage (**exact compensation**), while a slope of $b < 1$ will be unable to compensate completely for any changes **(undercompensation)**. Slopes of $b > 1$ imply **overcompensation**, the significance of which will become clear later.

A further insight into the nature of density-dependence can also be obtained by again plotting each k-value against the $\log_{10}$ density on which it acts, but in a **time sequence** (Varley and Gradwell, 1965; Figure 7.21). Different factors trace a different pattern depending on their mode of action; density-independent factors show an irregular, zigzag pattern (Figure 7.21a), while direct density-dependent factors show a more discernible straight-line pattern of points clustered within a narrow band (Figure 7.21b). A spiral pattern (Figure 7.21c) indicates delayed density-dependence, in which the action of the k-mortality is not felt until one or two generations hence. Insect parasitoids frequently act in this way for reasons which will be explained in subsection 7.3.7. Manly (1988) provided a statistical test for spiral patterns based on a comparison of the internal angles of the spiral.

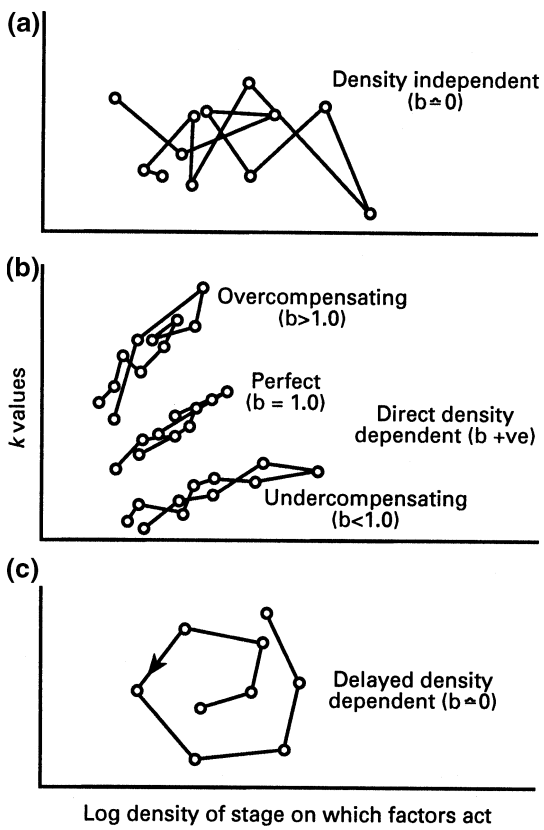

Figure 7.21 Time sequence plots showing how density relationships can be identified from the patterns produced. Source: Southwood (1978).

A Simple Inductive Model

At this point the 'formal' methodology associated with key factor analysis has been fully described, but further insights into how k-mortalities affect population dynamics can be derived from a simple **inductive model** constructed using the information obtained above (inductive models are those based on particular case studies, which yield general insights into population dynamics; philosophically, induction is the process of arguing from the particular case to the general case (*cf.* deduction, deductive models, subsection 7.3.7).

We begin by linking the numbers in each life stage to the next, through the mortalities expressed by $k_1, \ldots k_{n-1}$ as follows:

$$k_1 = m_1 - E_t + c_1 \tag{7.5}$$

$$L_t = E_t - k_1 \tag{7.6}$$

$$k_2 = m_2\ L_t + c_2 \tag{7.7}$$

$$P_t = L_t - k_2 \tag{7.8}$$

$$k_3 = m_3 P_t + c_3 \tag{7.9}$$

$$A_t = P_t - k_3 \tag{7.10}$$

where E_t, L_t, P_t and A_t are the $\log_{10}$ numbers of eggs, larvae, pupae and adults respectively at time t (m values are the regression constants for each equation, and c values are constants). Assuming a 50:50 sex ratio, we can find the $\log_{10}$ number of females (F) from:

$$10^{F_t} = 10^{A_t}/2 \tag{7.11}$$

or

$$F_t = A_t - 0.30103 \qquad (7.12)$$

In our example, $k_1 = 0.74$, $k_2 = 0.86L_t - 1.52$ and $k_3 = 0.96$. The number of eggs laid by adults can be estimated from either: (a) cohort fecundity experiments (performed in the laboratory [subsection 2.72] and/or in the field), (b) dissection of females and estimating potential fecundity (subsection 2.72), or (c) regression of eggs in year $t+1$ against the estimated number of females ($A/2$) in year t. Assuming the following relationship between female numbers and eggs deposited (Figure 7.22):

$$E_{t+1} = 0.86F_t + 2.1 \qquad (7.13)$$

We now have a series of equations which can be used sequentially to simulate dynamic changes from one generation to the next, over as many years as we require. Note that the **model** as it stands is completely **deterministic** in that it takes no account of the potential variation in the relationships, i.e. particular values for variables on the right hand side of the equations produce only one possible value for the variable on the left hand side. **Stochastic models**, on the other hand, *do* take account of the variability in the relationships, by including mathematical terms to describe chance events which may affect one or more of the relationships in the model. In this case, particular values for variables on the right hand side of the equations may produce a number of possible values for the variables on the left hand side. The methodology of stochastic modelling is discussed further in subsection 7.3.8, and a good introductory treatment can also be found in Shannon (1975).

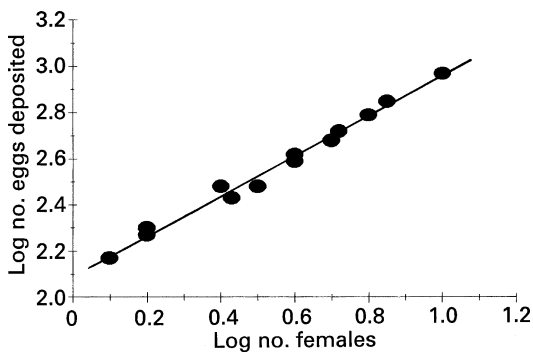

Figure 7.22 The relationship between female numbers and reproduction used in model 5.3.4. ($E = 0.86F + 2.1$; $R^2 = 0.99$).

Simulations of the model with different starting densities of eggs show that numbers approach an equilibrium within 2–3 generations, i.e. are strongly regulated (Figure 7.23a). Proof that regulation is provided by k_2 can be obtained by altering equation (7.8) such that k_2 becomes density independent ($k_2 = c_2$). Here, numbers either increase indefinitely or decrease to zero, depending on the other parameter values (Figure 7.23d), i.e. regulation is removed. Alternatively, increasing the strength of density-dependence by increasing the slope of the regression relationship between k_2 and L_t (e.g. $b = 1.2$ in equation (7.8), can produce oscillations of decreasing amplitude which eventually return to equilibrium (Figure 7.23b). Increasing the b-value even further (e.g. $b = 2.4$), however, can result in oscillations of increasing amplitude leading to the extinction of the population (Figure 7.23c). Thus, density-dependence is confirmed to be potentially either stabilising or destabilising in its effect, depending on its strength. It is also apparent that the weaker the density-dependence, the higher the equilibrium value becomes.

A Case Study: The Winter Moth

To appreciate the considerable number of studies on which key factor analysis has been performed, the reader is referred to Podoler and Rogers (1975), Dempster (1983), Price (1987), Stiling (1987, 1988) and Hawkins *et al.* (1999). There is no doubt, however, that it is Varley and Gradwell's own study (1968, 1970) of the winter moth *(Operophtera brumata)*, together with the various follow-up studies in England and Canada, which have made this perhaps the best understood and most widely-quoted example. It is worth reviewing briefly some of the features of this study, as it serves to illustrate some of the potential problems in

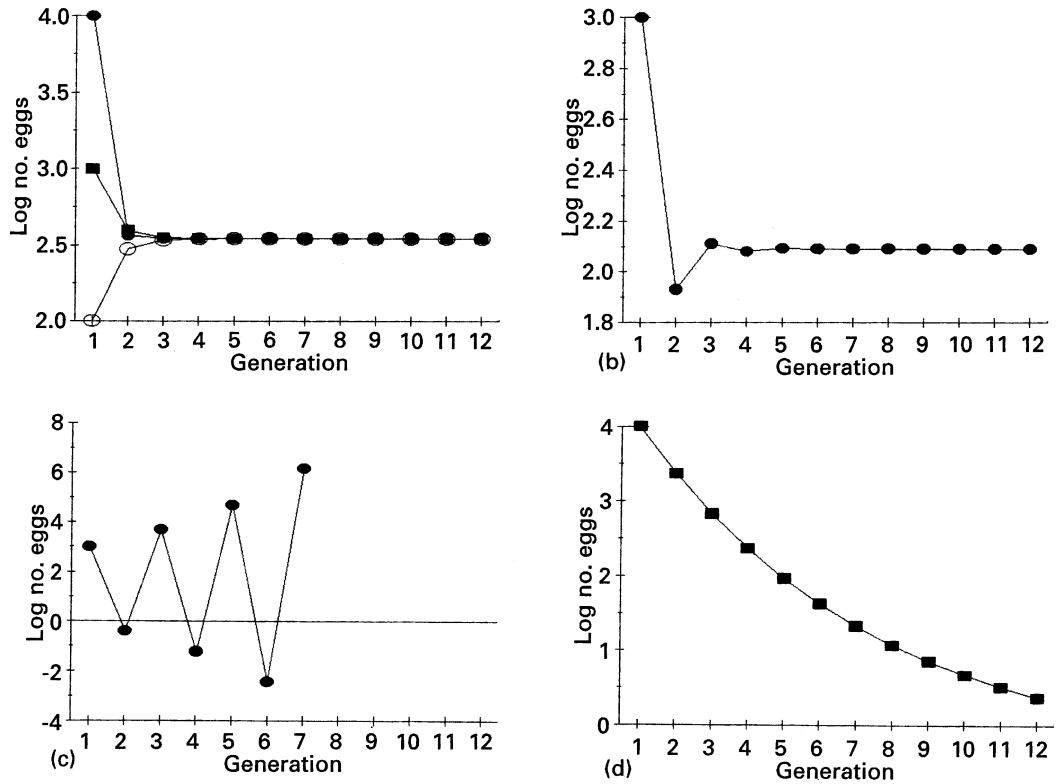

Figure 7.23 Predicted egg numbers over 12 generations: (a) with different starting densities of eggs; (b) with density dependence of larval mortality increased from $b = 0.86$ to $b = 1.2$; (c) with density dependence of larval mortality increased from $b = 0.86$ to $b = 2.4$, and (d) with density dependence of larval mortality removed ($b = 0$).

using key factor analysis, which we shall discuss shortly.

The winter moth feeds on a wide range of mainly deciduous trees, and occasionally defoliates oaks. The life-cycle at Wytham Wood, near Oxford, UK, where Varley and Gradwell's study was carried out, is as follows: eggs are laid in early winter in the tree canopy and hatch in spring to coincide with bud-burst; the caterpillars feed on the foliage until fully grown, whereupon they descend to the forest floor on lines of silk and pupate in the soil; adults emerge in November and December, the females ascending the trees to mate, the females than ovipositing in crevices on the bark. There is therefore one generation each year.

Data collected between 1950 and 1962 reveal that 'winter disappearance' (k_1), during the period between the egg stage and that of the fully grown larvae, is the key factor inducing population variation between years. Parasitism, disease, and predation (k_2–k_6) are relatively insignificant in this respect (Figure 7.24). The only significant regulating factor to be detected, however, was predation on pupae (k_5, Figure 7.25), subsequently shown to be caused mainly by shrews and ground beetles (Frank, 1967a,b; East, 1974; Kowalski, 1977). Parasitism showed no sign of being density-dependent, either at the larval stage (k_2) or at the pupal stage (k_6), leading the authors to suggest that the wide variations in densities from year to year, caused by the key factor 'winter disappearance', may be obscuring a possible delayed density-dependent relationship. The lack of any detectable regulating potential by the larval parasitoid

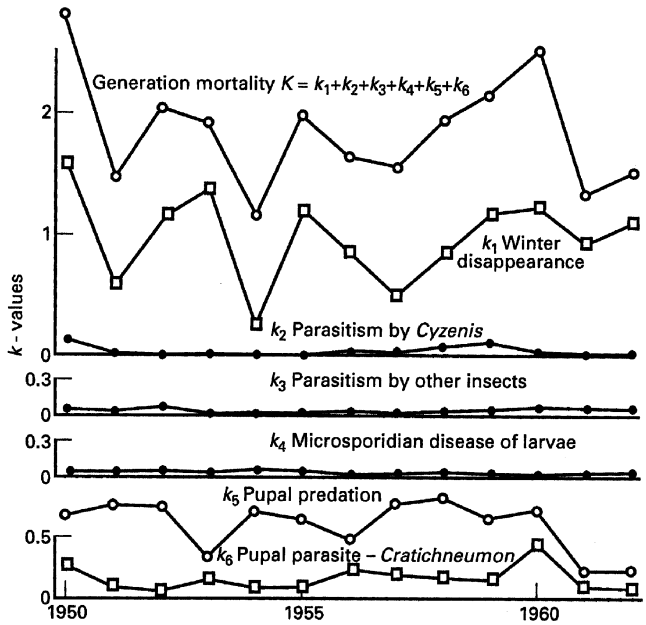

Figure 7.24 Key factor analysis of the mortalities acting on the winter moth. Source: Varley *et al.*, (1973). Reproduced by permission of Blackwell Publishing.

Cyzenis albicans (Diptera: Tachinidae) (k_2) was particularly surprising as this tachinid fly had previously been introduced in 1955 as a very effective biological control agent against winter moth in Nova Scotia, Canada (Embree 1966, 1971). This difference could perhaps be explained by higher levels of *Cyzenis* mortality in the UK. The parasitoid, although attacking the moth in the larval stage, continues to develop within the moth pupae throughout the summer and early winter and is therefore exposed to the same mortality factors as the moth pupae. Varley and Gradwell recorded as much as 98% mortality of *Cyzenis* puparia. This is higher than that for winter moth pupae, but understandable as *Cyzenis* spends 4–5 months longer in the soil, emerging in the spring.

A population model for the winter moth and its main parasitoids, *Cyzenis* (k_2) and the ichneumonid wasp *Cratichneumon culex* ($k_{6,}$), was developed by Varley *et al.* (1973), using basically the same approach which we elaborated above, but with two important differences. First, the variations in k_1 could not be predicted, so the observed values were used instead. Second, parasitism (k_2 and k_6) were modelled using the 'area of discovery' concept (subsection 7.3.7) rather than the simple regression relationships shown in Figure 7.25. There was good agreement between the model output and estimated field densities of the winter moth and its two parasitoids (Figure 7.26), although it has to be pointed out that testing the accuracy of a population model against the same data from which it is constructed, is not considered to be good modelling practice (subsection 7.3.8). However, collection of independent field data for acceptable validation of such life-table models is likely in many cases to prove impracticable, possibly involving years of extra work. This is one of a number of drawbacks associated with the Varley and Gradwell approach, which we shall now consider in detail.

Disadvantages of the Approach

The difficulty of obtaining additional field data for model validation highlights the single

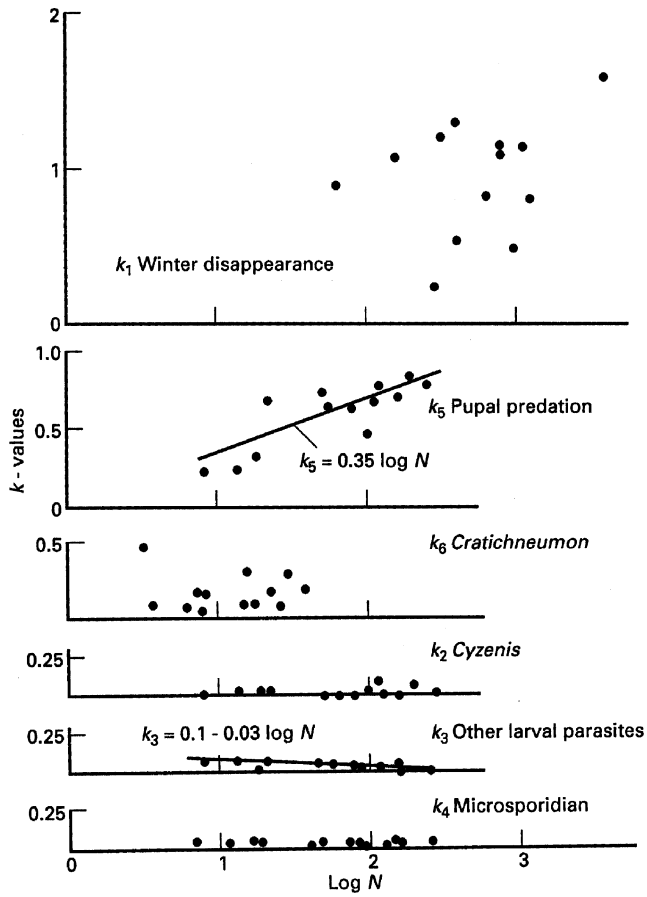

Figure 7.25 *k*-values of the winter moth mortalities plotted against the population densities on which they acted. k_1, k_2, k_4 and k_6 are density-independent; k_3 is weakly inversely density-dependent; k_5 is strongly density-dependent. Source: Varley *et al.*, (1973). Reproduced by permission of Blackwell Publishing.

biggest problem of the whole approach, namely that of securing a long enough sequence of data to perform the analysis with a reasonable likelihood of detecting statistically significant relationships (Hassell *et al.*, 1987). For insect populations having one generation a year, we may be contemplating the commitment of 15–20 years to a study, with no guarantees of success. The population processes affecting the main species may also change over the period of the study, with the result that key factors or density-dependent factors may alter or become obscured. Moreover, the method depends heavily on knowing all of the important factors to include in the study at the outset. There is not much scope for incorporation of new components at a later stage. There are a number of other problems as outlined in (A) to (F) below:

A: Contemporaneous and sequential mortalities Difficulties can arise when several agents act contemporaneously on a stage or when the precise sequence in which they act is unclear. Clearly, changes in the proportion killed by one agent will affect the number available to be attacked by other agents. Whether they are assumed to act concurrently or sequentially will have an important bearing on the results of the analysis. Buonaccorsi and Elkinton (1990) provide methods for estimating contemporaneous mortality factors using **marginal attack rates**.

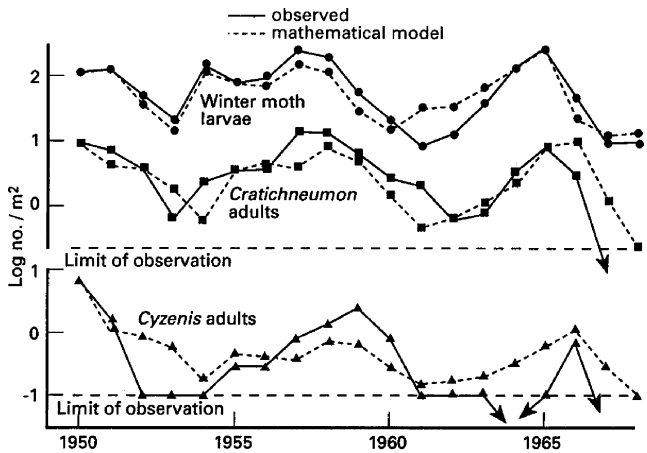

Figure 7.26 Observed changes in density of the winter moth and its two main parasitoids, and the densities predicted by the mathematical model. Source: adapted from Varley *et al*., (1973). Reproduced by permission of Blackwell Publishing.

The marginal attack rate of a mortality factor is equivalent to the proportion of the population which would be killed by the factor acting alone, instead of in combination with other factors (Bellows *et al*., 1992; Elkinton *et al*., 1992). The methodology can also be extended to give estimates of *k*-values (see Bellows *et al*., 1992 for a review). Assumptions about the sequence in which the mortalities act can strongly affect conclusions drawn, a point made forcefully by Putman and Wratten (1984) who audaciously illustrated their argument with a re-analysis of Dempster's (1975) study of the cinnabar moth (*Tyria jacobaeae*). In the original study, which assumed starvation of larvae to precede predation, Dempster's analysis showed starvation to be the key factor. Putman and Wratten reversed the sequence of these mortalities and found that predation became the key factor instead. We may question the justification for Putman and Wratten's re-ordering of the sequence of mortalities in this study, but we cannot ignore the point of the demonstration!

B: Composite mortalities Some of the mortality categories in the life-table may contain or mask a number of others which could be important key or regulating factors. This is particularly likely to be the case with poorly understood, wide categories, such as 'winter disappearance' in the winter moth example. Varley *et al*. (1973) accounted for this variable mortality as being mainly due to asynchrony between egg hatch and tree bud burst. Late opening of buds deprives young larvae of leaves to feed on, leading to death or emigration. However, other unstudied processes may also have had a part to play, for example, variations in adult fecundity, egg mortality from a number of possible sources, predation of early instars, etc..

C: Proving causation As Price (1987) has pointed out, the methods of life-table analysis, based as they are on correlation, do not always provide an unambiguous picture of cause and effect relationships. We can distinguish between **Type A density-dependence**, which is *causally* related to changes in population density and **Type B density-dependence** which is only *statistically related* (Royama, 1977), and to prove the former we need ideally to obtain life-table data both in the presence and absence of the suspected agent. Biological control introductions offer one potentially productive source of information on the causative role of natural enemies, but few 'before and after' life-table studies have in fact been carried out (subsection 7.2.2). Ryan (1997), however, has described a good example in the larch casebearer, *Coleophora laricella* (Lepidoptera), in North America, against

which two parasitoid species were introduced from Europe. Life-tables were carried out both before and after the introductions, and from those it became clear that one of the parasitoids, *Agathis pumila* (Braconidae) was the key factor inducing the decline of the moth. The parasitoid also appeared to be acting in a delayed density-dependent fashion, stabilising moth numbers at low densities. Another successful example, which we have already alluded to, is the study by Embree (1966,1971) of the introduction of two winter moth parasitoids, *Cyzenis albicans* and *Agrypon flaveolatum (*Ichneumonidae) into Nova Scotia. An opportunity to carry out a similar study was more recently afforded by the introduction of the winter moth to western Canada (Roland, 1990; see also Roland, 1994). The same two parasitoid species were introduced to British Columbia from 1979 to 1981, host populations subsequently declining to one-tenth of their peak density. Parasitism, mostly from *C. albicans*, rose from zero before introduction to around 80% in 1984 and declined thereafter to 47% in 1989. Mortality of pupae, interestingly, rose during the same period to a level higher ($>90\%$) than that caused by parasitism, suggesting a strong interaction between parasitism and subsequent mortality of unparasitised pupae. This effect was subsequently found to be present also in the Nova Scotia data. Roland suggested three possible explanations; these were:

1. Pupae parasitised by *Cyzenis* are present in the soil for twice as long as unparasitised pupae, so the greater availability of pupae after parasitoid introduction may be attracting higher numbers of pupal predators;
2. Predation and parasitism do not act independently of each other, predation rising in the presence of parasitism;
3. Pupal mortality factors in the soil have a minor effect at high population density and only exert a major effect after populations have declined.

To unravel the factors responsible, Roland carried out experiments with placed-out moth pupae (subsection 7.2.8), pitfall traps (subsection 6.2.1) to measure predator activity, and exclusion cages of different mesh size (subsection 7.2.3) to determine which predator sizes, if any, account for pupal mortality. Staphylinid beetles were found to be the most likely contenders, being also important predators of winter moth pupae in Britain. The results of Roland's experiments suggest that explanations (2) and (3) apply. Both have a part to play, predators showing a preference for unparasitised pupae (loading survival in favour of parasitoids and against the moth), predation becoming a major factor only after the parasitoid-induced decline (for an update on the winter moth analysis, see Roland, 1994).

D: Interaction effects Roland's analysis highlights the difficulties created when interaction effects occur between mortality factors. This is a problem which conventional life-table analysis is not equipped to deal with, assuming as it does that factors operate independently of each other. The only effective solution is to carry out factorial exclusion experiments (subsection 7.3.5) both in the presence and in the absence of the suspected interacting agents, under a range of relevant conditions, e.g. population density.

E: Compensatory effects An alternative method for evaluating the role of natural enemies from life-table data, discussed by Price (1987), might be to develop survivorship curves for cohorts of insects and to subtract from these the effects of specific natural enemies. A comparison could then made to assess the important contribution of natural enemies to mortality (see Figure 7.27). As Price himself points out, however, this type of analysis is likely to lead to very misleading conclusions, as it fails to recognise the possibility of compensation in the system. For example, removal of a high mortality due to natural enemies may be compensated for by a relatively higher mortality from other factors such as starvation or adverse weather conditions. An understanding of these potential compensatory mechanisms is crucial and again can only be gained adequately by experimentation.

F: Difficulties in detecting density-dependence It is possible for strongly regulated populations to show little variation from equilibrium, and this may make statistical detection of the

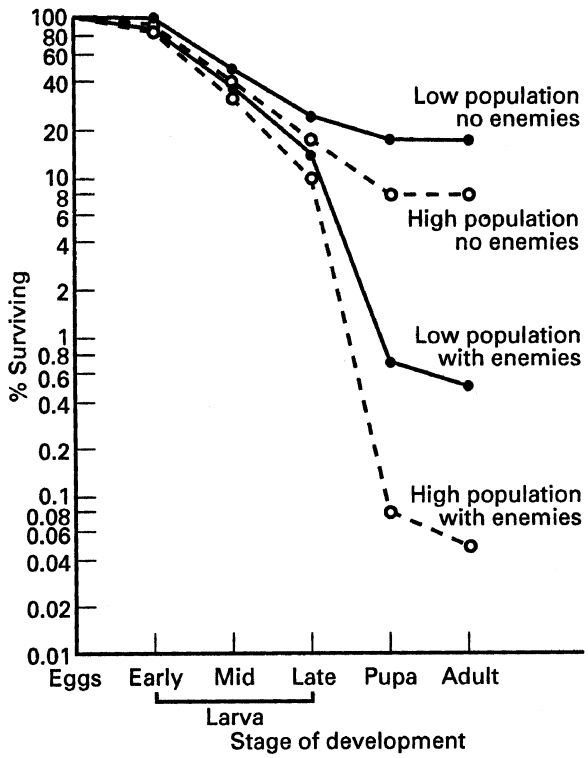

Figure 7.27 Survivorship curves for low and high populations of spruce budworm, *Choristoneura fumiferana*, together with those in which the effects of natural enemies have been removed. Source: Price (1987). Reproduced by permission of Elsevier Science.

processes of regulation difficult using traditional life-table methods (Gould *et al.*, 1990). Equally, stochastic variation may also obscure underlying density-dependent processes. Dempster (1983), for example, analysed 24 sets of data on Lepidoptera and could find in only three cases evidence of density-dependent mortality from natural enemies. He concluded that most insect populations are unlikely to be regulated by predators or parasitoids. However, Hassell (1985), using a simple model, showed that density-dependence can be present but remain undetected because of natural stochastic variation obscuring the relationships (see, however, Mountford, 1988). Hanski (1990) provides a useful review of the various problems inherent in detecting density-dependence from life-table and time-series data, together with a number of the statistical methods which have been proposed (see also Pollard *et al.*, 1987).

Being aware of the aforementioned pitfalls above is crucial before embarking on any population study based on age-specific life-tables, but it is in the nature of such long-term studies that unforeseen problems are likely to arise and may be difficult to correct after starting. For further, more detailed, treatments of age-specific or stage-structured life-table analysis, the reader is referred to Manly (1990).

Time-Specific Life-Tables

Time-specific (or vertical) life-tables are more suitable for use with populations in which the generations overlap, due to a short development time of the immature stages relative to the reproductive period of the adults (Kidd, 1979). Such populations (humans and aphids being examples) tend, after a period of time, to achieve a **stable age distribution** (Lotka, 1922) in which the proportion of the population in each age group or stage remains constant. In this situation, all the ecological processes affecting the population are, at least in theory, operating concurrently. This means that the relative numbers in each age group at any instant in time provide an indication of the proportional mortality from one age group to the next. However, we cannot deduce from this what mortality factors are operating, or whether any regulation is occurring, so the value of a time-specific life-table is limited in this respect.

Estimating mortality from parasitism may be easy to do with discrete generations (Varley, *et al.*, 1973; Van Driesche and Taub, 1983; but see subsection 7.3.2), but is more difficult when generations overlap. Van Driesche and Bellows (1988) provide an analytical method for doing this. Hughes (1962, 1963, 1972) developed a technique based on the time-specific life-table approach, which could be used for analysing aphid populations with a stable age (i.e. instar) distribution. Using a graphical method to compare population profiles at successive physiological time intervals (**Hughes' method**), Hughes was able to partition the mortalities

acting on the different instars, for example, parasitism, fungal disease and 'emigration'. As Hughes (1972) pointed out, however, there is no easy way of estimating errors in the construction of these life-table diagrams. In fact, the whole technique is critically dependent on the assumption of a stable age distribution. Although Hughes provided a simple statistical (χ^2) method to test the validity of the assumption, Carter *et al*. (1978) showed it to be insensitive to significant changes in the age distribution. Applying a more stringent test to Hughes' own field data for the cabbage aphid, *Brevicoryne brassicae* upon which his technique was developed (Hughes, 1962, 1963), Carter *et al*. (1978) found that these populations never achieved a stable instar distribution. Although Hughes' method has been widely used and was recommended for the International Biological Programme's study of the aphid *Myzus persicae* (Mackauer and Way, 1976), it should now only be used with extreme caution. Readers interested in the detailed methodology should consult Hughes (1972) and Carter *et al*. (1978).

Whilst Hughes' method is now considered to be of limited applicability, his work did lead directly to the development of the earliest simulation models for analysing insect populations with relatively complex population processes. For field populations with overlapping or partially overlapping generations, the use of such models is now the only sensible way forward. These techniques are discussed in detail below (subsection 7.3.8).

Variable Life-Tables

The term 'variable life-table' (or 'time-varying life-table') has been used to describe a particular class of computer-based, age-structured population model, in which the birth and survival rates experienced by each age-class change in a realistic way (Gilbert *et al*., 1976). The population life-table is in fact computer-generated from reproduction and survival relationships obtained in the field or laboratory, and as such becomes the output of the exercise rather than forming the basis of the analysis. The technique has therefore more in common with the methodology of simulation modelling than with that of life-table analysis, and will be discussed further in subsection 7.3.8.

7.3.5 MANIPULATION EXPERIMENTS

Convergence Experiments

The problems of detecting density-dependence from life-table data have already been discussed (subsection 7.3.4). One way of testing directly whether density-dependent mechanisms are operating is to carry out a 'convergence experiment' (Nicholson, 1957) in which densities of comparable subpopulations are manipulated to achieve artificially high or low levels and are then monitored through time. Convergence to a common density is then taken as evidence for density-dependent regulation. What constitutes an artificially high or low population density in the context of this type of experiment will vary according to the species under study, and can only be adequately assessed from some historical knowledge of past densities. Practical difficulties in manipulating densities of some species may also limit the usefulness of this technique. Amongst successful studies, Brunsting and Heessen (1984) manipulated densities of the carabid predator *Pterostichus oblongopunctatus* within enclosures in the field and found evidence for convergence within two years. Criticisms can be levelled at this technique in that enclosures may prevent emigration or immigration of beetles, leading to spurious mortality from 'artificial' sources. In this particular study, however, care was taken to note that beetle motility was naturally low and remained low even at the enhanced densities, there being no evidence for density-induced emigration. Gould *et al*. (1990) manipulated densities of gypsy moth by artificially loading eight forest areas with different densities of egg masses to achieve a wide range of infestation levels. This method revealed previously undetected density-dependent mortality in the larval stage, primarily due to two parasitoid species. Orr *et al*. (1990) also provide a good

example of a convergence experiment carried out in the laboratory using the freshwater predatory bug, *Notonecta*.

Factorial Experiments

Factorial experiments are used to determine whether factors potentially capable of limiting population numbers combine in a simple additive way, or show more complex patterns of interaction (Hilborn and Stearns, 1982; Arthur and Farrow, 1987; Mitchell *et al*., 1992). There are three criteria for successfully carrying out factorial experiments:

1. At least two factors need to be manipulated to at least two levels each;
2. A sufficiently long time-series of data must be available to assess equilibrium population levels around which numbers fluctuate;
3. There must be replication (Mitchell *et al*., 1992).

Mitchell *et al*. (1992) examined the interaction between resource levels (food and food/water ratios) and three population levels (zero, low, high) of the parasitoid *Leptopilina heterotoma* on laboratory populations of *Drosophila melanogaster*. This provided 12 different experimental combinations of the three potentially interacting factors. Both food and wasps showed significant effects on equilibrium levels, but without any significant interaction. With this type of experiment involving census data collected over time, the problem of serial autocorrelation is encountered (Arthur and Farrow, 1987), which makes the use of analysis of variance inappropriate. This can be circumvented using GLIM (see Mitchell *et al*., 1992; Crawley, 1993, 2002).

7.3.6 EXPERIMENTAL COMPONENT ANALYSIS

As explained in subsection 7.2.15, this approach is based on the assumption that the complexities of ecological interactions, such as those involving predation and parasitism, can be quantified in terms of a relatively small number of dynamic processes (Southwood and Henderson, 2000). Each process, reduced to its component parts, can be investigated experimentally and described by a series of equations. The equations describing all the component processes can then be incorporated into a system or population model, the accuracy of which can then be assessed by comparing its behaviour with real observations.

The so-called **components of predation** can be investigated experimentally using the important distinction between functional and numerical responses (subsection 7.2.15). To assess the significance of these responses, particularly to predator-prey and parasitoid-host population equilibrium levels and stability, two different modelling approaches can be adopted; one based on simple analytical models, the other involving the construction of more elaborate simulation models.

7.3.7 PUTTING IT TOGETHER: ANALYTICAL MODELS

Incorporating the Components of Predation

To assess the impact of parasitism or predation on an insect population, the information on functional and numerical responses needs to be incorporated into population models. Analytical models are usually based on systems of relatively simple equations which can be 'solved', usually by rearrangement, to provide straightforward answers. Some population models, however, have systems of equations which are too complex for solution, and so the only way of obtaining useful insights is to perform simulations with the model under differing conditions, for example by changing parameter values. Of course, there is no reason why models capable of analytical solution cannot also be used for simulation. Analytical solutions tend to be more tractable when a simple **deductive modelling** approach is adopted (deductive models are those based on very general, often intuitive, concepts, which can be useful in providing insights which might apply to particular case studies; philosophically, **deduction** is the process of arguing from the general case to the particular case (*cf*. induction, inductive models, subsection 7.3.4).

Whilst populations with overlapping generations and stable age distributions can be modelled analytically in continuous time using differential equations, this method is less suitable for the bulk of insect populations, at least in temperate regions, which have **discrete generations**, i.e. separated in time. A more appropriate modelling format is provided by difference equations which model population change in discrete time-steps, i.e. $N_{t+1} = f(N_t)$. The discrete time model which has been most widely used in insect population ecology is the host-parasitoid model of Nicholson and Bailey (1935), hereafter referred to as the **Nicholson-Bailey model**. Originally developed to explore the dynamic implications of parasitoid searching behaviour, the model has been extensively elaborated in recent years to examine other features of parasitoid, (and predator) biology (Hassell, 1978, 2000b; Waage and Hassell, 1982; Hassell and Waage, 1984; Godfray and Hassell, 1988; May and Hassell 1988; Hassell and Godfray, 1992; Mills, 2001). No doubt part of the appeal of this model lies in the simplicity with which it purports to capture the essence of the parasitoid-host interaction. Using a time-step of one generation, the model takes the following form:

$$N_{t+1} = F.\exp(-aP_t) \tag{7.14a}$$

$$P_{t+1} = N_t[1 - \exp(-aP_t)] \tag{7.14b}$$

where N_t and P_t are the numbers of hosts and parasitoids respectively at time t, F is the host net rate of increase in the absence of parasitism and a is the parasitoid's **area of discovery**, which is essentially the proportion of the habitat which is searched in the parasitoid's lifetime. A number of assumptions about the parasitoid and its host are implicit in these equations:

1. Generations of both populations are completely discrete and fully synchronised; the time-step (t) is therefore one generation;
2. One encountered host leads to one new parasitoid in the next generation;
3. The parasitoid is never egg-limited;
4. The area of discovery (= searching efficiency) is constant;
5. Each parasitoid searches the habitat at random.

This latter assumption is catered for in the model by using the Poisson distribution to distribute attacks at random between hosts. The zero term of the distribution (e^{-x}, where x is the mean number of attacks per host) defines the proportion of the host population escaping attack, in this case e^{-aP}, or $\exp(-aP)$. The proportion attacked is therefore $1-\exp(-aP)$. A more detailed description of the derivation of these equations is not provided here as it has already been covered in a number of texts (e.g. Varley *et al*., 1973; Hassell, 1978).

Following Hassell (1978), the equilibrium populations N^* and P^* can now be found by setting $N_{t+1} = N_t = N^*$ and $P_{t+1} = P_t = P^*$ giving the analytical solution:

$$N^* = F \tag{7.15a}$$

$$P^* = \frac{\log_e}{a} \tag{7.15b}$$

The equilibrium levels of both populations thus change with respect to both F and a.

However, the Nicholson-Bailey model *is inherently unstable*, a fact that can be confirmed either by simulation (Figure 7.28a) or by **stability analysis** (Hassell and May, 1973; Hassell, 1978, 2000b) (stability analysis is a technique which has been widely used in the analysis of deductive models, but the mathematics beyond the scope of this book; we refer readers to the appendices in Hassell and May, 1973, and Hassell, 1978). When perturbed from equilibrium, the model produces oscillations of increasing amplitude, which in the real world would result in the extinction of one or both populations (Figure 7.28a). Stability in the model could easily be produced, however, by the incorporation of a density-dependence component into F, to simulate, for example, competition between hosts for food resources. Progressively increasing the degree of density-dependence produces in the first instance **stable limit cycles** (Figure 7.28b) followed by **damping oscillations** (Figure 7.28c). Whilst density-dependence in F

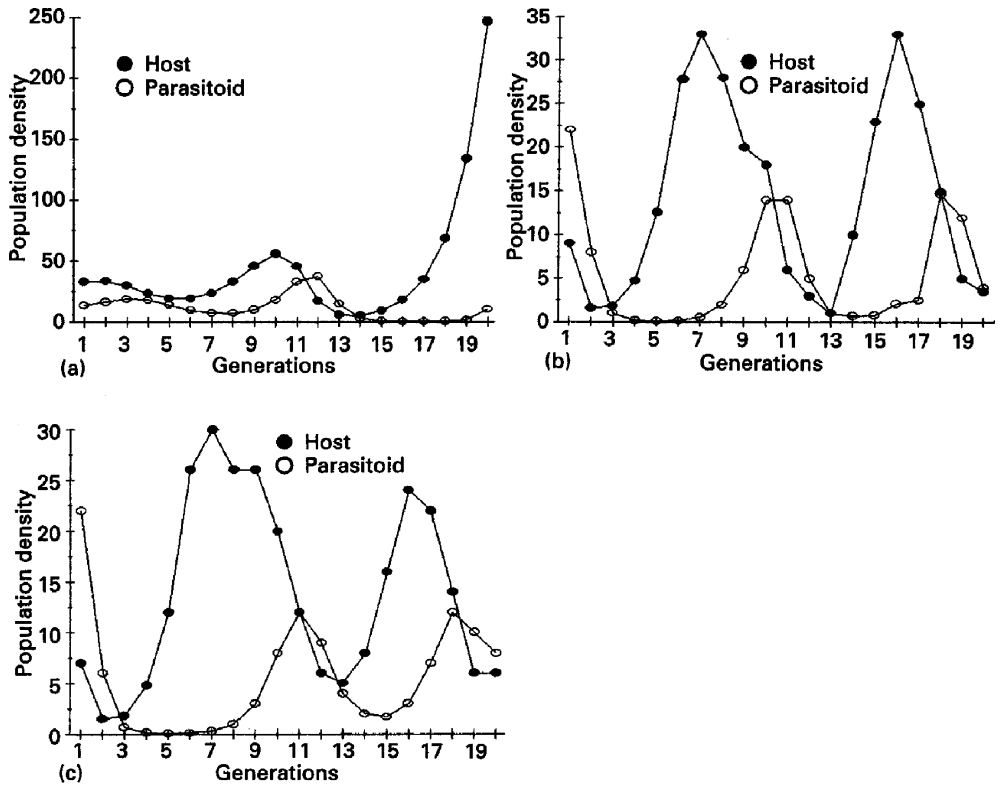

Figure 7.28 (a) Typical numerical changes predicted by the Nicholson-Bailey model. The incorporation of (increasing) density-dependence into the model results (b) in cyclical oscillations within an upper and lower boundary (limit cycles), followed by (c) damping oscillations which approach an equilibrium.

would be a reasonable component to incorporate, it does not advance our understanding of how features of parasitoid biology such as searching behaviour influence dynamics. To achieve that, more detailed descriptions of, for example, functional and numerical responses (see below) need to be incorporated into the model.

To facilitate the incorporation of the components of predation into the Nicholson-Bailey model, it is useful to begin with a more generalised form of the model (Hassell, 1978, 2000):

$$N_{t+1} = FN_t f(N_t P_t) \qquad (7.16a)$$

$$P_{t+1} = N_t[1 - f(N_t P_t)] \qquad (7.16b)$$

in which survival of hosts is a function of both host and parasitoid numbers. This survival function can now be explored in relation to the following components:

A: Functional responses: The assumption of a constant searching efficiency described by one component, a or a', is of course an oversimplification, given what we know of the way in which parasitoid and predator attack rates change with prey density (section 1.10). The way in which handling time affects this relationship have already been discussed (section 1.10) and can be described by **Holling's 'disc' equation:**

$$\frac{N_e}{P_t} = \frac{a'TN_t}{1 + a'T_hN_t} \qquad (7.17)$$

where N_e is the number of hosts encountered. This Type 2 functional response (section 1.10) can now be incorporated into the Nicholson-

Bailey model using the so-called **random parasite equation** first described by Rogers (1972):

$$N_a = N_t\left[1 - \exp\left\{-\frac{a'TP_t}{1 + a'T_hN_t}\right\}\right] \qquad (7.18)$$

Predator versions of equation 7.22 were also developed by Royama (1971) and Rogers (1972) to take account of gradual prey depletion during each interval t. Prey eaten by predators do not remain exposed to further encounters, in contrast to hosts which may be re-encountered and thus incur additional T_h costs to the parasitoid. Reproducing the Royama (1971) equation:

$$N_a = N_t[1 - \exp\{-a'P_t(T - T_h(N_a/P_t))\}] \quad (7.19)$$

Note that here N_a is present on both sides of the equation. The simplest way of finding N_a for given values of the other parameters is by **iteration** (i.e. by repeatedly substituting different values of N_a until both sides of the equation balance). Detailed mathematical derivations for both equations (7.18) and (7.19) are given by Hassell (1978). As the Type 2 functional response can be seen to be inversely density-dependent when percentage parasitism is plotted against prey density (Figure 1.15b), it is perhaps not surprising that when incorporated into the Nicholson-Bailey model as:

$$f(N_tP_t) = \exp[-(-a'TP_t)/(1 + a'T_hN_t)] \quad (7.20)$$

for parasitoids, its effect is to further destabilise the model. In general, the greater the ratio T_h/T the greater the destabilising effect, while the original Nicholson-Bailey model is re-established when $T_h = 0$ (Hassell and May, 1973). Although T_h thus determines both the degree of destabilisation and the maximum attack rate (= the plateau), both could equally well be influenced by egg-limitation (Hassell and Waage, 1984; section 1.10).

To explain the Type 3 (i.e. sigmoid) functional response, Hassell (1978) suggested a model which assumes that only a' varies with prey density, such that $a' = bN_t\,(1 + cN_t\,)$, with b and c constants. This gives a sigmoid analogue to the disc equation:

$$N_e/P_t = \frac{bN_t^2T}{1 + cN_t + bT_hN_t^2} \qquad (7.21)$$

where N_e is the number of prey encountered. For parasitoids, where hosts remain to be re-encountered:

$$N_a = N_t\left[1 - \exp\left(-\frac{bTN_tP_t}{1 + cN_t + bT_hN_t^2}\right)\right] \quad (7.22)$$

which can easily be incorporated into the Nicholson-Bailey model. Hassell (1978) also provides an alternative equation for predators, where prey are gradually depleted with time (see also Hassell *et al*., 1977):

$$N_a = N_t\left[1 - \exp\left\{-\frac{bP_t}{c}\left(T - \frac{T_hN_a}{P_t} - \frac{N_a}{bN_tP_t(N_t - N_a)}\right)\right\}\right] \quad (7.23)$$

N_a can again be found by iteration (see equation 7.23). Both equations (7.22) and (7.23) produce similar sigmoid relationships, but (7.22) is easier to use. Intuitively, such sigmoid responses might be expected to have a stabilising influence on population interactions, where the equilibrium falls within the density-dependent part of the response (Figure 1.15c). This was demonstrated by Murdoch and Oaten (1975) using continuous time differential equations (see below) with no time-delays. However, the time-delay of one generation inherent in the Nicholson-Bailey model is sufficient to prevent any sigmoid functional response, of the form of equation above, from stabilising an interaction (Hassell and Comins, 1978).

The above conclusion is, of course, restricted to those predators or parasitoids that are are prey- or host-specific, i.e. there is a **coupled interaction** between parasitoid host. Generalist predators and parasitoids, because they attack several prey species, are involved in a looser interaction, so the situation for these insects is somewhat different (Hassell, 1986). Here, predators may display switching behaviour (section 1.11). Hence, neither the predator numerical response nor its population density is likely to

be dependent on the abundance of any one prey type. The interaction between a generalist 'predator' and a *single* prey species was modelled by Hassell and Comins (1978) using equation (7.22) for a parasitoid population at equilibrium:

$$P_{t+1} = P_t = P^* \qquad (7.24)$$

This model was found to have two equilibria (Figure 7.29), the lower (S) being locally stable, while the upper (R) was unstable, such that prey exceeding R escaped parasitoid control and increased indefinitely. The important point in this model is that the total response of the parasitoid shows a sigmoid relationship with prey density. This can be achieved by a sigmoid functional response and a constant parasitoid density, as in the model, or by a rising numerical response to prey density, coupled with either a Type 2 or a Type 3 functional response (Figure 7.30).

At first sight the incorporation of functional responses into simple models seems to be fairly straightforward, but there are a number of complications which the reader needs to be aware of:

1. For a particular predator, the functional response is likely to vary with age or size of both predator and prey, and the model may have to be modified to incorporate the effects of age-structure (see **Other Analytical Modelling Approaches**, below);
2. Simple laboratory experiments to assess functional responses over a short time period (e.g. 24 hours) should be used in *generation-based* models with caution, as they may give a misleading impression of the predator's *lifetime* functional response. This, in the context of the Nicholson-Bailey model, is the relevant component if we are interested in understanding the effects of functional responses on population dynamics (Waage and Hassell, 1982; Kidd and Jervis, 1989). The problem may be avoided, however, when we consider the functional response in relation to predator aggregation in patchy environments (see below).

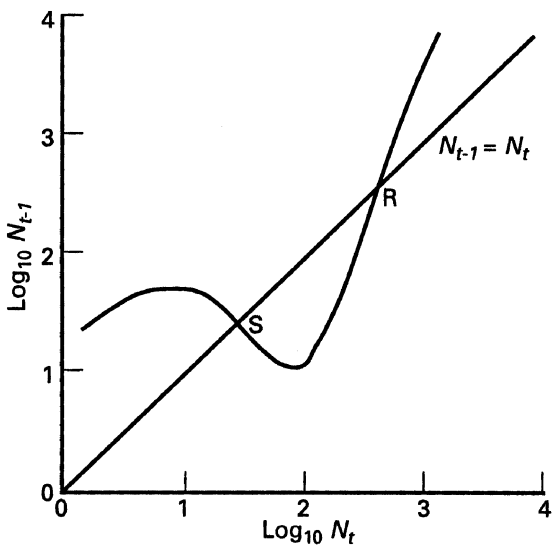

Figure 7.29 A population growth curve for equation 7.26. The intersections with the 45° line are the lower potentially stable equilibrium (S) and the upper unstable equilibrium (R). Source: Hassell and Comins (1978). Published by permission of Elsevier Science.

B: Aggregative responses: Although the Nicholson-Bailey model assumes random search by parasitoids (i.e. each host has the same probability of being parasitised), in reality natural enemies tend to show an aggregative response (defined in subsection 1.14.2). Examples have been widely reported and reviewed by a number of authors (Hassell *et al.*, 1976; Hassell, 1978, 2000b; Krebs and Davies, 1978; Lessells, 1985; Walde and Murdoch, 1988). This behaviour has already been discussed in relation to foraging behaviour (section 1.14), but here we are concerned with its implications for population dynamics.

Hassell and May (1973) modelled the effects of parasitoid aggregation in a simple way by first distributing hosts and parasitoids between n patches, then considering each patch as a sub-model of the Nicholson-Bailey model (Table 7.3). Thus, in each patch i, there is a proportion α_i of hosts and β_i of parasitoids.

As can be seen in Table 7.3, a greater proportion of parasitoids is placed in the high

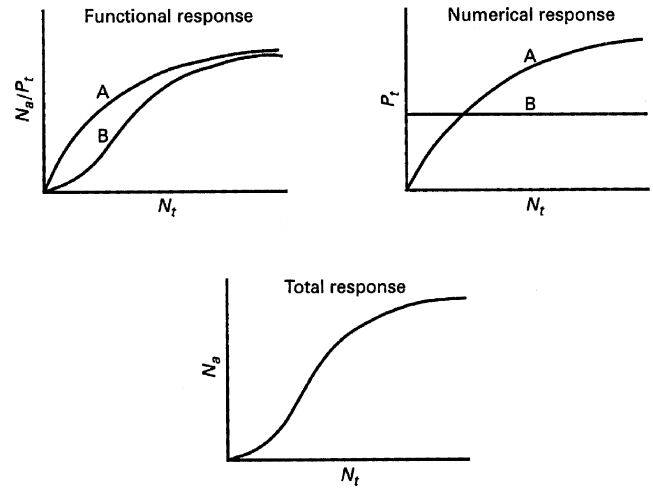

Figure 7.30 Alternative ways of achieving a sigmoid total functional response between prey eaten (N_a) by P_t predators and prey density N_t. The total response may be achieved by combining either response A or B with numerical response A or from functional response B with no numerical response. Source: Hassell, (1978). Reproduced by permission of Princeton University Press.

density patches of prey than in the low density ones. The parasitism function now becomes:

$$f(N_t P_t) = \sum_{i=1}^{n} \left[\alpha_i \exp(-a\beta_i P_t)\right] \qquad (7.25)$$

where a is the searching efficiency per patch. Equation (7.29) redistributes hosts and parasitoids as in the above scheme at the beginning of each generation. In the case where parasitoids are distributed evenly over all patches, we regain the property of random oviposition as in the original model. The stability analysis of Hassell and May (1973) shows that the model may now become stable with a sufficiently uneven prey distribution and enough parasitoid aggregation in high density host patches. To allow easier analysis of the properties of the model, Hassell and May (1973) used a single high host density patch (α) and distributed the rest of the host population evenly amongst the other patches $[(1-\alpha)/(n-1)]$. The parasitoid distribution was defined by a single **'aggregation index'** μ where:

$$\beta_i = c\alpha_i^{\mu} \qquad (7.26)$$

c being a normalisation constant. The degree of parasitoid aggregation is now governed by μ, $\mu = 0$ corresponding to random search and $\mu = \infty$ to the situation where all parasitoids are in the high host density patch. Stability is now affected by precise values of μ, F, α and $n-1$ (Figure 7.31).

Table 7.3 The proportional distribution of hosts (α) and parasitoids (β) between n patches to incorporate the aggregative response into the Nicholson-Bailey model

	Patch				
	1	2	3	4	5
α	0.5	0.2	0.1	0.1	0.1
β	0.8	0.1	0.05	0.03	0.02

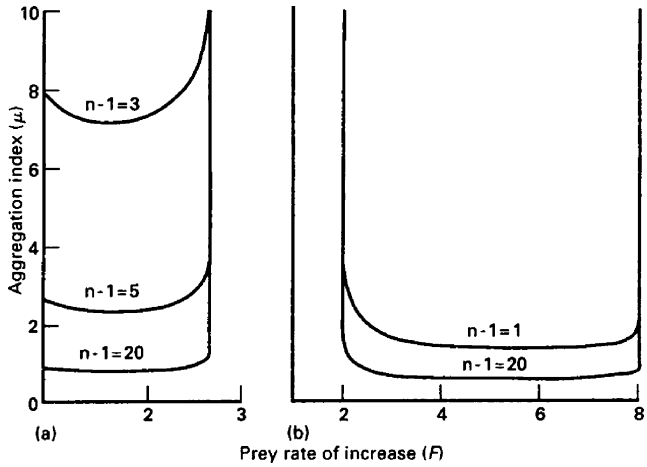

Figure 7.31 Stability boundaries between the aggregation index μ and the prey rate of increase F, for different values of $n-1$: (a) $\alpha = 0.3$; (b) $\alpha = 0.7$. Source: Hassell and May (1973). Reproduced by permission of Blackwell Publishing.

A more general ('phenomenological') model has also been developed by May (1978), to capture the essential features of parasitoid aggregation without the detail. This model uses the negative binomial to distribute parasitoid encounters between hosts. Thus:

$$N_{t+1} = FN_t[1 + (aP_t/k)]^{-k} \qquad (7.27a)$$

$$P_{t+1} = N_t\left[1 - (1 + (aP_t/k))^{-k}\right] \qquad (7.27b)$$

Here the parameter k (the exponent of the negative binomial) describes parasitoid aggregation, being strongest when $k \to 0$ and weakest when $k \to \infty$ (random). May's model, and variants thereof, has been used by a number of authors to include an aggregative response component into population models (Beddington *et al.*, 1975, 1976a, 1978; Hassell, 1980b). Hassell (1978, 2000b) provides a good account of the development and application of this model.

The stabilising potential of aggregation by predators and parasitoids must therefore temper our previous conclusions regarding the significance of functional responses, as measured in single-patch experiments (section 1.10). We can envisage the Type 2 and Type 3 response curves as essentially a 'within patch' phenomenon, with searching between patches defined by the aggregative response (Hassell, 1980a,b) (see also section **What is Searching Efficiency?**, below).

C: Mutual interference: The incorporation of mutual interference (subsection 1.14.3 gives a detailed discussion) into the Nicholson Bailey model was first carried out by Hassell and Varley (1969), using the inverse relationship between parasitoid searching efficiency and the density of searching parasitoids shown in equation (1.6). Removing the logarithms, this becomes:

$$a = QP_t^{-m} \qquad (7.28)$$

where Q and m are constants. When this equation is substituted into the Nicholson-Bailey model, it gives the equations:

$$N_{t+1} = FN_t \exp(-QP_t^{1-m}) \qquad (7.29a)$$

$$P_{t+1} = N_t[1 - \exp(-QP_t^{1-m})] \qquad (7.29b)$$

This modification has the effect of producing a stable equilibrium given suitable values of m and F (Figure 7.32). The higher the mutual interference constant m, and the lower F, the more likely stability becomes. Q has no effect on stability, but does affect the equilibrium level.

More elaborate, but behaviourally more meaningful, mathematical descriptions of

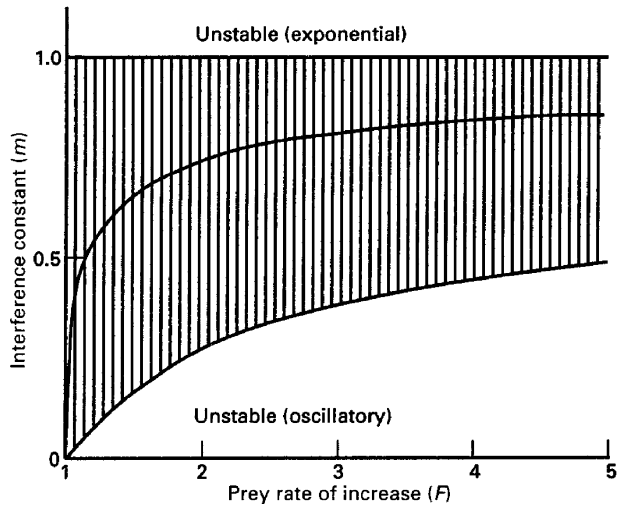

Figure 7.32 Stability boundaries for equation (7.33) in terms of the interference constant m and the prey rate of increase F. The hatched area denotes the conditions for stability, approached through exponential damping above the central curve and oscillatory damping below the curve. Source: Hassell and May, (1973). Reproduced by permission of Blackwell Scientific Publishing.

mutual interference have also been developed (Rogers and Hassell, 1974; Beddington, 1975) and explored in deductive models. While the precise stability conditions in these models may differ from the earlier version, the essential conclusion, that mutual interference can be a powerful stabilising force, remains intact.

D: Ratio-dependent functional responses: So far, searching efficiency (= attack efficiency) has been discussed in terms of separate attributes relating to either host (functional response) or parasitoid density (mutual interference). Some authors have instead proposed combining these effects in terms of the *ratio* of parasitoids (or predators) to hosts (or prey), to form the **ratio-dependent functional response** (Getz, 1984; Ginzburg, 1986; Arditi and Ginzburg, 1989). With respect to the Nicholson-Bailey model, this would replace the function $f(N_tP_t)$ with $f(N_t/P_t)$. Ratio-dependent functional responses have been developed by Beddington (1975), DeAngelis *et al*. (1975) and Getz (1984), sharing the general form:

$$N_a/P_t = N_t/P_t\{1 - \exp[-abP_t/(c + aN_t + bP_t)]\} \quad (7.30)$$

where a is the search rate, b the maximum number of hosts attacked per parasitoid and c is a constant. How far the functional response can be generalised in this way has been strongly debated in recent years (Murdoch and Briggs, 1996; Abrams, 1997; Abrams and Ginzburg, 2000; Hassell, 2000b), not least because of the way published functional response data have been re-analysed to fit the ratio-dependent format (Arditi and Akcakaya, 1990; Hassell, 2000b). What would help to settle at least some of the issues are some new studies specifically designed to detect ratio-dependence. Mills and Lacan (2004) provide a multifactorial protocol for parasitoid-host interactions.

Whatever the rights and wrongs of the ratio-dependence argument, the debate serves to emphasise the need to consider both the effects of hosts *and* those of parasitoid density on the overall functional response, the latter having perhaps been relatively neglected in the past (Mills and Lacan, 2004).

E: Numerical responses: The aggregative response could be said to be a form of numerical response, but here we use the term numerical response to refer specifically to changes in pred-

ator numbers from one generation to the next. In the Nicholson-Bailey model this is simply achieved by making the proportion of hosts killed by parasitoids in each generation, $[1-\exp(-aP_t)]$, equivalent to the number of parasitoids in the next generation. Each host killed therefore produces one living parasitoid. Gregarious larval development can be easily catered for by incorporating an additional component c into equation 7.14b, such that:

$$P_{t+1} = cN_t[1 - \exp(-aP_t)] \qquad (7.31)$$

Here, c is the average number of adult parasitoids to emerge from each parasitised host ($c = 1$ for solitary parasitoids and $c > 1$ for gregarious parasitoids, Waage and Hassell, 1982). As incorporated above, c has no effect on stability, but raising its value depresses the equilibrium (Waage and Hassell, 1982). However, c can also be further elaborated to cater for certain factors which may influence the number of parasitoids emerging per host. For conspecific superparasitism in solitary parasitoids, where only one larval parasitoid can survive, the situation will usually reduce to equation (7.14b) with $c = 1$ (exceptionally, superparasitism results in the death of both rivals in a host, in which case, $c < 1$). If there is mortality from factors such as multiparasitism or encapsulation, c will be < 1. Where clutch size affects larval survival adversely in gregarious species, this effect can be incorporated by replacing c in equation (7.31) with $c(1-\delta F)$, for values ≥ 0, where F is clutch size and δ is a constant defining the strength of density-dependence. This expression assumes a negative linear relationship between clutch size and progeny survival (although a negative exponential relationship may be more realistic), and will have a regulating effect both on the parasitoid population and the parasitoid-host interaction. To obtain realistic values for parameter c would require the construction of detailed life-tables for the parasitoid (see Hassell, 1969; Escalante and Rabinovich, 1979).

An additional parameter s can also be incorporated into equation (7.31) to take account of variation in the sex ratio of the parasitoid progeny. Thus:

$$P_{t+1} = scN_t[1 - \exp(-aP_t)] \qquad (7.32)$$

where s is the proportion of parasitoid progeny that are female (Hassell and Waage, 1984). Again, changes in s will have no effect on stability, but smaller values (i.e. male-bias in progeny) will raise the equilibrium. Density-dependence in s (subsection 1.9.2) (to incorporate density-dependence, s has to be altered to another form, i.e. $s = f$ [parasitoid density]), has a stabilising influence on the parasitoid-host interaction (Hassell *et al*., 1983; Hassell and Waage, 1984; Comins and Wellings, 1985; Mills and Getz, 1996).

For predators, the above models are inappropriate, as there is no simple relationship between the prey death rate and the predator rate of increase. The rate of increase of a predator population will depend on (Lawton *et al*., 1975; Beddington *et al*., 1976b; Hassell, 1978):

1. The development rate of the immature stages;
2. The survival rate of each instar;
3. The realised fecundity of the adults.

The biotic and abiotic factors affecting each of these components are considered in detail in sections 2.9, 2.10 and 2.7 respectively (Chapter 2). To build a general model of the predator rate of increase we would need to incorporate the effects of prey consumption on development and survival of the different instars, and on adult fecundity. This task would be beyond the scope of analytical modelling (Hassell, 1978), being more suited to simulation (subsection 7.3.8). Beddington *et al*. (1976b), however, took a simpler approach whilst retaining some of the features of predator reproduction. Adult fecundity, F, was related to the number of prey eaten during the predator's life by the equation:

$$F = c[(N_a/P_t) - \beta] \qquad (7.33)$$

where N_a is the number of prey attacked, c is the efficiency with which consumed prey are

converted to new predators and β is the threshold prey consumption needed for reproduction to start (see also equation [2.3] and related discussion). The model therefore takes account, in a simple way, of the predator's need to allocate some of the prey biomass assimilated to growth and maintenance (2.8.3). Incorporating this equation into the Nicholson-Bailey model yields the equations:

$$N_{t+1} = N_t \exp[r(1 - N_t/K) - aP_t] \qquad (7.34a)$$

$$P_{t+1} = c\left[\left\{N_t[1 - \exp(-aP_t)]\right\} - \beta P_t\right] \qquad (7.34b)$$

with the rate of increase of the prey population, in the absence of predation, defined by $(1-N_t/K)$, which includes a density-dependent feedback component (K being the carrying capacity). Of course, handling time, predator aggregation and mutual interference are not included. Nevertheless, the model can be used to show the stability differences between predator-prey models where $\beta > 0$ and those of parasitoid-host models where $\beta = 0$. The important effect of increasing β is to reduce the range of stable parameter space in the model. Furthermore, where $\beta = 0$, the model is **globally stable**, i.e. it returns to equilibrium irrespective of the degree of perturbation. With $\beta > 0$, only **local stability** is apparent, i.e. equilibrium is reattained only when perturbation is within certain limits. Thus, as predators need to eat more prey before reproducing, the chances of a stable interaction diminish.

Kindlmann and Dixon (2001) give examples of predator-prey systems where numerical and functional responses may be irrelevant to the system dynamics: predator reproduction should perhaps be correlated with the age of the prey patch rather than the number of prey present.

F: Other components: The number of relevant components of predator-prey and parasitoid-host interactions which could be examined in simple analytical (Nicholson-Bailey-type) models is potentially very large. We have attempted to summarise the approach with reference to some of the more widely discussed examples. Others which have been examined include: (a) differential susceptibility of hosts to parasitism (i.e. variability in host escape responses or physiological defences, temporal asynchrony between parasitoid and host) (Kidd and Mayer, 1983; Hassell and Anderson, 1984; Godfray *et al*., 1994); (b) parasitoid host-feeding (e.g. Yamamura and Yano, 1988; Kidd and Jervis, 1989; Murdoch *et al*., 1992b; Briggs *et al*., 1995; Jervis and Kidd, 1999); (c) competing parasitoids (e.g. Hassell and Varley, 1969; May and Hassell, 1981; Taylor, 1988b); (d) hyperparasitism (e.g. Beddington and Hammond, 1977); (e) multiple prey systems (e.g. Comins and Hassell, 1976); (f) host-generalist-specialist interactions (Hassell, 1986; Hassell and May, 1986); (g) combinations of parasitoids, hosts and pathogens (e.g. May and Hassell, 1988; Hochberg *et al*., 1990; Begon *et al*., 1999); (h) parasitoid egg-limitation (Mills and Getz, 1996; Mills, 2001); (i) dispersal between breeding sites (Weisser and Hassell, 1996) and local mate competition (Meunier and Bernstein, 2002); (j) incidence of diapause (Ringel *et al*., 1998).

Some of the multi-species interactions are discussed further in section 7.3.10. Some of the aforementioned authors have taken an **individual-based approach**. This recognises that properties such as parasitoid life-history, physiology and behaviour vary among individuals. It has involved (a) **state-structuring** (taking account of the physiological basis of foraging decisions e.g. size of egg load influences the decision whether to feed or oviposit, and (b) **stage-structuring** (taking account of size- and stage- variation among hosts, and the selective behaviour shown by parasitoids). Murdoch *et al*. (1992b), Brigg's *et al*. (1995, 1999) and Shea *et al*. (1996) in particular should be consulted.

Recent reviews of parasitoid-host and predator-prey models are provided by Mills and Getz (1996), Barlow and Wratten (1996), Barlow (1999), Berryman (1999), Hochberg and Holt, (1999), Hochberg and Ives (2000), Mills (2000, 2001), and Hassell (2000b).

What is Searching Efficiency?

Having reviewed the essential behavioural components involved in searching by natural

enemies (functional and aggregative responses, mutual interference), we are perhaps in a better position to answer the question of what is meant by the term 'searching efficiency'. This term has most often been used synonymously with 'attack rate', i.e. a more efficient predator kills more prey per unit time than a less efficient one. However, particularly in the context of population models, the detailed usage of the term has varied considerably. In the original Nicholson-Bailey model the area of discovery a defines a lifetime searching efficiency, whereas the 'attack' coefficient a' in equation (7.17), defines an 'instantaneous' searching efficiency in terms of numbers of prey attacked per unit time, T. In a patchy environment, however, searching efficiency is sensitive to two factors: (a) the patch-specific searching ability of the predators, and (b) the extent to which the distribution of the predators is non-random (Hassell, 1982a). To take account of this, Hassell (1978, 1982b) proposed a model for overall searching efficiency of predators (or parasitoids) where prey are gradually depleted:

$$a' = \frac{1}{n}\sum_{i=1}^{n}\left[\frac{1}{P_t T_i}\log_e\left(\frac{N_i}{N_i - N_{ai}}\right)\right] \qquad (7.35)$$

where n is the number of patches, N_a is the number of prey attacked, and N_i, N_{ai} and T_i are the number of hosts available, number of hosts parasitised, and the time spent searching, respectively on the ith patch. This equation represents an important step forward, as it views the functional response as essentially a within-patch phenomenon, i.e. occurring on a small spatial and temporal scale. Laboratory experiments to measure functional responses have, of course, been carried out on exactly this scale (section 1.10), so the within-patch interpretation affords a more realistic correspondence between experiment and modelling. It also circumvents the need for an average *lifetime* functional response measure to use in single-patch models, such as the original Nicholson-Bailey model (see above). Potentially, the patch model defined by equation (7.35) could be further expanded to include variation in the functional response with prey size, predator age and a range of other components, but the complexity involved would make this procedure more appropriate for the simulation approach discussed below (subsection 7.3.8).

An experimental measure of overall searching efficiency of a predator can be obtained by:

1. Estimating the average amount of time spent per predator in each patch (this can be calculated from the average number of predators found in each patch during a predetermined time period or over the course of the experiment (e.g. 24 hours);
2. Recording the average number of prey killed in each patch (providing a range of prey densities between patches).

These estimated parameter values can then be substituted in equation (7.39) to find a'. A suitable patch scale and experimental arena will need to be chosen, and this will depend on the prey species involved. Wei (1986), for example, used five clusters of rice plants, each set into a glass tube of water and interconnected by slender wooden strips to facilitate searching by the mirid bug *Cyrtorhinus lividipennis* for its prey, the brown planthopper *Nilaparvata lugens*. Ideally, a lifetime measure of searching efficiency could be obtained by repeating the procedure for each day of the predator's immature and adult life, and then either totalling or averaging the values of a' obtained. In practice, this may be very difficult due to the time involved and the large number of prey needed during the experiment.

Hassell and Moran (1976) proposed for parasitoids a measure of **'overall performance'**, A, that takes account of larval survival.

$$A = \frac{1}{P_t}\log_e[N/(N - P_{t+1})] \qquad (7.36)$$

where P_t and P_{t+1} are the densities of searching parasitoids in successive generations, and N is the number of available hosts. Clearly, a major constraint on the effectiveness of a parasitoid is likely to be mortality during the immature stages (subsection 2.10.2). 'Overall performance' may thus provide a more useful measure of

the relative usefulness of different parasitoid species in biological control (Hassell, 1982b) (subsection 7.4).

Other Analytical Modelling Approaches

So far, we have concentrated our attention on ways of expanding the Nicholson-Bailey model to include more realistic components of predation. Although space does not permit a detailed discussion, it should be mentioned that other analytical modelling frameworks are available and have been used successfully to gain insights into the dynamics of predator-prey interactions. We now briefly discuss two of these to provide the reader with a lead into the literature.

Models structured in terms of differential equations, to encompass continuous-time changes, have a long pedigree, beginning with the predator-prey interactions of Lotka and Volterra (Lotka, 1925; Volterra, 1931) (Berryman, 1999, provides a review). Host-parasitoid versions are also available (Ives, 1992; Hassell, 2000b). It has been shown that, where parasitoids aggregate in patches of high host density, such models are usually unstable (Murdoch and Stewart-Oaten, 1989), in contrast to their discrete-time counterparts. This serves to highlight the fact that there are important differences between the two model types, and also emphasises the point that the behaviour of analytical models can be highly sensitive to minor variations in their construction (Ives, 1992; Berryman, 1992). Perhaps more importantly, however, it points to a number of limitations of the discrete-time format, where artificial time-jumps of one generation are not only imposed on growth and mortality, but also on the behavioural attributes of the natural enemy (e.g. spatial redistribution within the habitat). It seems likely that future modelling work will increasingly place greater emphasis on the more flexible (but mathematically more complex) continuous-time format incorporating, for example, stage-structure (= age-structure) and developmental delays in the predator attack rate (Mills and Getz, 1996).

Continuous-time models incorporating age-structure have been developed by Nisbet and Gurney (1983), Gurney and Nisbet (1985) and extended to cover host-parasitoid systems (Murdoch *et al*., 1987; Godfray and Hassell, 1989; Gordon *et al*., 1991). Murdoch *et al*. (1987) showed that the incorporation of an age class which is invulnerable to parasitism into such a model with overlapping generations can, under certain circumstances, promote stability (subsection 7.3.2).

Discrete-time models incorporating age-structure have also been developed (Bellows and Hassell, 1988; Godfray and Hassell, 1987, 1989), using an elaboration of equation 7.31. Godfray and Hassell (1987, 1989) used this model form to demonstrate that parasitism can act to separate the generations of a host population, when otherwise they would tend to overlap. Whether host generations were separated in time depended on the relative lengths of host and parasitoid life-cycles.

An alternative modelling format which also allows for the incorporation of age-structure in populations is provided by matrix algebra (Leslie, 1945,1948). Matrices can also be used to incorporate quite complex age-specific variations in fecundity, survival, development and longevity, thus encompassing populations which show either discrete or overlapping generations. This flexibility also makes the matrix approach extremely suitable for simulation modelling (see below). The matrix methodology is also easy to use and has the advantage over computer-based models of having an easily communicated, mathematical notation. Williamson (1972) provides a good introduction to the use of the technique in population dynamics (see also Buckland *et al*. 2004).

7.3.8 PUTTING IT TOGETHER: SIMULATION MODELS

Introduction

The main value of analytical models has been to provide insights into the general possibilities of population dynamics and how they might alter

with changing conditions. Simulation models, on the other hand, attempt to mimic the detailed dynamics of particular systems and involve a somewhat different methodology. Simulation models of population dynamics can be constructed at different levels of complexity, from a relatively simple expansion of the analytical approach described in the previous section (Godfray and Hassell, 1989), to extremely elaborate systems of interlocking equations involving large numbers of components. However, they all share a common methodology for testing their accuracy **(validation)** and for assessing their behaviour **(experimentation and sensitivity analysis)** which will be discussed in detail. Whilst they can be formulated in conventional mathematical notation, simulation models are often constructed in practice as computer programs, which facilitate the complex calculations involved and also present the output in a readily accessible format. The models can be constructed in continuous or discrete time, again using systems of differential or difference equations respectively. The discrete time format has tended to be favoured by modellers interested in simulating the most complex insect population systems, involving age- or stage-specific fecundities and mortalities (Stone and Gutierrez, 1986, and Crowley *et al.*, 1987 give continuous-time examples). The aim of such inductive models is to encapsulate the detail of the particular system in question, with the emphasis on realism and accuracy. If successful, the model can often be used in decision-making, for example, in integrated pest management programmes.

To illustrate the way in which age-structure can be incorporated into a relatively simple simulation model, the model of Kidd (1984),which can be used to simulate populations with either discrete or overlapping generations, is discussed. The model considers a hypothetical population reproducing asexually and viviparously. Each individual is immature for the first three days, becomes adult at the beginning of the fourth day, reproduces on the fifth and sixth days, and then dies (Figure 7.33). The population is divided, therefore, into six

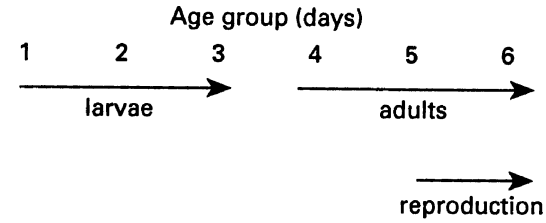

Figure 7.33 Life-history characteristics of an hypothetical population with discrete generations. Source: Kidd (1979). Reproduced from *Journal of Biological Education* by permission of The Institute of Biology.

one-day age groups, each adult producing, say, two offspring per day. To simulate population change from day to day, the computer:

1. Dimensions an array with six elements;
2. Places the initial number in each element of the array (initial age structure);
3. Calculates the total reproduction [REPMAX = (2* number of adults in age group 4) + (2* number of adults in age group 5) + (2* number of adults in age group 6)];
4. Ages the population by one day (this is done by moving the number in each age group N into age group N + 1);
5. Puts the number reproduced (REPMAX) into the one-day-old age group.

The model operates with a time-step of one day, and steps 3. to 5. can be repeated over as many days as required to simulate population growth. This very simple model can then be further elaborated to include additional components, such as mortality acting on each age group (e.g. either a constant proportional mortality, a uniformly-distributed random mortality or a density-dependent function) (Figure 7.34). By changing the length of immature life relative to reproductive life the model can be used to explore the behaviour of populations with either discrete, partially overlapping, or fully overlapping generations (see Kidd, 1979, 1984 for details and for a BASIC program).

This very simple deterministic simulation model provides the basic format for a number of modelling approaches, including the variable life-table models of Hughes and Gilbert (1968)

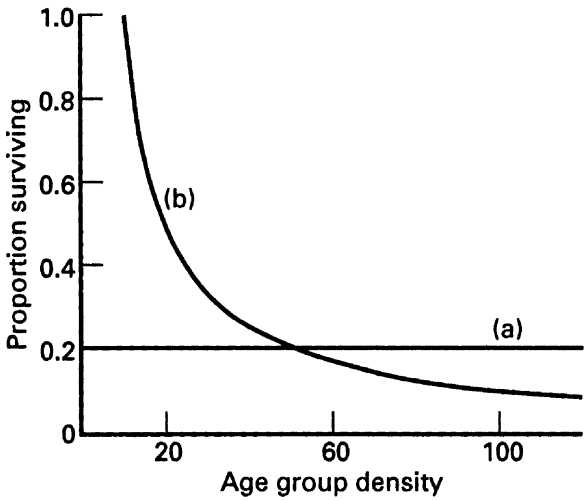

Figure 7.34 The effects of: (a) a density-independent factor: (b) a density-dependent factor used in the simulation model. Source: Kidd (1979). Reproduced from *Journal of Biological Education by* permission of The Institute of Biology.

and Gilbert *et al*. (1976) (see above). Variable life-table models have generally been used to determine how the relationships governing birth and death processes affect population dynamics in the field, and they rely on intensive laboratory and field observations and experiments during the model development and validation stages (Gilbert *et al*., 1976; Gutierrez *et al*., 1990; see Getz and Gutierrez, 1982 for an historical review and examples). The sequence of steps involves:

1. Estimating intrinsic relationships, such as development rates and growth rates;
2. Estimating extrinsic biotic relationships, including density-dependent effects, effects of natural enemies etc.;
3. Estimating abiotic effects such as weather factors.

Ideally, the accuracy of the model needs to be tested at each stage, before the modeller can confidently proceed with the incorporation of more complex components. In this way, the model increases progressively in realism and complexity, without sacrificing accuracy.

To illustrate the process, including the important techniques of validation and sensitivity analysis, the population study of Kidd (1990a,b) on the pine aphid, *Cinara pinea is* used as an example. This species infests the shoots of pine trees (*Pinus sylvestris*) and can be cultured in the laboratory as well as studied in the field. The first task was to construct a relatively simple model to simulate the changing pattern of aphid numbers on small trees in the laboratory. This model incorporated a number of relationships obtained from observation and experiment, including: (a) an increase in the production of winged migratory adults with increasing population density; (b) a decrease in growth rates (adult size) and development rates with crowding and poor nutrition; (c) a decline in fecundity with smaller adult size. The effect of variable temperature on development was included by accumulating day-degrees above a thermal development threshold of 0°C (subsection 2.9.3).

Model Validation

The output from this prototype model (Figure 7.35) was compared with the population changes on four small saplings using a 'least-squares' goodness-of-fit test. Testing the accuracy of model output against real data is the process of **model validation** and should involve some statistical procedures (Naylor, 1971), although many population modellers have in the past relied on subjective assessment of similarity (e.g. Dempster and Lakhani, 1979). One frequently used statistical method is to compare model output with means and their confidence limits for replicated population data (Holt *et al*., 1987). If the model predictions fall within the confidence range, then it can be assumed that the model provides an acceptable description of the data. Frequently, however, the replicates of population data show divergent behaviour, and for the model to be useful it needs to take this variability into account. This was the case with the pine aphid: the four populations on the small trees behaved differently, and simply to average the data for each sampling occasion would have,

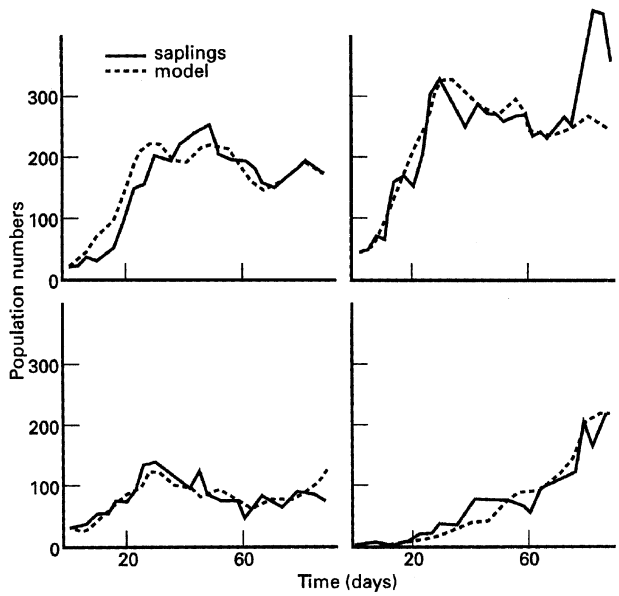

Figure 7.35 Population dynamics of pine aphids on four laboratory saplings and population numbers predicted by the simulation model. Source: Kidd (1990a). Reproduced by permission of the Society for Population Biology.

at best, lost valuable information and, at worst, been statistically meaningless. The 'least-squares' method used (see Kidd 1990a for details) took into account the variability both within and between the trees and, in fact, the model was able to explain 83% of the variation in the data. The model thus seemed to provide an acceptable description of aphid population behaviour in the laboratory, so was it adequately validated at this stage? The answer is *no* for another important reason: *for acceptable validation, models need to be compared with population data which have been independently collected and not used to provide data for the construction of the model*. The four sapling populations, in this case, had yielded data which had been used in the model. For acceptable validation an independent set of populations on four trees was used and here the model explained 78% of the variability within and between trees (Figure 7.36).

Experimentation and Sensitivity Analysis

At this stage it was possible to use the model to assess the relative contribution of each component to the aphid's population dynamics in the laboratory. This involved manipulating or removing particular components and observing the behaviour of the system. It was also possible to reveal those components to which model behaviour was particularly sensitive and which might repay closer investigation. For example, changing the reproductive capabilities of the adults, not surprisingly, affected the rate of population increase, but this effect was extremely sensitive to the nature of density-dependent nymphal mortality. Population growth rates and the periodicity of fluctuations were also found to be sensitive to changing development rates, mediated through changes in plant quality.

Having achieved a sufficient degree of accuracy in simulating laboratory populations, it was possible to then incorporate the complexities associated with the field environment. In the first instance, this meant: (a) revising some components of the aphid/ plant interaction to make them more appropriate for field trees; (b) including the more extreme variations in temperature associated with the field; (c) incorporat-

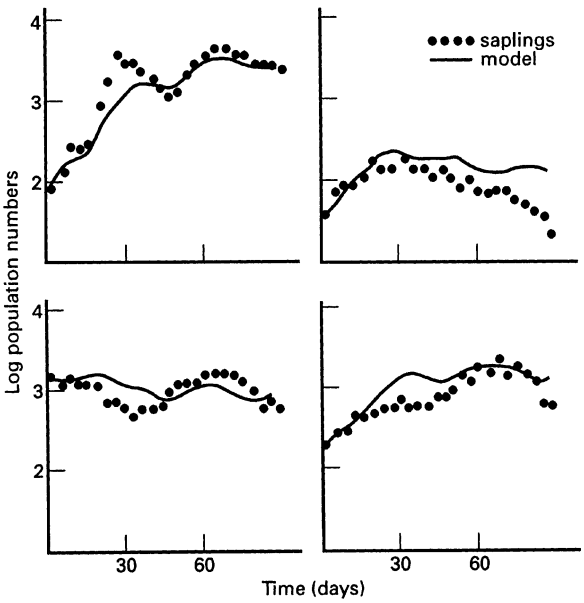

Figure 7.36 Population dynamics of pine aphids on four independent laboratory saplings and population numbers predicted by the simulation model. Source: Kidd (1990a). Reproduced by permission of the Society for Population Biology.

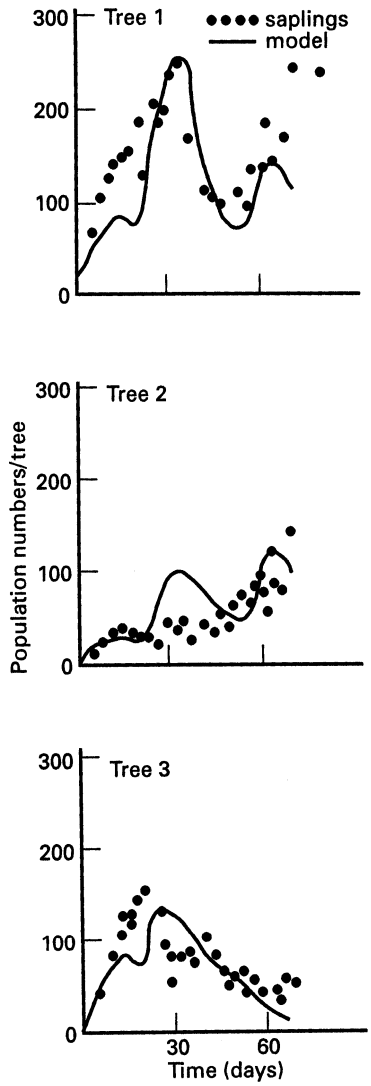

Figure 7.37 Pine aphid population dynamics on three field saplings from which predators were excluded and population numbers predicted by the simulation model. Source: Kidd (1990b). Reproduced by permission of The Society for Population Biology.

ing weather effects. At this stage mortalities due to natural enemies were excluded. Output from the revised model was then compared with populations on three field saplings covered by cages designed to exclude predators (Kidd, 1990b). At this stage the model was able to account for 52% of the numerical variation within and between trees (Figure 7.37); this is acceptable given the greater innate variability of field data. The model also predicted a pattern of numbers which was very close indeed to that of aphid populations on mature field pine trees, at least in the early season (Figure 7.38). Where predictions diverged from reality later in the season, this could probably be taken to reflect the impact of natural enemies which only become apparent after June.

While the model, as it stands, is purely deterministic in its construction, a stochastic element could have been included in any of the components by defining one or more parameters, not as constants, but in terms of their mean values and standard deviations. A random number generator could then have been used to produce a normally-distributed random number each time a parameter was used in the model. In this way, biologically meaningful variation could be reproduced. A number of standard computer programs are available for generating random

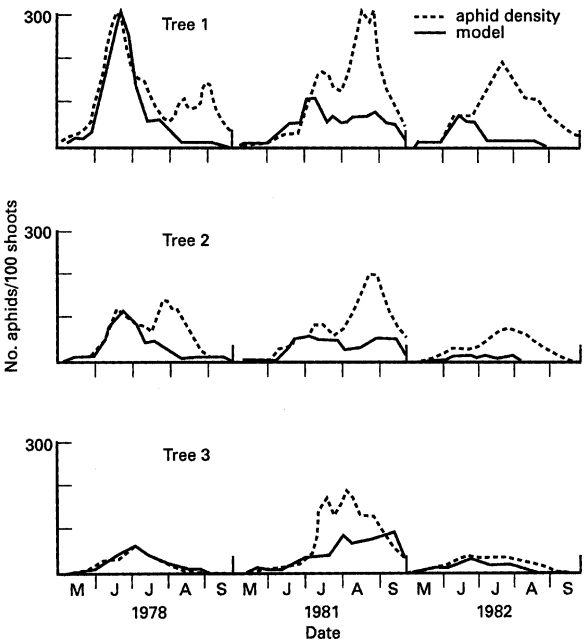

Figure 7.38 Pine aphid population densities on three mature field trees during three years, together with densities predicted by the model. (Source: Kidd, 1990b.) Reproduced by permission of the Society for Population Biology.

numbers for a range of possible distributions. Sometimes it is desirable in a simulation model to reproduce an entire or partial distribution of data, rather than a single random value. This technique, known as **Monte Carlo Simulation**, can easily be carried out using the random number generator in an iterative fashion.

We have dwelt at some length on this example in order to show the general procedures involved in a simulation study. Similar examples concerning aphid populations are provided by Hughes and Gilbert (1968), Gilbert *et al*. (1976), Barlow and Dixon (1980), and Carter *et al*. (1982) and for other insect groups by Gutierrez *et al*. (1988a,b) (cassava mealybug and cassava green mite), Holt *et al*. (1987) (brown planthopper), and Sasaba and Kiritani (1975) (green rice leafhopper). Clearly, there may be considerable variations in model construction, depending on the nature of the problem to be solved. For example, Stone and Gutierrez (1986) developed a model to simulate the interaction between pink bollworm and its host plant (cotton), which also included a detailed plant growth submodel (see also Gutierrez *et al*., 1988a,b; Gutierrez *et al*., 1994). Incorporating the effects of variable temperatures on development into simulation models has also been achieved in a number of different ways Stinner *et al*., 1975; Frazer and Gilbert, 1976; Pruess, 1983; Wagner *et al*., 1984, 1985; Nealis, 1988; Comins and Fletcher, 1988; Weseloh, 1989a; Kramer *et al*. 1991). With models based on a physiological time scale, for example, it is clearly impractical to use a time-step as short as one day-degree (one day may be the equivalent of about 20 day-degrees). In this situation, a physiological time-step corresponding to a convenient developmental period may be used. For *Masonaphis maxima*, Gilbert *et al*. (1976) used the **quarter-instar period or 'quip'** of 13.5 day-degrees Farenheit.

Incorporating the Components of Predation

Having constructed a basic simulation model, how can we best incorporate the components of predation or parasitism? This can be done in each of three ways:

1. By applying simple field-derived mortality estimates for predation (or parasitism) (subsection 7.3.2) in the prey (host) model; this tells us nothing of the dynamic interaction between predator and prey, however;
2. By constructing submodels for predators and parasitoids, involving age-structure if necessary, and including components of searching behaviour (e.g. functional and aggregative responses and numerical responses as already described in subsections 2.7.3, 2.9.2, 5.3.6 and 7.3.7); by constructing natural enemy submodels, but in this case using empirically derived estimates of predation and its effects.

These methods are now discussed in turn:

Method 1

Vorley and Wratten (1985) used a variable life-table model of the cereal aphid, *Sitobion avenae*

to predict population growth in the absence of natural enemies. The difference between predicted and actual numbers in the field was attributed to 'total mortality', i.e. parasitism plus 'residual mortality'. By discounting the effects of parasitism (estimated from dissections), residual mortality could then be calculated. By running the model with only residual mortality acting, the effects of parasitism on the dynamics of the aphid population could finally be estimated. Clearly, this technique, in common with others in this category, is capable only of assessing the 'killing power' of a mortality source during the period for which data have been collected. It has no reliable predictive power (i.e. it is an **interpolative** rather than an **extrapolative method**) and yields no information on the dynamic interaction between parasitoid and host. A similar approach was adopted by Carter *et al*. (1982) for parasitoid and fungal mortality of cereal aphids.

Method 2

A. **Age-specific:** Age-specific submodels have frequently been developed for parasitoids and predators for use in both theoretical models (see Godfray and Hassell, 1989, and Kidd and Jervis, 1989; for parasitoids) and in field or crop-based simulation studies (Yano, 1989a,b, for parasitoids and Gilbert *et al*., 1976 for parasitoids and hover-flies). These submodels are generally constructed in the same format as the main population model on which they act. For example, in the *Masonaphis maxima* model of Gilbert *et al*. (1976), the parasitoid *Aphidius rubifolii* was found to have an egg development time of six quips. In the theoretical model of Kidd and Jervis (1989), time and age groups were measured in days, the life-history features of the host and parasitoid having the structure shown in Figure 7.39.

B. **Searching behaviour:** As functional response relationships have usually been derived from short duration experiments (e.g. 24 hours; section 1.10), they are likely to be more meaningful when incorporated into simulation models with a short time-step of, for example, one day, rather than the one-generation time-step of the Nicholson-Bailey model (subsection 7.3.7). In the model of Kidd and Jervis (1989), parasitoids searched for hosts sequentially, either at random between patches, or in selected high host density patches. Type 2 functional responses were incorporated for both feeding and oviposition in a biologically realistic way, by allowing each parasitoid a maximum available search time per day (10 hours), the 'efficiency of search' being constrained by feeding handling time, oviposition handling time and egg-limitation. A full BASIC program listing for this model is provided by Kidd and Jervis (1989), to which we refer readers interested in developing the individual-based queueing techniques described.

Few attempts have been made to measure the searching efficiency of natural enemies in the field (e.g. Young, 1980; Hopper and King. 1986; Jones and Hassell, 1988; Weisser *et al*., 1999, Schenk and Bacher, 2002), probably due in large part to the obvious technical difficulties of confining particular densities of predators/parasitoids and prey/hosts within localised patches. Where the Holling disc equation (equation (7.21)) has been used to model a parasitoid-host interaction, parameter values have sometimes been estimated from field data. This can be done by iteration, i.e. by using a range of alternative parameter values in repeated simulations to find which fit the data best (Ravlin and Haynes, 1987). The difficulty here is that values of a' and T_h which produce accurate simulations may bear no resemblance to laboratory estimates of these parameters, casting some doubt on the realism of at least some components in the model. An alternative approach has been used by some workers (Godfray and Waage, 1991; Barlow and Goldson, 1993) to capture the essence of parasitoid search in a way that can potentially be used to describe their dynamic interactions

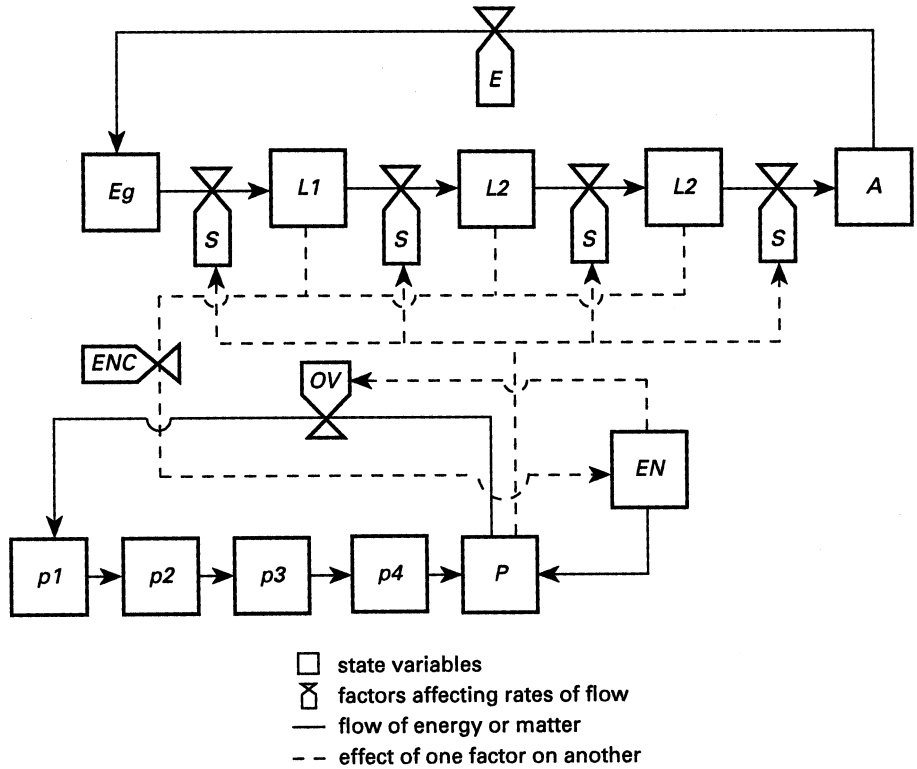

Figure 7.39 Simplified relational diagram of the parasitoid-host system: *Eg*, host egg age group; *L1-L3*, host larval age group; *A*, host adults; *S*, survival rate; *E*, host reproductive rate; *ENC*, encounter rate,. *p1-p4*, immature parasitoid age groups; *P*, adult parasitoids; *OV*, rate of parasitoid oviposition; *EN*, individual parasitoid energy stocks. Source: Kidd and Jervis (1989). Reproduced by permission of The Society for Population Biology.

with host populations in the field. This is done using the simple Nicholsonian component $[1-\exp(-aP^m)]$ to describe the proportion of hosts attacked during a defined time period, where P is the number of parasitoids, a is searching efficiency and m defines the strength of density-dependence in parasitoid attack (see equation (7.33). Barlow and Goldson (1993) used this term to model the interaction between the weevil pest of legumes, *Sitona discoideus* and an introduced braconid parasitoid, *Microctonus aethiopoides*. Thus:

$$q = 1 - \exp(-aP^m) \qquad (7.37)$$

where q is the estimated proportion of hosts parasitised. Leaving aside life-cycle complications, parameter values for a and m were simply estimated from the relationship between parasitoid densities (estimated from percentage parasitism) with percentage parasitism in the following generation (Figure 7.40). To do this, equation (7.37) was first linearised by rearrangement using the following steps:

$$1 - q = \exp(-aP^m) \qquad (7.38a)$$

$$\log_e(1 - q) = -aP^m \qquad (7.38b)$$

$$-\log_e(1 - q) = aP^m \qquad (7.38c)$$

$$\log_{10}[-\log_e(1 - q)] = \log_{10} a + m \log_{10} P \qquad (7.38d)$$

Thus, $\log_{10}[-\log_e(1-q)]$ can be regressed against $\log_{10}P$ with slope m and intercept $\log_{10}a$ to find values of m and a. The model derived by Barlow and Goldson (1993) gave acceptable

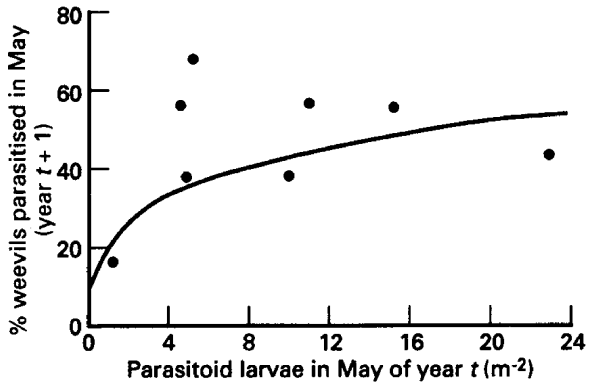

Figure 7.40 The relationship between the density of parasitised legume weevils in year t and the percentage parasitised in year $t+1$. Source: Barlow and Goldson (1993). Reproduced by permission of Blackwell Scientific Publishing.

predictions of parasitoid and host numbers over a ten-year period. This is an example of a **model of intermediate complexity** (Godfray and Waage, 1991; see also subsection 7.4.3).

A. **Numerical responses:** As indicated in subsection 7.3.7, the rate of increase of a predator population depends on a number of components, namely, the development rate of the immature stages, the survival rate of each instar, and the fecundity of the adults. To build a comprehensive submodel of the predator rate of increase we would need, therefore, to incorporate the various factors which might affect prey consumption (e.g. prey density, handling time, aggregative response, mutual interference), together with the effects of prey consumption on development and survival of the different instars, and on adult fecundity. The influence of prey consumption on predator growth and development, survival and fecundity has already been discussed at length, together with experimental methodologies and descriptive equations (subsections 2.9.2, 2.10.2, 2.7.3). Although the modelling of these processes is technically feasible with computer simulation, to obtain sufficiently detailed information on all of the components involved would be a potentially very time-consuming operation. To our knowledge, the task has not yet been fully carried out for any single predator species in the formal detail prescribed by Beddington *et al*. (1976b). However, Crowley *et al*. (1987) describe a detailed *sub*-model applicable to damselfly predators, but without an equally detailed model for the *Daphnia* prey. Nevertheless, this clearly remains an important challenge for those interested in modelling predator-prey processes, as a computer submodel, based on general theory and incorporating all of the components of predation in this way, would have immense practical as well as theoretical value.

Method 3

For simulation models specifically designed to mimic field population dynamics, the components of predation and parasitism have usually been incorporated into submodels in a more pragmatic way relevant to the particular system and problem in question. As a wide variety of approaches has been adopted, with no single unified methodology, we can only present a number of examples to illustrate the possibilities. For parasitism acting in their *Masonaphis maxima* model, for example, Gilbert *et al*. (1976) first established the duration (in quips, see above) of the four developmental stages of *Aphidius rubifolii* (egg, larva, pupa and adult). Adult females were assumed to search at random, ovipositing a constant (average) number of eggs per quip, in susceptible third and fourth instar and young adult aphids. No account was taken of how parasitoid oviposition might vary with aphid density, so it is perhaps not surprising that the model, at least in its early versions, provided poor predictions of aphid parasitism.

For syrphid predation, on the other hand, Gilbert *et al*. (1976) assigned predator larvae to four developmental periods, each of 10 quips duration and consuming 1, 2, 6 and 15 average-sized aphids per quip. This provided a consumption rate of 240 aphids for each syrphid to

complete development, a figure broadly agreeing with observations. In the model, syrphid eggs were simply laid in proportion to aphid densities, thus generating the number of larvae attacking the aphids. Aphids were assumed to be attacked at random, and were subtracted from the model during each time-step in proportion to the voracity and number of each syrphid age group present. No account was taken of predator starvation or survival, but, even so, Gilbert *et al*. found this simple model gave reasonably accurate predictions of predation in the field.

Some variations on this **'maximum consumption' method** have been carried out on other systems. Wratten (1973) and Glen (1975) respectively examined the effectiveness of the coccinellid, *Adalia bipunctata* and the mirid bug, *Blepharidopterus angulatus* as predators of the lime aphid, *Eucallipterus tiliae* using the methodology developed by Dixon (1958, 1970). The sequence of tasks involved in this approach are:

1. To monitor the age distribution and population densities of prey and predators at regular intervals (of, say, one week) over a period of months or years;
2. To measure the efficiency of capture (% encounters resulting in capture) of each predator instar in relation to the different prey instars in the laboratory;
3. To estimate the amount of time spent searching per day (for *A. bipunctata* this meant time exceeding a minimum temperature threshold for activity, corresponding approximately to the 16 hours of daylight);
4. To estimate the percentage of searching time spent on areas with prey (44% for *A. bipunctata);*
5. To calculate the area traversed/day (= distance travelled in 1 hour at mean temperature ∗16∗0.44∗R [R being the width of perception of the predator]);
6. To find the % of time spent on areas already searched;
7. To calculate the 'area covered' from 5. and 6., taking into account time wasted by covering areas already searched;
8. To estimate the time spent feeding as a proportion of the total time available.

Components 3. to 5. are all determined empirically from observations in the laboratory. It can be seen that some of the above procedures follow Nicholson and Bailey's (1935) methodology for calculating the 'area of discovery' (subsection 7.3.7) and effectively take into account handling time and aggregation of predation, but not mutual interference. Negative effects on the predators are again simply estimated from experiments to determine the minimum density of aphids required for predator survival at different stages (see reference to Gilbert *et al*., 1976, above).

For *A. bipunctata* (Wratten, 1973), the number of aphids of a particular instar consumed by each coccinellid instar per week (N_a) could be found from the equation:

$$N_a = 0.01A^*D^*0.01E^*C_o^*n^*C_f \qquad (7.39)$$

where A is the area covered/larva/week, D is the density of aphids, E is the capture efficiency, C_o is a correction factor for aphid distribution, n is the density of larvae, and C_f is the correction factor for the proportion of available time spent feeding up to satiation. This calculation was made for every combination of coccinellid and aphid instar in the populations on each sampling date, so as to arrive at an overall number of aphids in each instar removed per week. Assuming that these aphids would have remained in the population in the absence of predation, Wratten (1973) was able to estimate the potential aphid population size and structure in the absence of predation from information on the development times and reproductive patterns of the aphids, obtained by confining them in leaf cages. The difference between the observed aphid population and the predicted population in the absence of predation thus demonstrated the effect of coccinellid mortality. A later study incorporating these components of coccinellid predation, together with similar ones for the mirid predator (Glen, 1975), into a simulation model of the lime aphid (Barlow

and Dixon, 1980), showed that both species can have a destabilising effect on aphid numbers.

Clearly, where prediction of the prey death rate over a limited period (e.g., one season) is the sole aim of the model, fairly crude representations of predation may suffice. However, where a longer time-frame is being simulated, especially one involving 'coupled', i.e. monophagous predator-prey interactions, a more accurate submodel incorporating a predator numerical response will be needed. A number of approaches which could be used to achieve this end are described in detail for aphid-coccinellid interactions by Frazer *et al*. (1981a), Gutierrez *et al*. (1981), Mack and Smilowitz (1982) and Frazer and Raworth (1985). In practice, none of these studies attempted to simulate the interaction beyond one field season. To take just one example in detail, Frazer *et al*. (1981a) derived empirical relationships for coccinellid reproduction and survival by confining adult beetles with aphids in screen-walled cages in alfalfa fields. Ives (in Frazer *et al*., 1981a) had found in the laboratory that female coccinellids required 1.3 mg live weight of aphids/quip for maintenance, additional prey being converted to eggs at a rate of 0.7 eggs/mg of aphid. A direct relationship could then be established between the predation rate and the reproductive rate of adult females. Overall survival from egg to adult was estimated by comparing the expected numbers of eggs laid in the cages with the number of beetles recovered at emergence, and was subsequently found to show a sigmoid relationship with aphid density (using total aphid density during the first larval instar of the beetle, although a running average could also have been used).

7.3.9 COMBINING TESTABILITY AND GENERALITY

We have emphasised above the distinction between analytical and simulation models, and also the difference between the deductive and inductive approaches. The latter, essentially philosophical, distinction is equivalent to that proposed by May (1974b), who referred instead to **strategic** and **tactical models**. Strategic models attempt to describe and abstract the general features of population dynamics while ignoring the detail. Tactical models, on the other hand, are developed to explain the complex dynamics of particular systems, being particularly useful in population management programmes. Tactical models, however, may not readily lead to general conclusions about population dynamics. Although they can readily be tested against real system behaviour (subsections 7.3.4 and 7.3.8), the testing of strategic models is more problematical, as more than one model can often be invoked as a plausible explanation of a particular phenomenon. Murdoch *et al*. (1992a) suggest a way of making models both testable and general, by first building tactically-orientated models, then progressively stripping out the detail, whilst testing the new models at each stage to determine the loss in predictive capacity. Murdoch *et al*. (1992a) provide an example of how the methodology might be used by referring to the work of Murdoch and McCauley (1985) and McCauley and Murdoch (1987 and related papers) on *Daphnia*-algae interactions. A similar approach has also been advocated by Berryman (1990) and Kidd (1990c).

7.3.10 MULTISPECIES INTERACTIONS

Introduction

So far in this chapter our consideration of how to model the role of natural enemies in population dynamics has been largely confined to two-species interactions, whether they be predator-prey or parasitoid-host in nature. We need to be aware, of course, that these simplified models ignore a whole range of potential influences from the wider multispecies environment. In recent years, some of these effects have begun to be explored in more depth, using either a modelling or an experimental approach.

Apparent Competition

It has long been recognised that two species sharing a resource that is in short supply are likely to enter into a state of competition

(resource competition) which may result in one species being disadvantaged and possibly even excluded by the other. What has now become clear is that a similar outcome, known as **apparent competition**, may occur if the two species are *not* in resource competition, but share a natural enemy. Here, the increase in abundance of an alternative prey leads to a numerical increase in the predator, with increased levels of attack on both prey species. Over time, one prey species may be eliminated. While this phenomenon has been extensively explored through analytical modelling (see Bonsall and Hassell 1999, for a review), empirical evidence has tended to be circumstantial and anecdotal (but see below). Bonsall and Hassell (1999) also provide a critical review of some of these studies and their experimental designs. Experimental conditions may be difficult to maintain in practice, particularly where the species involved are especially mobile (Morris *et al*., 2001). A good field demonstration of apparent competition (with appropriate experimental design) involving two aphid species and their shared coccinellid predator is provided by Müller and Godfray (1997). See also van Nouhuys and Hanski (2000) and Morris *et al*. (2001) for examples involving two primary parasitoid species with a shared secondary parasitoid.

Apparent Mutualism

Apparent competition is not the only 'indirect' interaction mediated by shared natural enemies. Higher densities of one prey species may, through switching, satiation or egg-depletion, cause predation pressure on the other prey species to be relaxed. The second prey species can thus benefit from the presence of the first, an interaction known as **apparent mutualism** (Holt, 1977; Holt and Kotler, 1987; Abrams and Matsuda, 1996). Hoogendoorn and Heimpel (2002) reported on one such interaction between two coccinellids with a shared parasitoid. One of the coccinellids was a relatively poor host for the parasitoid, acting as a mortality 'sink' for its eggs. This effect acted to reduce parasitism on the other coccinellid and raised its equilibrium levels, as demonstrated by an analytical model of the interaction.

Competing Predators

The reciprocal three-species interaction of two predators sharing a single prey species has been analysed extensively using the simplified parasitoid-host format of the Nicholson-Bailey model (see Hassell, 2000b, for a review). Here, a host species is attacked by two parasitoid species, each of which aggregates its attacks independently of the host and the other parasitoid. Using the model of May (1978) (described in 7.3.7 above), with the parameter k describing the degree of aggregation of attacks, the conditions for stability were shown to depend on: (a) the sequence of parasitoid attack, (b) the outcome of multiparasitism and (c) the stability of the individual host-parasitoid links. The possibility of the three species coexisting is greatly reduced if only one parasitoid contributes strongly to stability, while coexistence becomes impossible if the two parasitoids attack randomly (unless some other density-dependence is introduced). May and Hassell (1981) also consider the effects on equilibrium levels (as opposed to stability), while Hassell (2000b) discusses these outcomes in relation to the practical issue of multiple biological control introductions (see also section 7.4).

Interactions Across Three Trophic Levels

'Bottom-up' and 'Top-down' Effects

The relative extent to which insect herbivore populations are constrained by natural enemies or by plant resources, i.e. whether the herbivores are mainly 'top-down' (natural enemy) or 'bottom-up' (plant resource) constrained, has recently been subject to much discussion. The debate has centred upon the argument put forward in the classic paper by Hairston *et al*. (1960) that herbivore (*sensu lato*) populations tend not to be food-limited. This has often been interpreted as saying that the world is green

because herbivores are maintained at low abundances by top-down, rather than bottom-up forces. The observation that significant intra- and interspecific competition occur rather infrequently in insect herbivores, has lent weight to the top-down argument (but see Denno *et. al*., 1995), as have the dramatically successful cases of classical biological control (Strong *et al*., 1984). Most food web and population dynamics theory applied to insect herbivores has assumed that bottom-up forces have, in general, minimal effects on abundance (Hawkins, 1992).

However, it is increasingly being argued that the importance of bottom-up effects ought not to be underestimated; while plants may appear to be a superabundant resource for insect herbivores, in reality they are not, because food quality varies significantly and may thus act as a major constraint, both upon herbivore abundance (e.g. Hunter and Price, 1992; Ohgushi, 1992; Wratten, 1992) and upon herbivore quality (from the standpoint of the growth, development, fecundity and survival of the natural enemies consuming their tissues, see Thaler, 2002, and subsection Chapter 2). Bottom-up constraints are now receiving more equitable treatment in the study of herbivore population dynamics and it is increasingly being accepted that, instead of it being a case of either top-down or bottom-up, an interaction between the forces is the more realistic view to take (e.g. Kato, 1994; Stiling and Rossi, 1997; Kidd and Jervis, 1997). However, certain barriers to progress continue to impede our understanding of the relative roles of top-down and bottom-up forces in tri-trophic systems.

Foremost of these barriers is the semantic confusion existing over the dynamic nature of the 'forces'. Some authors refer to these in the sense of 'regulation', where density-dependent effects act to increase stability and/or persistence. Others continue to use the term 'control', which could mean either 'regulation' or simple density-independent population *suppression*. When trying to disentangle the relative roles of top-down and bottom-up constraints, it is clearly important to understand which processes are being analysed (Hassell *et al*., 1998). Hunter *et al*. (1997) used two-way analysis of variance on 16 years of time-series data for the winter moth, in an attempt to partition out the relative contributions of top-down and bottom-up forces to variations in numbers. 'Tree' and 'year' were taken as main effects in the analysis, with Hunter assuming that a measurable proportion of tree variation could be attributed to bottom-up effects (budburst phenology = *density-independent*), while a proportion of year-to-year variation could be assigned (using correlation analysis) to delayed *density-dependent* top-down effects. Thus, by confusing different processes, Hunter failed to separate out the relative contributions of bottom-up and top-down processes to *either* regulation or population suppression. A number of statistical problems with Hunter's study have also been detected and these are discussed in detail by Hassell *et al*. (1998).

Despite its problems, Hunter's analysis has been of some value in pointing to the need for adequate population data, rigorous analysis and clarity of definitions, before significant progress in this area of the bottom-up/top-down debate can be made. In a more recent paper, Hunter himself makes these arguments (Hunter, 2001), and outlines some possible approaches which may circumvent some of the difficulties. These may involve time-series analysis, life-table analysis (taking into account female oviposition preferences) and experimental methods. The latter are likely to involve detailed factorial experiments in which plant resources and predation pressure are manipulated. Stiling and Rossi (1997), for example, manipulated small island populations of *Asphondylia* flies, their four species of parasitoid, and the seaside plant, *Borrichia frutescens*, on which the flies produce galls. Using two-way repeated-measures analysis of variance, the effects of parasitism and plant quality (as manipulated by nitrogen fertiliser) on the number of fly galls could be determined. A significant interaction between parasitism and plant nitrogen was detected, with parasitism being important only on the high-nitrogen plants where galls were abundant. Of course, this study was not designed to separate out

regulating from non-regulating influences, but it does serve as a reminder of the complex interactions between natural-enemy and plant-mediated effects which may occur.

As we have argued previously (Kidd and Jervis, 1997), the issue of 'regulation' may not actually be of major importance in the bottom-up/top-down debate. What is more at issue is whether particular populations are routinely constrained by plant resources alone, or whether the impact of natural enemies acts to keep numbers below the resource ceiling. In an analysis of 32 forest pest species, we found that 12 were reported to have population densities largely determined by resource constraints, while in 20 cases natural enemies were considered to have a major role (Kidd and Jervis, 1997). Even in the 12 resource-limited cases, however, we argued that natural enemies may have a more important role to play than simple mortality or percentage parasitism estimates might suggest. Using a simple simulation model, it was possible to demonstrate some profound and unexpected dynamical effects from the interaction between resource limitation and relatively low predator-induced mortalities (see Kidd and Jervis, 1997 for details). This emphasises the point that the interactions between bottom-up and top-down effects are likely to be complex in many cases, and may continue to defy our simplistic attempts to assess their relative contributions. Walker and Jones (2001) provide a recent synthesis of the subject.

Bottom-up effects will not not necessarily be exerted by the host's or prey's food plant *per se*, but by fungal endophytes, which will influence herbivore and parasitoid/predator performance (subsection 2.9.2) and ultimately affect herbivore and natural enemy abundance and dispersion patterns. Therefore, they could influence parasitoid-host population dynamics. See Omacini *et al*. (2001).

Modelling Bottom-up/Top-down Interactions

Despite the wealth of modelling techniques available (see sections 7.7 and 7.8), surprisingly few attempts have been made to model *natural* tritrophic systems of the plant-herbivore-predator variety (see, however, Barlow and Dixon, 1980; Kidd, 1990c; Larsson *et al*., 1993; Mills and Gutierrez, 1999). We suspect that this may be partly due to a lack of information on the plant-herbivore side of the interaction. The same, however, is not true of agriculture, where both analytical and simulation models have been constructed to help integrate biological control programmes into crop-pest systems (e.g. Gutierrez *et al*., 1984, 1988a, 1994; Holt *et al*., 1987; Mills and Gutierrez, 1999), or to understand the effects of plant resistance on the pest-natural enemy interaction (Thomas and Waage, 1996). In the latter case, it has been shown by both modelling and experimentation that the presence of natural enemies (usually parasitoids) can enhance (additively or synergistically) the effects of plant resistance. As parasitism tends to be density-independent, its proportional effect is greater on partially resistant crop varieties. More complex tritrophic outcomes were explored by Hare (1992) using a series of graphical models, but a more rigorous theoretical treatment (and review) is provided by Thomas and Waage (1996).

Higher-level Tritrophic Interactions

Moving up one trophic level, in our survey of tritrophic interactions, brings us to a consideration of the interaction between a prey species, its predator (A) and a further predator species (B), attacking predator A. Its simpler host-parasitoid-hyperparasitoid equivalent has been considered, at least theoretically. Nicholson and Bailey (1935) extended their basic host-parasitoid model to include a randomly attacking hyperparasitoid, so that equation 7.14 now becomes:

$$N_t + 1 = F\exp(-aP_t) \tag{7.40a}$$

$$P_{t+1} = N_t[1 - \exp(-aP_t)][\exp(-aQ_t)] \tag{7.40b}$$

$$Q_{t+1} = [1 - \exp(-aP_t)][1 - \exp(-aQ_t)] \tag{7.40c}$$

where Q is the hyperparasitoid population.

The effect of including Q is to raise the host equilibrium, although the interaction remains unstable. However, including a density-dependent component to the host rate of increase F stabilises the interaction and allows a more meaningful analysis of the effects of Q. As Beddington and Hammond (1977) have shown, Q always raises the host equilibrium and the range of parameter space allowing stability is smaller than in the absence of Q. However, stability is more likely if the hyperparasitoid has a higher searching efficiency than the primary parasitoid, P.

May and Hassell (1981) have also explored the above interaction, where both parasitoids aggregate their searching behaviour. In this situation, stability can occur in the absence of host density-dependence. The interaction is always stable if both parasitoid species strongly aggregate their searching behaviour and if Q has a higher searching efficiency than P. In all cases the effect of the hyperparasitoid is to raise the host equilibrium, a conclusion which has serious implications for biological control (see section 7.4). See also Ives and Jansen (1998) for a stochastic approach to modelling host-parasitoid-hyperparasitoid interactions.

Intraguild Predation

When a species feeds on more than one trophic level it is showing **omnivory**, a feature which appears to be relatively common in natural communities (Polis *et al*., 1989), but is of debatable significance in stabilizing community structure. A particular category of omnivory is shown by **intraguild predation**, where two predator species potentially compete for the same prey, but one of them also feeds on its competitor (Polis *et al*., 1989). The overall result is thought to be a reduction in predator pressure and a decrease in top-down control of the shared prey (Rosenheim *et al*., 1995; Snyder and Ives, 2001). In a more detailed analysis using analytical models, Holt and Polis (1997) were able to demonstrate that for the three-species interaction to be stable, the **intermediate predator** (the one acting as both predator and prey) has to be the better competitor for the shared prey. If the latter becomes scarce, the **top predator** (the one acting solely as a predator) may become extinct, as a result of its inferior competitive ability. As the prey becomes more abundant, the risk of extinction of the *intermediate* predator increases as a result of apparent competition with the shared prey. Empirical evidence to support these predictions is scarce, possibly due to the same difficulty in carrying out suitably designed experiments as were discussed in relation to apparent competition (see above). Demonstrating that it is not impossible, however, Finke and Denno (2003) carried out replicated 2×2 factorial (randomised block) experiments to test for intraguild predation between a wolf spider and a mirid bug predator, feeding on a shared planthopper prey. In support of theory, the presence of both predators reduced planthopper numbers *less* than did each predator in isolation. Mirid numbers were suppressed by the presence of spiders, but spider numbers were unaffected by the mirids, a result again predicted by theory, for higher prey densities (see also section 7.4 for a discussion of this study in relation to biological control).

Rosenheim *et al*. (1995) provide a comprehensive review of intraguild predation in insects, and review also empirically-based simulation models and general analytical models of intraguild predation in relation to biological control (see also section 7.4; also see Müller and Brodeur, 2002).

7.3.11 SPATIAL HETEROGENEITY AND PREDATOR-PREY MODELS

One of the most important conceptual advances has been the realisation of the importance of spatial heterogeneity in the dynamics of predator-prey and parasitoid-host interactions. It is now well established from deductive modelling that direct density-dependent relationships from patch to patch, resulting from the aggregative responses of natural enemies, can be a powerful stabilising influence (Hassell, 1978, 1980b, 2000a,b). Whilst optimality theory (section 1.12) predicts that such patterns of **direct**

spatial density-dependence ought to be common (Comins and Hassell 1979; Lessells, 1985), surveys of published studies suggest that they are in fact in the minority. Examples of the opposite, i.e. **inverse spatial density-dependent** relationships, where predation or parasitism are concentrated in the lowest density patches, are just as common, as are examples with no relationship at all, i.e. **density-independent** spatial relationships (Morrison and Strong, 1980, 1981; Lessells, 1985; Stiling, 1987; Walde and Murdoch, 1988). An inverse spatial density-dependent relationship may be found if hosts or prey in high density patches are less likely to be located than those in low density patches, perhaps due to greater host or prey concealment. Price (1988) showed this for parasitism by *Pteromalus* of the stem-galling sawfly *Euura lasiolepis* on willow (*Salix lasiolepis*). Even without this concealment effect, it is still theoretically possible for parasitism among patches to be density-independent or inversely density-dependent (Lessells, 1985). A mechanistic explanation (subsection 1.2.1) for this lies in the balance between two counteracting processes (Hassell *et al*., 1985): (a) the spatial allocation of searching time by parasitoids in relation to host density per patch, and (b) the degree to which exploitation is constrained by a relatively low maximum attack rate per parasitoid within a patch. Inverse density-dependent parasitism can theoretically result from insufficient aggregation of searching time by female parasitoids in high density patches to compensate for any within-patch constraints on host exploitation imposed per parasitoid by time-limitation, egg-limitation or imperfect information on patch quality. Density-independent relationships, on the other hand, can result if processes (a) and (b) are in balance.

These observations would appear to undermine the importance of spatial density-dependence as a regulating factor in natural predator-prey and parasitoid-host systems, but Hassell (1984) was able to demonstrate, using the approach encapsulated in equations (7.27) and (7.28) above, that patterns of inverse spatial density-dependence can be just as stabilising as patterns of direct spatial density-dependence; whether direct or inverse relationships have the greater effect depends upon the characteristics of the host's spatial distribution (Hassell, 1985; also Chesson and Murdoch, 1986). In fact, it is now apparent that even where parasitism between patches is density-independent, the spatial distribution of parasitism, if sufficiently variable, can also promote stability (Chesson and Murdoch, 1986; Hassell and May, 1988). The biological interpretation seems to be that as long as some patches of hosts or prey are protected in **refuges** from natural enemy attack, whether in high density or low density patches, stability remains possible. This may be so, but we should exercise caution in making instant biological inferences from the behaviour of such models – other interpretations may be equally plausible or preferable (McNair, 1986; Murdoch and Reeve, 1987).

The conclusion derived from deductive models that different spatial patterns of parasitism have a powerful stabilising potential opened up a whole new area of study and debate in population ecology. Consequently new methods for studying spatial patterns of parasitism in the laboratory and field began appearing. These are now reviewed in turn before we address some of the more contentious issues surrounding the subject.

Detecting Spatial Density-Dependence

Laboratory Methods

The general methodology for studying spatial variation in parasitism or predation in the laboratory is described in subsection 1.14.2. Hassell *et al*. (1985) provide a good example of how two species of parasitoid attacking bruchid beetles *(Callosobruchus chinensis)* in experiments can show inverse spatial density-dependence. The beetle itself is a pest of legumes and commonly breeds in stored dried pulses. Black-eyed beans *(Vigna unguiculata)* were used in these experiments, which were carried out in clear perspex arenas (460 mm × 460 mm × 100 mm). Twenty-five patches of equal size were marked out on white paper sheets in an hexagonal grid, with 75 mm spacing between the centres. Differ-

ent densities of beans containing 13-day-old hosts were allocated at random to patches and the number of beans in each patch made up to 32 with the addition of the required number of uninfested beans. Twenty-five parasitoids of one species were introduced into each arena and, after 24 hours, infested beans transferred to vials to await parasitoid emergence. The variable pattern of density-dependence found might be explained by both: (a) the allocation of parasitoid searching time in patches of different host density, and (b) the maximum attack rate per parasitoid constraining the extent of exploitation within patches. Parasitoids showed no tendency to aggregate in some patches over others, while their maximum attack rates per patch were limited by handling time constraints (see above) (Hassell *et al*., 1989b give further details; see both papers for methods of culturing the beetle and its parasitoids).

Field Methods

Traditional techniques for analysing field population data for density-dependence seldom take account of spatial variation in patterns of mortality. The Varley and Gradwell method (subsection 7.3.4), for example, explores variations in *k*-mortalities over several generations, but takes no account of spatial variation amongst subunits of the population within a generation (Hassell,1987; Hassell *et al*., 1987). Stiling (1988) surveyed 63 life-table studies on insects of which about 50% failed to detect any density-dependence acting on the populations. Hassell *et al*. (1989a), however, argued that density-dependence may still have been present but undetected due to: (a) the inadequate length of time over which some studies were conducted, and (b) the inability of the analyses to take account of spatial variations in patterns of mortality amongst subpopulations.

If traditional methods are inappropriate, how should we go about detecting spatial density-dependence in the field? The first problem is to decide on a suitable spatial scale (e.g. leaf, twig, tree) for the collection of data. The most appropriate scale is that at which natural enemies recognise and respond to variations in host density (Heads and Lawton, 1983; Waage, 1983, also sections 1.5 and 1.12). As this may be difficult to determine initially, samples are best taken in a hierarchical manner, so that analyses can be carried out at a number of different scales afterwards (Ruberson *et al*. 1991). Within each level of patchiness, patch density can then be related by regression analysis to percentage mortality or *k*-value, although the statistical validity of regression in this context is questionable (subsection 7.3.4). Hails and Crawley (1992) proposed an alternative logistic regression analysis based on generalised linear interactive modelling. Using the cynipid wasp, *Andricus quercuscalicis*, which forms galls on Turkey oak, as a test system, the method was able to detect spatial density-dependence in 15% of cases, with 66% of those being inversely density-dependent. Hails and Crawley manipulated patch densities by controlling the oviposition of adults on the buds of the trees. A similar study was carried out by Cappuccino (1992), who manipulated densities of another gall-making insect, the tephritid fly *Eurosta solidaginis*. Spatial variation in predation of the fly by a beetle, *Mordellistena* (Mordellidae), was noted at three scales, together with parasitism of the beetle. Interestingly, spatial variation in beetle parasitism depended, not on beetle patch density, but on the density of the fly.

Pacala *et al*. (1990) and Hassell *et al*. (1991) showed, again using the simple deductive models of host-parasitoid systems, that the contribution of spatial heterogeneity in parasitism to stability can be assessed using a simple rule. This states that the **coefficient of variation squared (CV^2)** ($=$ [variance/mean]^2) of the density of searching parasitoids close to each host must exceed approximately unity for the heterogeneity in parasitism to stabilise the interaction, i.e. $CV^2 > 1$. Moreover, CV^2 can be partitioned into the component of heterogeneity that is independent of host density (C_i) and the component that is dependent on host density (C_D), such that $CV^2 = C_i C_D - 1$. To estimate CV^2 directly the local density of searching parasitoids needs to be known. In most field systems, however, this is

impracticable and consequently little information on this parameter is available (Waage, 1983). However, a considerable body of information is already available on percentage parasitism as a function of local host density and a procedure was provided by Hassell and Pacala (1990) to estimate the relevant parameters required from these data. The reader is warned, however, that the calculations require some mathematical facility, and the procedure is only applicable to restricted types of host-parasitoid interaction, as Hassell and Pacala were careful to point out. Readers interested in applying the technique should consult Pacala *et al*. (1990), Pacala and Hassell (1991), Hassell *et al*. (1991) and Hassell (2000b).

While Hassell and Pacala (1990) provide a detailed account of how to derive CV^2 from field data using 65 examples, it needs to be pointed out that most studies on spatial distribution of parasitoids have only been conducted over a very short time-span (e.g. one generation) (reviews by Lessells, 1985; Stiling, 1987; Walde and Murdoch, 1988; Hassell, 2000b). Observed spatial distribution patterns may not therefore be typical of the interaction (Redfern *et al*., 1992), and data collected over a number of generations or years is more likely to give a representative picture. Redfern *et al*. (1992) studied patterns of spatial density-dependence amongst parasitoids of two tephritid fly species over a period of seven years. CV^2 values were calculated for total parasitism and each parasitoid species separately. The CV^2 values (together with their statistical significance) were found to be highly variable from year to year, making it difficult to draw conclusions. What drives fluctuations in CV^2 from generation to generation is unclear, but will need to be fully elucidated, if techniques such as the CV^2 rule are to have wider applicability. A further problem with the method is that it only applies to interactions where parasitism is of overriding importance and other regulating effects upon the host are negligible. How the latter might affect the stability conditions of the interaction are unclear and may be a major limitation of the technique. A recent review and assessment of the debate is given by Hassell (2000b).

Spatial Density-Dependence Versus Temporal Density-Dependence in Regulation

As spatial (i.e. within-generation) density-dependence or heterogeneity in the pattern of natural enemy attack appears to be a potentially powerful stabilising mechanism, we need to examine its relationship with temporal (i.e between-generation) density-dependence, upon which conclusions about regulation have traditionally been based. We have already seen that conventional methods for the analysis of life-table data are unsuitable for the detection of regulation resulting from spatial density-dependence (Hassell, 1985), so that many previous conclusions regarding the failure of natural enemies to regulate in particular systems may eventually need to be revised. Hassell (1985) postulated that much of the regulation in natural populations is likely to arise from within-generation variation in parasitism and predation, with only weak dependence on between-generation variation.

Not all authors have agreed with this view, however. Dempster and Pollard (1986) have argued that regulation must ultimately depend on temporal density-dependence and should therefore be detectable, at least in principle, by conventional analyses. They doubted that spatial density-dependence leads necessarily to temporal density-dependence. This raises interesting questions about the relationship between spatial and temporal density-dependence and the way the former operates. For regulation to occur, some temporal feedback is required. Using the discrete generation model of DeJong (1979), Hassell (1987) was able to show that, in the absence of stochastic variation, spatial density-dependence acting within generations translates directly into temporal density-dependence acting between generations. With variability added to the parameters governing spatial distribution and survival, however, temporal density-dependence becomes obscured and is less likely to be detected by the conven-

tional method of plotting mortality against population density from generation to generation. Murdoch and Reeve (1987) also point out that we should not necessarily expect to detect spatial density-dependence from life-table data on prey or host populations, as spatial density-dependence acts on the *natural enemy* population, not the host or prey. The authors demonstrated their point by a closer analysis of the terms of the Nicholson-Bailey model which indicate that stability results from a decline in parasitoid efficiency as parasitoid density increases. Unless life-tables also contain data for the relevant natural enemies, then this information would be overlooked.

Hassell *et al*. (1987) analysed 16 generations of life-table data for the viburnum whitefly *(Aleurotrachelus jelinekii)* and found no evidence for temporal density-dependence. Spatial density-dependence of egg mortality between leaves was apparent in eight generations. Again using a variant of the DeJong model to simulate the whitefly population, Hassell *et al*. (1987) claimed to show that this spatial density-dependence could regulate the population in the absence of temporal density-dependence. Stewart-Oaten and Murdoch (1990), however, disputed this conclusion, arguing that the DeJong model has implicit temporal density-dependence which Hassell *et al*. (1987) had overlooked. With this temporal density-dependence removed, Stewart-Oaten and Murdoch were able to show that the spatial density-dependence in the resulting model can indeed lead to stability, but that destabilisation is also a strong feature at low population levels and when whitefly clumping increased. The Stewart-Oaten and Murdoch model thus demonstrates the important point that stability in population models is often sensitive to both changes in parameter values and to any subtle variations incorporated (Murdoch and Stewart-Oaten, 1989; Reeve *et al*., 1989).

We have tried to provide a brief overview of the subject of spatial heterogeneity and aggregation in predator-prey systems Much of the progress made in the study of their roles is directly attributable to the use of deductive models, but as we have seen, the conclusions which can be derived from these models are, to some extent, dependent both on how the models are constructed and on the parameter values used. Paradoxically, while progress using this approach can be rapid, with new ideas and hypotheses being generated, often as many questions are raised as are answered. Thus, much of the subject remains speculative at his stage, and firm conclusions regarding the precise role of spatial heterogeneity in population dynamics remain elusive.

Metapopulation dynamics

Any consideration of spatial heterogeneity in population dynamics inevitably impinges on the concept of the metapopulation, which was introduced and defined in section 6.1. To recap briefly, **local populations** can be defined as units within which local population processes (e.g. reproduction, predation etc.) occur, and within which movements of most individuals are confined. **Regional populations** (or **metapopulations)** are collections of local populations linked by dispersal. Between-patch variations in parasitism and predation, as discussed above, are deemed to influence dynamics at the local population level, but regional metapopulation effects may also be important, although the distinction between such 'within-population' and 'between-population' processes may be somewhat artificial in many systems (Taylor, 1990).

Dispersal between local populations has frequently been proposed to account for the persistence of regional populations despite unstable fluctuations or extinctions at the local level (DeAngelis and Waterhouse, 1987, and Taylor, 1990, give reviews). For predator-prey systems, two different approaches have been used to model this situation (see Taylor, 1990, for details):

1. Those in which extinctions and recolonisations of local 'cells' (= populations) occur frequently – the so-called **cell occupancy models**;
2. Those in which within-cell dynamics are described explicitly by standard predator-prey models.

These models consistently show that persistence at the regional level can be enhanced by dispersal between local populations, provided that: (a) populations fluctuate asynchronously between cells, (b) predator rates of colonisation are not too rapid relative to those of the prey, and (c) some local density-dependence is present. While the degree of density-dependence may be quite low, resulting in frequent local extinctions, the metapopulation may persist for a long time (Hanski *et al*., 1996; Kean and Barlow, 2000). These conclusions are consistent with the results of a number of laboratory studies exploring the effects of spatial structure and dispersal on persistence of predator-prey systems (Huffaker, 1958; Pimentel *et al*., 1963; Holyoak and Lawler, 1996; Holyoak, 2000; Bonsall *et al*., 2002). Pimentel *et al*. (1963), for example, examined the interaction between a parasitoid wasp and its fly host in artificial environments consisting of small boxes connected by tubes. The interaction persisted longer with more boxes and with reduced parasitoid dispersal. Bonsall *et al*. (2002a) developed a similar system of interconnecting boxes (Figure 7.41) to study a bruchid beetle-parasitoid metapopulation interaction, with comparable results. While agreement with theory may be encouraging, the small scale on which these experiments, by necessity, have to be carried out, is unlikely to reflect processes at the regional metapopulation level. The results may be equally well explained by local population, between-patch, spatial dynamics. Similarly, Murdoch *et al*. (1985) invoked metapopulation processes to explain persistence of a number of field predator-prey and parasitoid-host systems, despite apparent local extinctions. However, as Taylor (1990) points out, extinction in these examples was either not proven or occurred at a scale more consistent with local population processes.

Evidence for the importance of metapopulation processes in field predator-prey systems is still relatively scarce, despite a number of recent attempts at detection in the field (see Walde, 1994; Harrison and Taylor, 1997; Davies and Margules, 1998). One of the best-studied examples involving an arthropod predator-prey system is provided by the Glanville Fritillary butterfly, *Melitaea cinxia*, and its specialist braconid parasitoid, *Cotesia melitaearum*, occupying the Åland Islands (Finland) (Lei and Hanski, 1997, 1998). In the study area, around 1700 suitable patches of dry meadow were available for colonisation, of which several hundred were

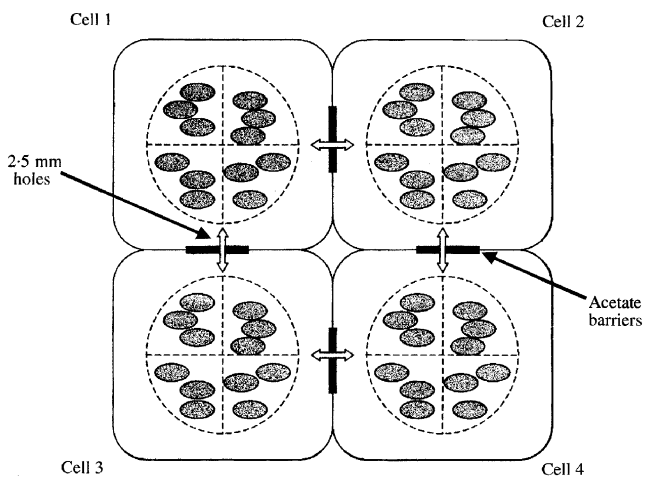

Figure 7.41 Schematic diagram of the experimental four-cell metapopulation studied by Bonsall *et al*. (2002a), Cells were linked and dispersal was restricted using acetate gates. Reproduced by permission of Blackwell Publishing.

occupied by the butterfly and about 15% by the parasitoid. As predicted by theory, the dynamics of the butterfly were greatly influenced not only by the area and isolation of patches, but also by the parasitoid, which, provided that hyperparasitism was not too high, increased the risk of local extinction in the butterfly population. Clearly, more detailed empirical studies of this nature are required to provide something of a 'reality check' to a burgeoning theoretical literature. Despite the current imbalance between theoretical and practical studies, there is no doubt that the metapopulation will remain a critical consideration for the future of population dynamics. For reviews, see Hanski (1999) and Hassell (2000a,b).

Metapopulations and Multispecies Interactions

The metapopulation concept has been extended to include multispecies interactions, including some of those described in section 7.3.10. So far, these efforts have been largely confined to relatively simple host-parasitoid systems and explored using analytical models (see Hassell *et al*., 1994; Comins and Hassell, 1996; Wilson and Hassell, 1997). As a general rule, it has been shown that adding a third species to a two-species interaction (an extra host, a competing parasitoid or a hyperparasitoid) can result in stable coexistence, although under stricter conditions than for a two-species system and always dependent on some form of 'fugitive coexistence'. Hassell (2000b) provides a detailed review.

7.4 SELECTION CRITERIA IN CLASSICAL BIOLOGICAL CONTROL

7.4.1 INTRODUCTION

In this section, we deal mainly with the criteria used in the selection of natural enemy species for introduction in classical biological control programmes (defined in subsection 7.2.2). Some of these criteria will, however, apply to agent selection in other types of biological control programme (see section 8.6, and below). We realise that the majority of readers may never actively practise biological control, but we hope that what follows will provide a framework for research such that they can nevertheless make an indirect contribution to the subject.

Several steps are involved in a classical biological control programme (Beirne, 1980; DeBach and Rosen, 1991; Waage and Mills, 1992; Van Driesche and Bellows, 1996; Neuenschwander, 2001):

1. **Evaluation** of the pest problem in the region targeted by the programme. Information should ideally be gathered on: (a) the precise identity of the target pest species and its area of origin; (b) the distribution and abundance of the pest; (c) crop yield loss and economic thresholds of pest numbers; (d) the identity and ecology of indigenous natural enemies that may have become associated with the pest in its exotic range; (e) the climatic features of the target region.
2. **Exploration** in the pest's region of origin. It should ideally involve: (a) determination, through quantitative surveys, of the composition of the natural enemy complex associated with the pest in its natural habitat(s) (see section 6.3 for methods of recording parasitism). Samples of parasitoids and predators should be taken from areas of low, as well as high, pest abundance; (b) quantification of the impact of these natural enemies on populations of the pest (see sections 7.2 and 7.3 for methods); (c) determination of their degree of specificity, i.e. their host/prey ranges (see section 6.3 for methods). Exploration may also involve determining which natural enemies are associated with taxonomically related pests (the search for 'new-associations', see below). Of the range of species available, one or more are chosen on the basis of their potential to control pest populations; in a typical classical biological control programme, only a small fraction of the natural enemy complex of a pest is chosen for future study (i.e. the biological control practitioners apply selection criteria even at this early stage in the programme – **first stage selection**). The constraints upon selecting a larger fraction include: (a) the limited

resources (finances, time) available to practitioners; (b) the unsuitability of certain species due to their polyphagous habits (which may result in non-target effects, see 7.4.4, below) or their potential to interfere with other enemy species (facultative hyperparasitoids, intraguild predators); (c) the difficulty sometimes encountered, in multiple parasitoid introductions, of achieving establishment of a species, following establishment of another (Ehler and Hall, 1982; Waage, 1990; Waage and Mills, 1992; see below). If 'new associations' are to be employed, then one or more members of the natural enemy complex of a species taxonomically related to the pest, but not of the complex associated with the pest itself, will be selected in a programme.

3. **Quarantine**. This involves maintaining the chosen agents in an escape-proof facility in the target (i.e. non-native) region. One of the purposes of quarantine is to eliminate any associated hyperparasitoids, parasitoids of predators or insect and plant pathogens that have inadvertently been introduced into cultures along with the agents. Further selection of agents may occur during this phase, based on ease of handling and culturing and on other criteria (see below) (**second stage selection**).
4. **Release** of suitable agents occurs after clearing through quarantine. A multidisciplinary approach is required here, involving skills in population ecology, population genetics, meteorology, engineering and sometimes even aeronautics (agents may be released by aircraft).
5. **Monitoring** then takes place (ideally, monitoring of the host population should commence before any agents are introduced; see sections 7.2 and 7.3 for discussion of monitoring methods). This requires knowledge of pest and crop life-histories and phenologies, as well as expertise in population dynamics. Evidence should be sought of the successful establishment of agents (e.g. see Brewer *et al*., 2001): crops and associated vegetation needs to be scrutinised, and signs of predation and parasitism looked for. A pest sampling programme should be carried out, to determine what (if any) effects the introduced agents have had/are having on pest population dynamics and demography. The use of control plots is essential, as correlation does not mean causation (see subsection 7.2.2).
6. **Evaluation** involves assessing the degree of attainment of the programme aims and objectives, asking the following questions: (a) has the introduction proved successful in controlling the pest? (b) has control been partial, substantial or complete (*sensu* DeBach, 1971)? (c) has the programme been cost-effective (economic analysis, e.g. see Bokonon-Ganta *et al*., 2002)? (d) has the local (human) community benefited? (e) have there been any non-target effects (e.g., have the population dynamics of other organisms been affected (see section 7.4.4)?

A more detailed discussion of the steps involved in classical biological control programmes is provided by Van Driesche and Bellows (1996). Ways of improving the outcome of classical biological control programmes are discussed by Kareiva (1990), Waage and Barlow (1993), Barlow (1999), Freckleton (2000), Mills (2000) and Shea and Possingham (2000) (see also Fagan *et al*., 2002). Shea and Possingham (2000) applied stochastic dynamic programming, linked to a metapopulation approach, in identifying optimal release strategies (number and size of releases), and they derived useful rules of thumb that can enable biological control workers to choose between management options. Van Lenteren *et al*. (2003) discuss the data requirements for assessing establishment by imported natural enemies.

7.4.2 WHAT WORKED (OR DID NOT WORK) PREVIOUSLY – THE USE OF HISTORICAL DATA

The history of biological control shows that it has been largely an 'art', aided by the knowledge of what worked successfully in the past either against the same pest species or a taxonomically related species (van Lenteren, 1980; Waage and Hassell, 1982). Biological control is

becoming much more of a science, and historical databases, although deficient in many respects (see below), can still prove useful to practitioners in helping them both to make generalisations and to erect hypotheses, informed by ecological theory (Greathead, 1986; Waage and Greathead, 1988). The principal database of classical biological control introductions is BIOCAT, developed at the CAB International Institute of Biological Control (now part of CABI Bioscience). It is the most comprehensive database on biological control developed to date (Greathead and Greathead, 1992). Unfortunately, like other databases, it is composed of records whose reliability is in some cases questionable. As Waage and Mills (1992) point out, ''the record of classical biological control is troubled by erroneous identifications of pests and natural enemies, occasional errors in dates and places and the arbitrary (and sometimes entirely incorrect) interpretation of success''.

Nevertheless, analyses carried out with the BIOCAT database are providing useful insights into the habitat-, pest- and natural enemy-related factors that influence the success of natural enemy introductions (e.g. see Greathead, 1986; Mills, 1994; Jervis *et al*., 1996a; Hawkins and Cornell, 1994; Kidd and Jervis, 1997; Hawkins *et al*., 1993, 1999). For example, the results of analyses tell us that introductions are particularly successful against Homoptera and Lepidoptera (Greathead, 1986; Mills, 2000), and that success rates are substantially greater in exotic, simplified, managed habitats than in more natural habitats, particularly when involving parasitoids (Hawkins *et al*., 1999). The database has also been widely used to test ecological hypotheses concerning enemy-victim interactions (e.g. see Hawkins, 1994; Kidd and Jervis, 1997; Mills, 2001), including the hypothesis that the action of natural enemies in classical biological control is not fundamentally different from that of indigenous natural enemies (Hawkins *et al*., 1999) (see subsection 7.2.2).

For details of how analyses of the BIOCAT database are conducted (including ways of minimising bias), see Stiling (1990), Hawkins (1994), Mills (1994), Jervis *et al*. (1996a), Kidd and Jervis (1997) and Hawkins *et al*. (1999). Comparative statistical techniques (subsection 1.2.3) should be employed to overcome pseudoreplication.

7.4.3 NATURAL ENEMY ECOLOGY AND BEHAVIOUR

Introduction

During the exploration phase of a biological control programme, a decision will need to be made as to whether the natural enemies are to be collected from the pest species or from other, taxonomically closely related, species. The **theory of new associations** (Pimentel, 1963; Hokkanen and Pimentel, 1984) states that natural enemy-pest interactions will tend to evolve towards a state of reduced natural enemy effectiveness, and that natural enemies not naturally associated with the pest (i.e. species presumed to be less coevolved with the target pest) either because they do not come from the native area of the pest or because they come from a related pest species, may prove more successful in biological control. Hokkanen and Pimentel (1984) analysed 286 successful introductions of biological control agents (insects and pathogens) against insect pests and weeds, using data from 95 programmes, and concluded that new associations were 75% more successful than old associations. However, the validity of this conclusion was called into question by more refined analyses (Waage and Greathead 1988; Waage, 1990). The latter showed that new associations can be as effective as old ones once the natural enemy is established, but that establishment of a natural enemy on a new host is very difficult. Therefore, in practice, studies on the target pest in its region of origin remain the most promising approach to finding an effective biological control agent rapidly, but the potential usefulness of new associations should also be considered (Waage and Mills, 1992).

If only a fraction of the natural enemy complex of a pest can be used in classical biological control, it is essential that the potentially most important and least 'risky' species (i.e. in terms of their potential impact on pest populations

and their threat to non-target organisms, respectively) are used from among the candidates available (Waage and Mills, 1992). Decisions on the relative merits of natural enemies can be based on **reductionist criteria** and **holistic criteria** (Waage, 1990). As we shall see, many of these criteria are based mainly or entirely on theory. Gutierrez *et al*. (1994) question the reliance of biological control practice on theory, arguing that the latter has contributed little either to increasing the rate of success of introductions or to an understanding of the reasons for failures (see also Kareiva, 1990, and Barlow, 1999). As we have already mentioned, ecological theory has provided useful insights into the performance of past biological control programmes, identifying some factors that influence success and failure, and it has provided guidance for future programmes. A decade on, Gutierrez *et al*.'s point concerning the contribution of ecological theory to biological control practice remains a valid one, but this is not to say theory should be discarded. Instead, ways should be sought of applying theory more effectively, so that the record of success in classical biological control programmes can be improved. Mills (1994) estimated that of 1,450 unique pest-introduced parasitoid combinations, only 38% resulted in establishment (i.e. successful colonisation) of the agent, and of 551 parasitoid introductions that have resulted in establishment and have provided some degree of control, only 17% have been successful – clearly, there is considerable room for improvement in classical biological control. Constraints upon the contribution of ecological theory to biological control, and some ways of overcoming them, are considered below.

Reductionist Criteria

Introduction

The reductionist approach involves selecting agents on the basis of particular biological attributes. Reductionist criteria are mostly derived from the parameters of the analytical parasitoid-host or predator-prey population models discussed in 7.3, in particular those parameters which are important to the lowering of host or prey population equilibria and/or which promote population stability. Stability of the natural enemy-pest interaction is seen as desirable: (a) because it reduces the risk that the pest population will be driven to (local) extinction by the control agents, which themselves would otherwise become locally extinct; and (b) it reduces the likelihood of the pest population exceeding the economic threshold.

Despite their frequent use in theoretical studies, reductionist criteria have rarely been used in practice, one reason being the difficulty of estimating parameter values (Godfray and Waage, 1991). As Waage and Mills (1992) point out, laboratory measures of parameters are unlikely to reflect the values of parameters in the field, where the environment is more complex (although, as shown in Chapter 1 of this book, with a little thought, experiments can in some cases be designed which take environmental complexity more into account).

A further criticism that can be aimed at the reductionist approach concerns the validity of separating a parasitoid's or predator's biology into components, and of assuming that there are natural enemies that have 'ideal combinations' of such components (see below). The whole organism, not its component parts, is what forms the basis for prediction of success in biological control (Waage and Mills, 1992). However, models have been developed (see subsections 7.3.7, 7.3.8) in which biological attributes are assembled in a more realistic fashion (Hassell, 1980a; Murdoch *et al*., 1987; Gutierrez *et al*., 1988a, 1994; Godfray and Waage, 1991). Even so, a major difficulty with many models is that a burdensome number of parameters has to be estimated in order for the model to be operated. A notable exception to this is the Godfray and Waage (1991) model of the mango mealybug-parasitoid system, which incorporated a few, easily measurable parameters such as the stage of the host attacked by the different parasitoids *(Gyranusoidea tebygi* and *Anagyrus* sp.), age-specific development rates for host and parasitoids, age-specific survivorship of hosts in the field, adult longevities and daily

oviposition rates. Some parameters such as searching efficiency were more difficult to measure, and so a range of realistic values was tested in each case using sensitivity analysis. Godfray and Waage (1991) categorise their model as one of intermediate complexity, in that it is more complex than the analytical models of theoretical ecology e.g. the Nicholson-Bailey model (subsection 7.3.7) but less complex than many detail-rich simulation models, e.g. that of Gilbert *et al*. (1976) (subsection 7.3.8).

Godfray and Waage's (1991) was a **prospective model** (i.e. constructed prior to introduction) as opposed to a **retrospective model** (i.e. constructed following introduction). Prospective models are a potentially very useful tool in decision-making in biological control (Godfray and Hassell, 1991), but an important constraint upon their development and use in biological control programmes is the need for practitioners to achieve control of the pest as rapidly as possible (Waage, 1990). Mills and Gutierrez (1996) devised a prospective model for the dynamics of a heteronomous hyperparasitoid in a cotton-whitefly parasitoid-system.

Thus, selection of agents based on comparisons of species' attributes will probably never be a top priority; indeed so far it has been very rare for candidate species available in culture and cleared through quarantine, *not* to be introduced (Waage, 1990). Nevertheless, pre-introduction studies on candidate species, aided by modelling, still have a significant contribution to make to classical biological control programmes; at the very least they could bring about a re-ordering of the sequence of introduction of species destined for release, i.e. help to move the more effective agents to the front of the queue. Bearing in mind that programmes usually end before all candidate agents have been released, prioritising agents on the basis of their likely efficacy would ensure that the 'best' species are released (Waage, 1990).

Before we go on to discuss which attributes of candidates are considered to be particularly desirable, it is important to remind the reader that most biological control models are based on equilibrium population dynamics, and that in using such models, one seeks to determine to what extent a low, stable pest equilibrium can be achieved. The assumption that low, stable host equilibrium populations result in good pest control was questioned by Murdoch *et al*. (1985) who argued that, instead of considering biological control in terms of local population dynamics, theoreticians and practitioners should view it terms of the more biologically realistic scenario of metapopulation dynamics (subsection 7.3.10). By doing so, local instability – and thus a high risk of extinction of local populations – is not necessarily a problem for biological control practitioners, as persistence may be possible in the metapopulation. Note that whereas successfully controlled pests often appear to fluctuate because of local extinctions (Luck, 1990; *Aonidiella aurantii* being a notable exception, see Murdoch *et al*., 1985), metapopulation persistence is perceived to be generally the case following successful natural enemy establishment. Furthermore, there is no evidence that any failure in biological control has been the result of a lack of the persistence of a successfully established agent (Waage, 1990). However, even if metapopulation dynamics ensure persistence, local instability can remain a pest management problem: high temporal variation in pest densities increases the risk of the pest population exceeding the economic threshold (see Murdoch, 1990). The level of this risk depends on the degree to which the host population is suppressed by the biocontrol agent: if the degree suppression is such that pest numbers, despite fluctuating greatly, never exceed the economic threshold, then high temporal variation in pest numbers can be acceptable; indeed, successful pest control is theoretically possible through suppression alone (Kidd and Jervis, 1997).

Another point concerning the equilibrium population dynamics basis of most biological control models is that the models will be more appropriate for some pests than for others (Godfray and Waage, 1991). In some agroecosystems, e.g. arable crops and glasshouse systems, cultural practices and/or seasonal factors will prevent the population ever coming close to equilibrium, i.e. **transient dynamics** will per-

tain. Thus, some of the selection criteria discussed below have little or no relevance to certain crop systems. Even in classical biological control, it is questionable whether biological control introductions ever attain equilibrium, at least at the local population level (Murdoch *et al*., 1985), and thus a measure of the **transient impact** of an introduced parasitoid may be more appropriate for predicting control potential (Hochberg and Holt, 1999; Mills, 2000, 2001).

Biological control models have mainly been of the deterministic analytical type. Murdoch *et al*. (1985) suggested the use of **stochastic boundedness models** as a preferable alternative. With the latter, emphasis is placed on estimating the probabilities that either host or parasitoid (or prey and predators) may become extinct, or that the pest exceeds an economic threshold, rather than on stability analysis (Chesson, 1978b; 1982).

Listed below are several attributes (some of which are common model parameters) of natural enemies considered to be among the most desirable for biological control, based on theoretical modelling, practical considerations and past experience. Implicit to the reductionist approach is the notion that any combination of life-history parameters is possible (Waage, 1990). However, it is becoming increasingly apparent, through studies on natural enemies, that there are trade-offs between one attribute and another, for example, that between adult reproductive capacity and larval competitive ability in parasitoids (see **Holistic Criteria)**. It would therefore be better to concentrate on 'suites' of often counterbalancing attributes. Waage argues that for reductionist criteria to be useful, they need to be derived at a higher level where the traits are integrated in the patterns or strategies that we observe in nature, and he cites one approach as being the computation of intrinsic rates of natural increase (r_m) (see section 2.11 for methods).

High Searching Efficiency

The density responsiveness of candidate biological control agents has been compared through short-term (24 hour) functional response experiments (see section 1.10 for design). For example, if one is comparing the potential effectiveness of two parasitoid or predator species, the species with the higher maximum attack rate (which is set by handling time and/or egg-limitation) may be selected, as it will, all else being equal, depress the pest equilibrium to a greater extent. Sigmoid functional responses, because they result in density-dependent parasitism or predation, are potentially stabilising at low pest densities. However, in a coupled interaction responses have to be very pronounced, and the destabilising time-delays in the population interaction small, for the stabilisation to be marked (Hassell and Comins, 1978; subsection 5.3.7). Using the BIOCAT database, Fernández-Arhex *et al*. (2003) tested for, but were unable to detect, a relationship between the form of the functional response and success in classical biological control (Type 2 versus Type 3).

As pointed out by Waage and Greathead (1988), the natural enemy functional response offers a good conceptual framework for understanding the action of agents in inundative releases. Ratio-dependent functional responses (subsection 7.3.7) are now being considered as more relevant (Mills and Lacan, 2004).

Spatial Heterogeneity in Natural Enemy Attack

There are two issues here: (a) the degree to which a refuge from parasitism contributes to host suppression, and (b) the degree to which a refuge contributes to population stability. The following discussion refers to proportional refuges (which are generally thought to be more realistic approximations of variation in risk to parasitism than constant number refuges, see Hassell, 1978, 2000b, and Holt and Hassell, 1993). Note that the refuge can be an attribute of the host (or its food plant), rather than of the parasitoid (see references in Mills and Getz, 1996), but that even if the former applies, other parasitoid characteristics can determine the population dynamic effects of the refuge.

Concerning suppression, whatever the nature of the refuge, if it is very large, most of the hosts

can escape from parasitism, and so the impact of the parasitoid population on the host population will be small no matter what other attributes the biological control agent may possess (Hochberg and Holt, 1999; Mills, 2000, 2001). Hawkins *et al*. (1993), through a BIOCAT database analysis, provided evidence to support the view that the larger the refuge from parasitism, the lower the probability of success in classical biological control. In this case, the maximum level of (percentage) parasitism achieved in the target region was used as the measure of refuge size (for a critique, see Myers *et al*., 1994).

Mills (2001) showed that the ability of a parasitoid to suppress a host population depends on the size of the host refuge from parasitism, the host net rate of increase (F in the Nicholson-Bailey model) and on whether the parasitoid is egg-limited (limited in its attacks by the number of eggs it has available for laying) or 'host-limited' (i.e. limited in its attacks by the female's ability to find hosts) (for a model, see Mills' paper). Even in the absence of a refuge, an egg-limited parasitoid will be unable to suppress the abundance of a host if the latter has a high F. However, such a parasitoid can suppress host abundance substantially if the host has a sufficiently low F and a minimal refuge from parasitism. [A sufficiently low F could, theoretically, be achieved by the use of partially resistant crop cultivars]. With a host-limited parasitoid, substantial host suppression can occur when the refuge is sufficiently small in relation to F (Hochberg and Holt, 1999).

Concerning the effects of refuges on population stability, spatial heterogeneity in parasitism, through its host refuge effect, is recognised as being one of the major stabilising factors that can lead to the persistence of parasitoid-host and predator-prey populations in models of parasitoid-host interactions (subsection 7.3.10) (but see Murdoch and Stewart-Oaten, 1989). This applies both to aggregation in patches of high or low host or prey density and to aggregation in patches independent of host or prey density. Heterogeneity in parasitism, whether the result of aggregation or other (i.e. host- and/or habitat-related) factors, can have a stabilising effect at even the lowest population levels, unlike host resource limitation or mutual interference (Hassell and Waage, 1984; May and Hassell, 1988) (see subsection 7.3.7).

Because of its stabilising potential, parasitoid aggregation has been promoted as a primary selection criterion for parasitoids (Murdoch *et al*., 1985 and Waage, 1990), but it is debatable whether it ought to be promoted as an independent criterion. Firstly, there is the question of whether the operation of a local stability-promoting factor, of whatever kind, is necessary for successful biological control (see above). Secondly, refuges have generally been shown in models to raise equilibrium population levels (Murdoch, 1990). Not only in the case of refuges but also with regulating factors such as density-dependent sex ratio, there is a strong trade-off between the degree of stability and the degree of host suppression. This phenomenon, has been termed the **paradox of biological control** (Luck, 1990; Arditi and Berryman, 1991). However, Arditi and Berryman (1991) showed that the paradox can be resolved with the Lotka-Volterra model if a ratio-dependent functional response is assumed (see subsection 7.3.7). While ratio-dependent functional responses have been disputed to occur in nature (see Abrams, 1994; Murdoch, 1994) there is evidence that they can occur in some parasitoids (including species attacking hosts whose dynamics can be described by Lotka-Volterra type rather than Nicholson-Bailey type models) (Hoddle *et al*., 1998; Jones *et al*., 1999; Faria *et al*., 2000; Hoffmann *et al*., 2002; Mills and Lacan, 2004), although they may be a highly constrained trait, phylogenetically speaking (Mills and Lacan, 2004). The question remains of how the paradox of biological control can be resolved through the Nicholson-Bailey modelling framework.

The implications of natural enemy aggregation for biological control have generally been considered at the local population level. Ives and Settle (1997), however, employing a metapopulation model, found that as predator aggregation increases in fields in which pest abundance is

high, pest equilibrium densities increase. This is because high-pest density fields are late in the pest population trajectory, so predators have less of an effect on the maximum pest density achieved, and these fields retain predators that could be more effective, in pest control terms, by moving to more recently infested fields.

Despite the attention that spatial pattern of attack has received from researchers, there is very little empirical information relating to the spatial patterns of attack by natural enemies used in biological control (Mills, 2000). Exceptions include parasitoids attacking California Red Scale (*Aonidiella aurantii*). Murdoch and co-workers (Murdoch, 1994; Murdoch *et al.*, 1996) carried out experimental manipulations of both the distribution and the abundance of the scale insect on individual citrus trees, and concluded that the spatial heterogeneity in parasitoid attack that characterises this agent-pest system did not account for either local stability or the success of reduction in scale abundance.

Temporal Heterogeneity in Natural Enemy Attack

For parasitoids and predators, hosts and prey are a temporally heterogeneous resource, altering in both vulnerability and quality as they pass through the various stages in their life-cycle. Parasitoids respond to this, practising differential allocation of progeny (sex ratio, clutch size) (Murdoch *et al.*, 1987, 1992, 1997; Godfray, 1994; Briggs *et al.*, 1995; subsection 1.5.7). Murdoch *et al.* (1997) argue, from modelling of stage-dependent parasitoid oviposition behaviour, that these parasitoid responses have a common outcome: stabilising delayed density dependence in the *per capita* recruitment rate of the parasitoid population. Size-selective destructive host feeding (Kidd and Jervis, 1991) reinforces the delayed density dependence in *per capita* recruitment rate that is induced by size-selective clutch size allocation (for evidence of the latter, see Murdoch *et al.*, 1997; but also see Murdoch *et al.*, 1992b)

Murdoch *et al.*'s (1987) stage-structured parasitoid-host model (in which either the adults or the juveniles of the pest can be made invulnerable to attack by the parasitoid) incorporates a developmental delay in both the host and the parasitoid (Figure 7.42). The stability of this model depends on the length of the parasitoid time lag, T_2, relative to the duration of the invulnerable stage, T_A. The parasitoid's time lag is destabilising; the longer the developmental period of the parasitoid is relative to that of the host, (i.e. high T_2/T_A), the more difficult it is to obtain stability. A longer development time also leads to exponential increases in the pest equilibrium. Therefore, Murdoch (1990) considered a short parasitoid development time to be a desirable

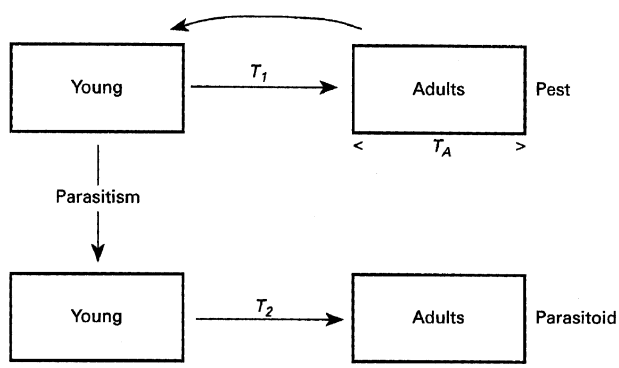

Figure 7.42 Diagram of a parasitoid-pest model in which both species have an immature stage lasting for T_1 and T_2 days respectively. The adult pest is invulnerable to attack by the parasitoid, and lives for an average of T_A days. Source: Murdoch *et al.* (1990). Reproduced by permission of Intercept Ltd.

attribute of a parasitoid species for biological control. For methods of measuring development times of parasitoids, see subsection 2.9.2.

A High Maximum Level of Parasitism in the Host's Native Range

Hawkins and Cornell (1994) using the BIOCAT database, obtained a positive, albeit 'noisy', correlation between maximum level of parasitism in the pest's country of origin, and the degree of success in classical biological control. In Hawkins and Cornell's data set, a cut-off in control outcomes exists at maximum parasitism rates of approximately 35%; below this level biological control very rarely achieves economic success (Hochberg and Holt, 1999). Although conditions in the native environment and those in the exotic one will be similar to some extent, there will be enough differences to make strict extrapolation problematical. Nevertheless, all else being equal, if a parasitoid species achieves a maximum level of parasitism many times higher than that of another, then based on Hawkins and Cornell's results, there would be a strong case for employing the former.

Kean and Barlow (2000) confirmed the relationship between parasitism level and biological control in parasitoid-host models, but they also found that if the host's r_m is low (a rare condition for many pests), substantial host reduction does not necessarily require high levels of parasitism.

A High Parasitoid Fecundity

There is no clear empirical evidence that a high parasitoid fecundity increases the probability of agent establishment, although a future analysis taking phylogenetic confounding effects into account might reveal a different result (Lane *et al*., 1999). Establishment rate declines from egg through to prepupa in parasitoids attacking Lepidoptera, and this may well be the result of the decline in fecundity observed for species attacking successively later stages in the host life cycle (Price, 1975; Mills, 1994). Establishment rate increases at the pupal stage, possibly due to the trend towards polyphagy among pupal parasitoids of Lepidoptera (they can utilise non-target host species).

Lane *et al*. (1999) and Mills (2001), through modelling, identified parasitoid fecundity (a key determinant of **attack capacity** – the maximum number of hosts that a parasitoid can attack in its lifetime, see Mills, 2001) as a major factor in pest suppression, irrespective of whether transient or equilibrium dynamics best represent the real dynamics of agent-pest interactions. This view is supported by Lane *et al*.'s (1999) literature survey, which revealed a positive correlation between attack capacity and success of classical biological control introductions against Lepidoptera (there was no correlation for parasitoids of Homoptera). Lane *et al*. also showed that, in their model (which incorporated parasitoid fecundity limitation, a refuge from parasitism, and a density-dependent host population growth function) a high fecundity should provide stable control of a host population over a wider range of parameter space. Lane *et al*. stress, however, that low fecundity should not always be considered to be a disadvantage in a biological control agent (see their paper for details).

Gregariousness/Number of Female Parasitoids Produced Per Clutch

Parasitoid gregariousness, or the number of female parasitoids produced per clutch, has been identified as a major factor in pest suppression (Mills, 2001). Even small increases in gregariousness lead to dramatic reductions in host abundance. Support for this conclusion came from an analysis of the BIOCAT database (Mills 2001). Gregarious species were found to represent a significantly greater proportion of parasitoid introductions that have led to success than of those that have led to failure.

A High Degree of Seasonal Synchrony With the Host

Populations of hosts and parasitoids with discrete generations frequently show imperfect phenological synchrony, with the result that

some host individuals experience a reduced or even zero risk of parasitism, i.e. there is a temporal refuge effect. Compared with perfect synchrony, imperfect synchrony will result in a raising of the host equilibrium level. Models developed by Münster-Swendsen and Nachman (1978) and Godfray *et al*. (1994) have shown that it will also stabilise the parasitoid-host population interaction.

The degree of 'phenological matching' between parasitoid and pest can be investigated in the laboratory, by subjecting the insects to a range of temperatures and/or photoperiods (Goldson and McNeill, 1992). Parasitoid populations taken from different localities from within the host's native range may show different diapause characteristics and therefore will show different degrees of synchrony with pest populations that originate from only one locality.

Seasonal synchrony with the host is not an important selection criterion in biological control programmes involving inoculative release and inundative release, since synchrony can be achieved by the grower through the release of parasitoids when most hosts are in the susceptible stage for parasitism (van Lenteren, 1986).

Parasitoid Guild (Host Stages Attacked/Killed)

Mills (1994) found, through a BIOCAT database analysis of parasitoid introductions against Lepidoptera, that: (a) it is easier to achieve establishment with parasitoids that attack earlier stages of host development, although pupal parasitoids also perform well; (b) the overall success of egg parasitoids is poor (they have the highest probability of establishment – probably due to the egg parasitoid category of his database being dominated by *Trichogramma* spp., which tend to be polyphagous and therefore can utilise non-target hosts (c) the earlier the parasitoid completes development in (i.e. kills) later host stages, the higher the success rate.

A High Intrinsic Rate of Natural Increase (r_m)

As noted above, the agent's intrinsic rate of population increase has been proposed as a primary selection criterion, in that it comprises an integrated suite of natural enemy traits and is therefore biologically more realistic (but note that Neuenschwander, [2001, in reviewing the biological control programme against cassava mealybug, concluded that r_m was, in retrospect, a poor predictor of agent effectiveness in that particular case).

Modelling by Hochberg and Holt (1999) has shown that r_m (which they estimated from a partial derivative of their host refuge model) is enhanced by a greater searching efficiency, a greater attack capacity (maximum number of hosts attacked over the parasitoid's lifetime) and a greater mean number of parasitoids emerging from a parasitised host. It was also shown that that in highly productive environments (high K, the host's carrying capacity of the environment), it is parasitoid fecundity alone that determines the conversion of hosts to parasitoids and therefore the transient impact of parasitism on the host population. If the clutch sex ratio is biased towards females (as is often the case for gregarious parasitoids), then a gregarious species will have a higher r_m than a solitary species with the same fecundity (see Mills, 2001).

As pointed out by Huffaker *et al*. (1977), it is a common error to conclude that a natural enemy having a lower r_m than that of its host or prey would be a poor biological control agent. The parasitoid (or predator) need only possess an r_m high enough to offset that part of the host's r_m that is not negated by predation or parasitism (and that part not negated by other mortality the natural enemy may inflict, e.g. through host-feeding and host-mutilation, see Jervis *et al*., 1992a).

Rapid Numerical Response/Short Generation Time Ratio

The calculation of r_m values yields no insights into the dynamic interaction between natural enemy and the pest, whereas the numerical response does (subsection 7.3.7). Whilst some biological control workers have assumed a rapid numerical response to be a desirable feature of a

natural enemy, this conclusion needs some qualification. While it is likely that a slow numerical response to a pest with a high population growth rate will lead to delayed density-dependence and limit cycles (subsection 7.3.7), a rapid numerical response to slow pest population growth may result in overcompensating fluctuations and decreased stability. Therefore the potential value of a numerical response needs to be assessed in relation to the population characteristics of the target pest.

Modelling by Godfray and Hassell (1987) in relation to parasitoids, and Kindlmann and Dixon (1999) in relation to predators, has pointed to the role of the relative lengths of natural enemy and pest generation times in determining equilibrium levels. Godfray and Hassell's (1987) simulations indicate a slight raising of pest equilibrium density when the **generation time ratio (GTR**, the ratio of the natural enemy's generation time to that of its host/prey**)** is greater than one, while Kindlmann and Dixon's (1999) simulations reveal that the suppressive effect of a predator is inversely related to the natural enemy's development time (see Kindlmann and Dixon's, 1999, 2001, papers for a functional explanation). Predators are considered to be less effective than parasitoids: *q*-values (subsection 7.2.2) for introduced predators are an order of magnitude larger than those for parasitoids (Table 1, in Kindlmann and Dixon, 1999) – a difference considered to be due in large part to the difference in GTR. Note that predators are, on the whole, better at controlling long-lived pests (smaller GTR) such as scale insects than shorter-lived ones such as aphids (longer GTR) (Kindlmann and Dixon, 2001).

Mode of Reproduction

Modes of reproduction in parasitoids are discussed in section Chapter 3 (section 3.3) Stouthamer (1993) considered the merits of arrhenotoky and thelytoky in parasitoid wasps from the standpoint of classical biological control; some of his conclusions were that:

1. Arrhenotokous forms (species or 'strains') will be able to adapt more rapidly to changed circumstances. If environmental conditions in the area of introduction are different from those in the form's native range, arrhenotokous wasps may have the advantage;
2. Assuming that a thelytokous form and an arrhenotokous form produce the same number of progeny, the thelytokous form will (all else being equal): (a) have a higher rate of population increase, and (b) depress pest populations to a lower level (see parameter *s* in the modified Nicholson-Bailey model (subsection 7.3.7);
3. Arrhenotokous forms must mate to produce female offspring; therefore, in situations where the wasp population density is very low, males and females may have problems encountering one another (an Allee effect). Thelytokous forms are therefore better colonisers.

A reaction-diffusion model comparing arrhenotokous parasitoids with sexually reproducing diploid ones predicted that haplodiploidy permits successful establishment in parasitoid populations that are 30% smaller – diploid populations suffer more from an Allee effect (Hopper and Roush, 1993). Mills (2000) outlines a post-introduction protocol for assessing the influence of mating on parasitoid establishment (see also Hopper, 1996). It includes releasing cohorts of increasing size (released as mature pupae) in spatially replicated locations, then dissecting the resulting female parasitoids at host patches, to assess whether they have been inseminated or not (see subsection 4.4.3).

Destructive Host-Feeding Behaviour

In the literature on biological control, the occurrence of non-concurrent destructive host-feeding behaviour (in which different host individuals are used for feeding and oviposition, see section 1.7) has been given as a reason for assuming a particular parasitoid species to be a potentially effective biological control agent. This is not an unreasonable assumption to make, in view of the fact that: (a) this type of host feed-

ing behaviour is an additional source of mortality to parasitism, and (b) several parasitoid species have been shown to kill far more hosts by host-feeding than by parasitism. However, theory informs us that destructive host feeding is an undesirable attribute in a biological control agent (Jervis *et al*., 1996a):

1. With regard to *establishment rate*, one model predicts destructive host-feeders to be no better than other types of parasitoids, while another model predicts them to be worse (Jervis *et al*., 1996a; Jervis and Kidd, 1999);
2. With regard to *success rate*, destructive host feeders are predicted to be inferior, compared to other parasitoids, in suppressing host abundance, due to their lower numerical response (for a review of models used, see Jervis *et al*., 1996a, and Jervis and Kidd, 1999).

Analyses of the BIOCAT database reveal destructive host-feeders to be either just as likely or more likely to become established than other parasitoids, and to be more successful in controlling the host compared to other parasitoids. The conclusion drawn is that destructive host-feeders are as good as, and probably no worse than, other parasitoids (Jervis *et al*., 1996a).

Collier and Hunter (2001) suggest that destructive host-feeding behaviour might influence multiple agent interactions and pest suppression in biological control, where parasitoids feed on hosts that have been parasitised by a potential parasitoid competitor.

High Dispersal Capability

Techniques for studying dispersal by natural enemies are discussed in subsection 6.2.11. If a natural enemy has a high ability to disperse (either as an adult or as an immature stage within the host), then it can be expected to spread rapidly from the initial release point. Thus, fewer resources (time, money) may need to be invested in large numbers of point releases over a region to ensure that the natural enemy becomes established over a wide area. Another reason for favouring high dispersal capability in classical biological control agents is that it can minimise a time-delay in re-invasion of areas where the enemy has, for reasons of local instability, become extinct; a significant delay can allow the pest population to reach undesirable levels.

Wilson and Hassell (1997) have shown, through modelling, that demographic stochasticity increases the probability of extinction of small local populations, and that because of this, higher dispersal rates are required to ensure persistence of the metapopulation.

Fagan *et al*. (2002) suggest there may be a life-history trade-off between spatial spread rate and suppressive ability. They found evidence, albeit weak, for their hypothesis, but they do not explain how the trade-off would operate at the organismal level.

In biological control programmes that involve inundative releases of parasitoids, the parasitoids are used as a kind of 'biopesticide', so it is important that the insects do *not* disperse rapidly away from the crop. Parasitoids can be encouraged to remain within the crop either by 'pre-treating' females with host kairomones so as to stimulate search following release (Gross *et al*., 1975), by applying kairomones directly to the crop to act as arrestants (defined in section 1.5) (Waage and Hassell, 1982) or by applying non-host foods (section 8. 1) to the crop.

High Degree of Host Specificity/Absence of Intraguild Predatory Behaviour

Practical approaches to studying host specificity in parasitoids and predators are discussed in subsections 1.5.7 and 2.10.2, and section 6.3.

One explanation for the poor performance, overall, of predators compared with parasitoids in classical biological control in perennial crop systems is their tendency to be more polyphagous. Among introductions of coccinellids, success rates are higher for monophagous species than for polyphagous ones (Dixon, 2000). It is argued that, because of parasitoid or predator polyphagy, a pest cannot be maintained at low equilibrium populations, as the natural enemy will concentrate on the more

abundant alternative host or prey species (see switching behaviour, section 1.11). However, as Murdoch *et al*. (1985) point out, a polyphagous natural enemy can survive in the absence of the pest in the event of the latter's local extinction, and it can therefore be ready to attack the pest when it re-invades. For this reason, polyphagy may not be as undesirable an attribute in classical biological control as it is commonly assumed to be. However, polyphagous natural enemies pose risks to non-target organisms (see subsection 7.4.4).

An argument has been made, in relation to phytoseiid mite predators, for using those generalist predators that have a high degree of plant-specificity (McMurtry, 1992).

A lack of host specificity is not a problem in programmes aimed at pests of protected crops i.e. in glasshouses, as the environment is a simple one, usually containing unrelated pest species at each of which different indigenous parasitoid species are targeted (van Lenteren, 1986).

The general perception is that restraint should be exercised in using either: (a) facultative hyperparasitoids – these are dynamically equivalent to 'intrinsically superior' primary parasitoids (but see Rosenheim *et al*., 1995), although the predicted dynamic consequences of introducing them vary with the particular type of model used (see Table 7.4); or (b) predators (including host-feeding parasitoids) that not only attack the pest but also attack other natural enemy guild members (intraguild predation), as they can seriously interfere with suppression (see Polis and Holt, 1992; Rosenheim *et al*., 1995; Murdoch *et al*., 1998; Rosenheim, 2001), also subsection 7.3.10). Snyder and Ives (2001), in a series of manipulation experiments involving an indigenous parasitoid-generalist predator-aphid system, showed how the generalist predator, which acted primarily as an intraguild predator, can disrupt 'natural' control. In a similar study (Snyder and Ives, 2003) the same researchers showed that the impact of a specialist and several generalists was additive rather than disruptive, although a simulation model fitted to their data suggested that longer-term experiments would have revealed non-additive effects. Even so, except in cases where predators strongly attacked aphid mummies, combined control by both types of natural enemy was predicted to be more effective than with either acting separately. Population cage experiments by Hunter *et al*. (2002), involving an autoparasitoid attacking a whitefly host and also a heterospecific parasitoid and conspecifics, showed that there was no disruption of biological control. Finke and Denno (2002) showed how the structural characteristics of the herbivore's habitat can mediate the effects, upon planthoppers, of intraguild predation by wolf spiders upon mirid bugs. In contrast to structurally simple laboratory 'habitats', more complex habitats increased the combined effectiveness of the predators in suppressing planthopper populations. This effect was attributed to the existence of a refuge for mirids within structurally more complex vegetation (thatch-rich as opposed to thatch-free): it was found that in complex salt-marsh habitats the predatory mirids were relatively more abundant than in simple ones. Finke and Denno's findings suggest both that in classical biological control the dynamic significance of intraguild predation will vary according to the type of agroecosystem involved and/or the type of habitat management practised, and that habitat effects can be tested for through simple experiments.

High Degree of Climatic Adaptation

The optimum range of temperatures or humidities for development, reproduction and survival of a candidate biological control agent (subsections 2.5.2, 2.9.2, 2.7.3 and 2.10.2) may be different from that of the pest, and the parasitoid may either fail to become established or prove ineffective owing to the direct or indirect effects of climate in the region of introduction. The conventional wisdom is that a parasitoid species should be selected for which climatic conditions in the region of introduction are optimal (DeBach and Rosen, 1991) (subsection 2.9.3). This view is supported by the database analysis

Table 7.4 Implications of competition theory for classical biological control (adapted from Murdoch *et al*. 1998).

Theory	Outcome	Recommendation for Release(s)
Simple models (exploitation competition)	Best competitor gives most control	All species; best competitor wins
Enemies interfere (interference competition)	Coexistence can lessen suppressive effect	Best agent, not winner
Enemies interfere and are also self-limiting		
*Intra*specific > *inter*specific limitation	Coexistence reduces pest density	Many species
*Intra*specific < *inter*specific limitation	Added species may lessen suppressive effect	Best agent, not best competitor
Stage-structured pest population	Winner may lessen suppressive effect on key pest stage and even lessen the effect on the total host population	Best agent, not best competitor

of Stiling (1993) which showed that the climatological origin of parasitoids has a large influence on establishment rate. However, the climatic adaptation criterion should not be rigidly applied: *Apoanagyrus lopezi*, which successfully controlled cassava mealybug in West Africa, originated from Paraguay, where the climate is very different (Gutierrez *et al*., 1994; Neuenschwander, 2001).

Having determined the thermal requirements of an agent and/or knowing the climatic conditions in areas where it has already successfully invaded, a **climate diagram** can be used to predict where the insect is likely to become established. Samways (1989) describes such an approach for a coccinellid predator of scale insects. However, **climatic matching programmes** such as CLIMEX (Sutherst and Maywald, 1985) offer a more rapid method, not only for predicting the establishment prospects of species with known thermal requirements, but also for identifying sites for exploration (see Worner *et al*., 1989).

Mills (2000) recommends investigating, post-importation, the role of climatic matching as follows: either (a) release fixed numbers of parasitoids from a single climatically characterised founder population along a climatic gradient in the target region, or (b), using either unique genetic markers (subsection 3.2.2) or morphometric markers (Phillips and Baird, 1996) for different geographic strains of a single parasitoid species, release, in combination, equal numbers of several strains at a series of climatically different locations in the target environment. The latter method can allow the success of local establishment to be related to the degree of climatic match between original and target localities for each strain.

Ease of Handling and Culturing

Greathead (1986) concluded, from an analysis of the BIOCAT database, that the most important factors in choice of natural enemy in classical biological control programmes have, perhaps, been ease of handling and availability of a technique for culturing the insects. The case of biological control of the mango mealybug, *Rastrococcus invadens*, is an illustration of how ease of rearing can influence selection. Two encyrtid parasitoids, *Gyranusoidea tebygi* and *Anagyrus* sp., were being considered for introduction into West Africa. Despite the latter

species being the dominant parasitoid in rearings from field-collected mealybugs in India, the former species was selected as the first candidate for introduction, owing to the ease with which it could be cultured (see Waage and Mills, 1992 for a discussion). A reason given by Waage (1990) for the more extensive use of Ichneumonidae compared with Tachinidae in programmes aimed at controlling exotic Lepidoptera is the greater difficulty encountered in culturing the latter parasitoids (other dipteran parasitoids such as Pipunculidae are notoriously difficult to culture). It is noteworthy that the ranking of culturable agents for introduction usually follows the sequence in which they are established in culture (Waage, 1990).

Practical approaches to rearing and culturing parasitoids and predators are discussed by Waage *et al*. (1985).

Holistic Criteria

Introduction

The holistic approach to the selection of agents considers less the properties of the agent and emphasises instead the interactions between candidate species and between agents and mortalities acting on the pest in its area of introduction. One important consideration in this approach is that the relationships between natural enemies in biological control releases need to be viewed as dynamic, not static. Examples of this approach are:

Collecting Parasitoids From Non-outbreak Areas in the Native Range of the Pest

Selection of agents can begin during the exploration phase of a programme by confining exploration to low density populations of the host. The species composition of parasitoid complexes varies with the density of the host population (subsection 6.3.6). It has been argued that parasitoids collected from host population outbreak areas may not necessarily be those best-suited to maintain the pest at low densities in its exotic range (Pschorn-Walcher, 1977; Fuester *et al*., 1983; Waage, 1990; Waage and Mills, 1992). To increase the likelihood of obtaining the 'better' species, Waage (1990) and Waage and Mills (1992) recommend the use of experimental host cohorts placed out in the field (subsections 6.2.8 and 7.2.9).

Selection of Agents that Follow, Rather than Precede, Major Density-dependent Mortalities in the Pest Life-cycle

Additional density-dependent mortalities acting later in the pest's life cycle can influence the effect of mortality caused by a natural enemy that attacks the pest earlier on (May *et al*., 1981; May and Hassell, 1988). Indeed, if the density-dependence is over-compensating, too high a level of mortality caused by an early-acting parasitoid can lead to an increase in the host population above the parasitoid-free level! A density-dependent mortality acting upon a host – whether due to intraspecific competition (van Hamburg and Hassell, 1984) or the action of natural enemies (Hill, 1988) – can be described by the following model (Hassell, 1975):

$$S = N(1 + aN)^{-b} \qquad (7.41)$$

in which S is the number of survivors, N is the initial prey density, a is a constant broadly indicating the densities at which survival begins to fall rapidly, and b is a constant that governs the strength of the density-dependence ($b = 1$ is perfect compensation, $b < 1$ is under-compensation, and $b > 1$ is over-compensation, subsection 7.3.4). Figure 7.43 shows, for the stem-borer *Chilo partellus* (Lepidoptera), a hypothetical example where S is plotted against N, for three density-dependent functions with different values of b (N in this case refers to larval densities). When $b = 1$ (curve A), the density-dependence tends to compensate for any early-acting (egg) parasitism, as long as the initial larval density is not reduced to lie on the steeply rising part of the curve. When $b < 1$ (curve B), however, there is only partial compensation, and egg parasitism will always reduce the numbers of larvae ultimately surviving. When

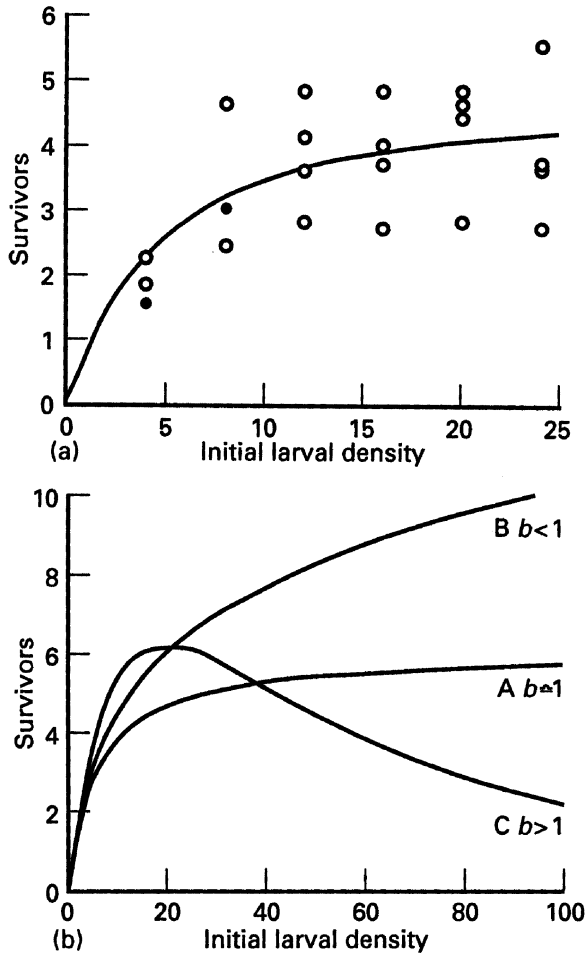

Figure 7.43 (a) The density-dependent relationship between the number of surviving stem-borer, *Chilo partellus* (Lepidoptera: Pyralidae), larvae on plants and the initial densities of first-instar larvae in a glasshouse experiment. The data are described by equation 7.41. Solid circles indicate duplicate points. (b) Three hypothetical examples of the density-dependent relationship in equation 7.41 to show the effect of varying the parameter b which governs the strength of the density-dependence in the host population. Curve A uses the values estimated from the *C. partellus* data in graph (a) ($b = 1.089$). Curve B shows how the number of survivors following egg parasitism continues to increase with initial larval density when $b < 1$. Curve C shows overcompensation when $b > 1$. Source: van Hamburg and Hassell (1984). Reproduced by permission of Blackwell Publishing.

$b > 1$ (curve C) there is overcompensation, and the introduction of egg mortality will lead to *more* larvae eventually surviving unless the initial larval density is reduced to lie on the rising part of the curve.

Van Hamburg and Hassell (1984), in discussing augmentative releases of *Trichogramma* against stem-boring Lepidoptera, concluded that the success of a programme will be largely influenced by the level of egg parasitism, the level of the subsequent larval losses, and the degree to which the latter are density-dependent. Furthermore, these factors will vary between different agricultural systems and different pest species, and should be evaluated for each situation where augmentative releases are being contemplated.

See also Suh *et al*. (2000) on this topic.

Reconstructing Natural Enemy Communities on Exotic Pests/Selection of Agents on the Basis of their Complementary Interactions with Other Agents

With either single or multiple agent introductions the potential exists for interactions between natural enemies, involving the agent(s) and indigenous natural enemy species. Theoreticians have sought to predict the consequences of enemy–enemy interactions both for coexistence and for pest suppression. For parasitoid-parasitoid interactions, it is not possible to provide a general recommendation to biological control practitioners, as theory based on different mechanisms of competition or coexistence generates conflicting advice on releases (Table 7.4) (for a review, see Murdoch *et al*., 1998).

Both local and metapopulation models allow parasitoid coexistence under certain circumstances, while coexistence is common feature of real parasitoid-host systems (Murdoch *et al*., 1998; Mills, 2000). Note, however, that there are several known cases of competitive exclusion/displacement (see Murdoch *et al*., 1998, on parasitoids, Dixon, 2000, on coccinellids, and Schellhorn *et al*., 2002, for a theoretical study of aphid parasitoids).

Zwölfer (1971) noted that within indigenous, i.e. 'natural', parasitoid guilds coexistence is facilitated by species-complementary life-history trade-offs, such as between larval competitive ability and searching efficiency: one species may be a poor larval competitor compared with another, but is superior with respect to host-finding capability. This phenomenon is known as **counterbalanced competition** (Zwölfer, 1971), and has been modelled in the case of biological control introductions (May and Hassell, 1981; Kakehashi *et al*., 1984) (see also Bonsall *et al*., 2002, on the life-history trade-offs that enable coexistence in a 'natural' complex of *Drosophila* parasitoids).

Intuitively, it makes good sense with multiple introductions to attempt to reconstruct, to at least some extent, a pest's natural enemy complex, as its members are most likely to possess complementary traits. Protocols for determining which of a pair of species is the superior larval competitor are discussed in subsection 2.10.2, while the measurement of searching efficiency is discussed in subsection 7.3.7. When investigating interactions between natural enemies, researchers should consider the possibility that parasitoid species may interfere with one another behaviourally, through patch-marking and patch defence (subsection 1.5.3 and section 1.13 respectively).

For a study where reconstruction of a parasitoid complex was contemplated and was approached experimentally, see Patil *et al*. (1994).

It may be the case that a pest, in its exotic range, is already being attacked by one or more indigenous generalist natural enemies. It has been argued that candidate agents for introduction may need to be selected on the basis of their potential for interaction with the generalist (see May and Hassell, 1981, 1988). Specialist egg parasitoids may be easier to establish than larval or pupal ones, particularly if the pest has a relatively low net rate of increase and already suffers significant mortality from generalist natural enemies.

The implications of intraguild predation for natural enemy coexistence and pest suppression have been explored theoretically by Polis *et al*. (1989) and Polis and Holt (1992) (see also 7.3.10). Rosenheim *et al*. (1995) caution that models which assume the two predators to compete for a single prey resource may be inappropriate for most practical situations, given that predators engaging in intraguild predation are likely to be generalists.

We have discussed what theoreticians have to say about multiple agent introductions, but what does the historical record of biological control tell us about such introductions, and what evidence is there that biological control practitioners been listening to theoreticians? Denoth *et al*. (2002) showed, through a BIOCAT database analysis comparing multiple agent with single agent releases, that establishment rate was higher with single agent programmes, but that there was no relationship between the number of agents released and success rate. Denoth *et al*. concluded that negative agent–agent interactions underly these patterns. Myers *et al*. (1989) had shown that in 68% of successful multiple agent programmes a single agent was shown to be responsible for successful control, and Denoth *et al*. concluded from this result that, in a majority of biological control projects, multiple agents are released to increase the likelihood of eventual success (this is termed the **lottery model** in contrast to the **cumulative stress model** [Harris, 1985; Myers, 1985]), rather than to achieve a cumulative control effect. Thus, it appears that ecological theory has not informed introduction strategy.

Selection of Agents Whose Effectiveness is Least Likely to be Compromised by Host Plant Effects

In real parasitoid-host systems the pest's food plant has a potentially important role to play in the enemy-victim interaction (e.g. see Verkerk *et al*., 1998; van Lenteren and van Roermund, 1999). The effect upon host suppression may in some cases be positive (additive, synergistic), but in others it may be antagonistic. An antagonistic effect may vary with the species of candidate natural enemy. For example, behavioural research may show that one candidate agent's searching efficiency is more negatively affected

by crop architecture or foliar pubescence compared with another agent. By the same token, allelochemicals or *Bacillus thuringiensis* (Bt) toxins in the crop may negatively influence larval survival (subsection 2.9.2**)** to a greater degree in one parasitoid species than in another species. There is a strong case for employing multitrophic models in biological control (Gutierrez *et al*., 1990; Mills and Gutierrez, 1999; Mills, 2000), given the potential for significant bottom-up effects (subsection 7.3.10).

7.4.4 NON-TARGET EFFECTS

Introduction

There is increasing concern over the threats posed by biocontrol, especially classical biological control, to natural biodiversity. Introduced biological control agents may either already possess or evolve the ability to attack non-target host or prey species (Secord and Kareiva, 1996; Hawkins and Marino, 1997; Follett *et al*., 2000; which may include not only indigenous herbivores but also indigenous natural enemies. A significant number of biological control introductions have been reported to have adversely affected non-target native species, either directly or indirectly (see reviews by Lynch and Thomas, 2000, and Louda *et al*., 2003). Reductions in non-target abundance that can eventually result in extinctions are a major concern (Simberloff and Stiling, 1996). Consequently, some ecologists feel (in some cases very strongly) that insufficient attention is being paid in biological control programmes to environmental risks posed by introduced agents (e.g. Pimentel *et al*., 1984; Howarth, 1991; Simberloff and Stiling, 1996, Louda *et al*., 1997.

Biological control practitioners retort that: (a) many of the perceived problems with classical biological control derive from early programmes where procedures were less regulated and the risks less appreciated (nowadays, deliberate introductions of vertebrate generalist predators such as toads and mongooses would not be allowed under any circumstances) (Waage, 2001); (b) there is little empirical support for the view that introduced insect parasitoids and predators (as opposed to introduced vertebrates) have seriously affected endemic species (Van Driesche and Bellows, 1996, but see Stiling and Simberloff, 2000) (Lynch *et al*., 2002, note that the quality of evidence on negative impacts is highly variable, ranging from the anecdotal to the relatively quantitative); (c) in Hawaii the negative effects both of accidental introductions of organisms and of habitat loss, have dwarfed those of biological control (e.g. see Follett *et al*., 2000); (d) biological control can be favourable for conservation and is even potentially useful in ecosystem management (e.g. see Samways, 1997; Headrick and Goeden, 2001); (e) the risk of adverse effects arising from biological control (the latter often being last a resort tactic adopted when other control options have failed) has to be weighed against those of doing nothing (see Lynch and Thomas, 2000; Neuenschwander, 2001) – for example, the pest could be a human disease vector or it could be devastating a staple food crop or some other important resource, so a remedy of some sort is considered essential.

In response to the concerns of ecologists and environmentalists, a code of conduct was drawn up by The Food and Agriculture Organization (FAO) (FAO, 1996; see also Schulten, 1997, and Kairo *et al*., 2003) with the aim of minimising risks to non-target organisms. In the code, which considers pre-introduction screening, a key responsibility of the importer prior to importation is stated to be an analysis of the host specificity of the biological control agent and any potential hazards posed to non-target hosts *sensu lato*.

Current Screening Practices

Current selection and screening practices differ significantly between weed control and insect and pathogen control:

1. In weed control programmes, selection and screening is done according to the **centrifugal phylogenetic screening technique (CPST)**. This method is based on the premise that closely related plants species are morphologically and biochemically more similar to one another than unrelated plants. Closely related non-target plant species are

the first to be used in screening, followed by progressively more distantly related species, until the host range of the candidate agent has been circumscribed. The potential host range of the herbivore agent being thus identified, any agent deemed 'risky' can be eliminated from the programme.

2. With insect predators/parasitoids, and with pathogens, the screening is less rigorous (see Thomas and Willis, 1998; Messing, 2001). Also, non-target testing tends not to consider all host/prey species at risk but focuses on those chosen for their conservation (endemics) or other importance (e.g. see Babendreier *et al*., 2003). The CPST is applicable to parasitoids, but must be used with circumspection, given that many parasitoids are host niche-specific as opposed to taxon-specific (Messing, 2001; van Lenteren *et al*., 2003) (as Messing points out, knowledge of behavioural cues in host selection could be useful in predicting which non-target hosts are likely to be vulnerable). While some pre-release investigations (not based on CPST) have accurately predicted post-release host range (e.g. Barratt *et al*., 2002), the possibility remains of **host shifts** (the incorporation of host or prey species into the agent's host/prey range) eventually occurring (see Secord and Kareiva, 1996).

Minimising the Risk

Given the poor level of predictability of classical biological control, there appears to be little hope of accurately predicting effects on *non-target* organisms. What can be done to minimise the risk of non-target effects? The following are measures suggested by biological control workers and ecologists:

1. Undertake better evaluation studies before control (Thomas and Willis, 1998). Some organisms perceived to be pests are found not to be pests, once their effects have been evaluated, so there is no need to control them. In other cases, control may be needed, but this could involve only simple measures such as mechanical control (physically removing pests and disposing of them) or a change in cultural practices.
2. Comply with the FAO code of conduct regarding screening of agents (Thomas and Willis, 1998). There is a need to refine and standardise non-target testing techniques (van Lenteren *et al*., 2003, provide a standard methodology, applied to exotic agents used in inundative releases, but which is largely applicable to agents used in classical biological control). Certainly, biocontrol practitioners need to test a much wider range of potential target organisms than they do at present. With regard to pre-release specificity testing, it is often found that when progressing from simple no-choice tests to more biologically realistic cage and field trials, the relative impact of parasitoids on non-target hosts typically declines, and may even become zero (e.g. see Orr *et al*., 2000). A result of this kind can, however, be highly misleading: even if zero impact is maintained for some time following release, host shifts may eventually occur. Van Lenteren *et al*.'s (2003) standard methodology enables risk assessment rating and therefore ranking of candidate agents.
3. Consider potential non-target effects within an explicit population dynamics context (Holt and Hochberg, 2001; Lynch *et al*., 2002; Louda *et al*., 2003). This has been convincingly demonstrated through modelling by Lynch *et al*. (2002). Their models show that, despite a parasitoid showing low acceptance of a non-target species, it may nevertheless have a large impact on the latter's population abundance. Introductions may cause extinction at the local level, but as Lynch *et al*. argue, while local extinctions may not be significant in themselves, they may translate, via metapopulation dynamics, into broad-scale declines in non-target abundance. The predictions of the model are reasonably approximated with the following formula:

$$N_{min} = K_N \exp(-a_N K_H) \qquad (7.42)$$

where N_{min} is the predicted minimum density

to which the non-target is depressed, and K_H and K_N are the carrying capacities of the target and the non-target hosts respectively, and a_N is the searching efficiency of the parasitoid in relation to the non-target. This formula can be expanded to include various parasitism functions (Lynch *et al*., 2002), for example:

$$N_{min} = K_N f(c_H d K_H) \qquad (7.43)$$

in which f is any function determining the proportion of hosts escaping parasitism in relation to agent density, and $c_H\ d\ K_H$ is a term calculating the peak density of agents in the non-target habitat (c_H being the conversion of parasitised target hosts into the next generation of agents; d being the relative density of agents in non-target habitats compared to target habitats, so portraying the degree of overlap and/or dispersal of the agent between populations).

Note that Lynch *et al*.'s (2002) models focus on transient effects that occur soon after agent introduction, so making short-term pre-introduction laboratory experiments (involving parasitoid/target/non-target) more pertinent than they would otherwise be.

Holt and Hochberg (2001) provide several population dynamics theory-based 'rules of thumb' to be applied when contemplating agent introductions; see their chapter for details.

As has been shown through modelling by Schellhorn *et al*. (2002), indirect non-target effects may depend on agricultural practices.

4. View potential non-target effects within a quantitative food web context, where time and resources permit (Memmott, 2000). Memmott (2000) argues that, by constructing quantitative food webs prior to an introduction and also by manipulating them, important questions can be answered; these include: (a) can introduction lead to extinction of non-target species?; (b) can the introduced biological control agent become a keystone species?; (c) will the introduced agent alter the structure of the natural community? For details, see Memmott (2000).
5. Exercise restraint with regard to multiple agent introductions, given that the risk of non-target impacts will increase with the number of agents used. Denoth *et al*. (2002) advise that: (a) with sequential releases, additional agents should be released only if the first species does not control the pest; and (b) with concurrent releases, the different agents should be released separately in infestations sufficiently isolated from one another to permit monitoring studies to be carried out.
6. Undertake better post-release studies. Non-target effects have, hitherto, rarely been considered as part of the post-release monitoring protocol. The FAO code of conduct recommends such studies. Lynch *et al*. (2002) emphasise the need to have monitoring programmes in place before biological programmes are launched; this is particularly important when dealing with transient population effects (see above).

Lynch and Thomas (2000) also discuss non-target effects in relation to inundative and augmentative releases. Van Lenteren *et al*. (2003) discuss risk assessment in biological control, with particular reference to agents employed in inundative releases.

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