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Modelling the biological control of insect pests: a review of host–parasitoid models

N.J. Mills *, W.M. Getz

Department of Environmental Science, Policy and Management, University of California at Berkeley, 1050 San Pablo Ave, Albany, CA 94706, USA

Abstract

Successful biological control results when an introduced natural enemy, very often a parasitoid, is able to suppress the abundance of an insect pest to a level at which it no longer causes economic damage. We review the host–parasitoid models that have been developed to describe the process of biological control by parasitoids or more specifically, the dynamics of interacting host and parasitoid populations. We trace the origins and basic framework for both discrete time (Nicholson–Bailey) models and continuous time (Lotka–Volterra) models, through the search for stabilizing mechanisms in local populations to the more recent focus on spatial and temporal heterogeneity in the distribution of parasitoid attack. In particular, we review the functional response that underlies all consumer–resource models, partial host refuges generated by spatial heterogeneity or temporal asynchrony of parasitoid attack or by host stage structure, the co-existence of competing parasitoid species, and size-dependent host feeding and sex allocation by parasitoids. The mechanistic explanations for biological control derived from these host–parasitoid models are then compared with the few case studies of successful biological control projects that have received sufficient study. We conclude by questioning, and suggesting improvements for, the basic assumptions of discrete-time and continuous-time models for biological pest control.

Keywords: Functional response; Heterogeneity; Pest management; Population dynamics; Stability

1. Introduction

Classical biological control is the purposeful introduction and establishment of one or more natural enemies from the region of origin of an exotic pest, specifically for the purpose of suppressing the abundance of the pest in a new target region to a level at which it no longer causes economic damage (DeBach, 1964; DeBach and Rosen, 1991). Pests that can be controlled through natural enemy introductions include invertebrates, vertebrates, weeds and plant diseases, but here we confine our attention to insect pests as the most frequently used targets of classical biological control. The organisms that function as natural enemies of insect pests include vertebrate predators (e.g., birds, reptiles, amphibians and fish), invertebrate predators (primarily arthropods), parasitoids (holometabolous insects that live parasitically during their larval stage on a single individual of an arthropod host, causing the death of the host), other macroparasites (primarily nematodes), and microparasites or

* Corresponding author. Fax: (1) (510) 642-0875.

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microbial pathogens (viruses, bacteria, fungi and protozoa). We again restrict our attention to just one group of natural enemies, the parasitoids, which are the most frequently used group of natural enemies in classical biological control and historically have been the focus for models of biological control.

Classical biological control of insect pests, henceforth referred to as biological control, began in 1888 when the now legendary predator, the vedalia beetle (which functions as a parasitoid), was imported from Australia and established in California, where it very rapidly suppressed populations of the cottony cushion scale that had been decimating the developing citrus industry (Caltagirone and Doutt, 1989). This pioneering project was spectacular for several reasons; (a) it reduced populations of the pest to very low densities, where they remain to this day unless disrupted by the intervention of broad-spectrum insecticides, (b) it provided the first experimental evidence that parasitoids can act as keystone species determining the functioning and organization of an agroecosystem, and (c) it generated tremendous interest and activity in promoting the use of parasitoids for the control of invading pests throughout the world.

To date there have been more than 3600 purposeful introductions of parasitoids against more than 500 arthropod pests in almost 200 countries and islands around the world (Greathead and Greathead, 1992). In contrast, very few introductions of microbial pathogens have been attempted, for reasons documented by Maddox et al. (1992). Not all introductions have been as successful as the vedalia example, in fact only 30% of introductions have resulted in establishment of the natural enemy in the target region, and of these, only 36% have resulted in substantial or complete control of the target pest (Greathead and Greathead, 1992). The elusive nature of success in biological control was apparent to even the earliest researchers involved in such programs, and has presented the challenge of developing a scientific framework to explain the reasons for success and failure.

Biological control is essentially a population phenomenon, resulting from the action of a natural enemy population interacting with a host population. The discipline of insect population dynamics grew out of one of the earliest biological control projects, when Howard and Fiske (1911) were faced with the need to explain the rapid expansion of the gypsy moth, an accidentally introduced pest, in the New England states of the U.S. around the turn of the century and to consider the potential for biological control. The spectacular examples of biological control, such as the vedalia story, exhibit two key characteristics: firstly, the *regional density* of the pest is reduced to a very much lower level of abundance following the establishment of the natural enemy and *local densities* of the pest may sometimes be reduced to extinction; and secondly, once reduced, the regional density of the pest population is maintained at low densities unless the interaction is disrupted (Beddington et al., 1978; Murdoch et al., 1985; May and Hassell, 1988; Murdoch, 1990). In this paper we review the use of host–parasitoid models and how they relate to the biological control of insect pests. We first present the general framework upon which these models are based, we then review the origins of population models that seek to explain these characteristics of successful biological control and the subsequent extensions of these earlier models. We examine the importance of key features, such as host refuges, and their influence on population models and host suppression. Then finally, we re-appraise the basic assumptions and directives upon which the models are based, in an attempt to redirect the focus of population modelling to reduce the broadening gap between the theory and practice of biological control.

2. Basic framework of host–parasitoid models

Models of biological control have a long history of theoretical development that have focused on the interaction of a parasitoid and its host. Host–parasitoid models have been favored as they allow several simplifying assumptions that would not be possible for other groups of natural enemies; the specificity of many parasitoids allows the system to be considered closed and allows generation times of host and parasitoid to be considered equivalent, and since host attack is confined to the parasitoid adult female stage only, this allows age structure to be ignored or handled in a more simplified form.

A difference equation framework for a coupled, synchronized host–parasitoid system with discrete generations can be written (after Hassell, 1978; May and Hassell, 1988) as the generalized model:

$$N_{t+1} = d(N_t) N_t f(N_t, P_t) \tag{1a}$$

$$P_{t+1} = cN_t\{1 - f(N_t, P_t)\} \tag{1b}$$

where N_t and N_{t+1} and P_t and P_{t-1} are the host and parasitoid population abundance in generations t and $t+1$ respectively, $d(N_t)$ is the per capita net rate of increase of the host population, $f(N_t, P_t)$ is the proportion of host individuals that escape attack by the parasitoid, and c embodies the numerical response or the average number of parasitoids that emerge per host individual parasitized. P is generally interpreted as the abundance of adult parasitoid females and N as the abundance of host adults and thus c includes the number of parasitoid eggs laid per host, the survival of the parasitoid in the attacked hosts and the sex ratio of the emerging parasitoid adults. This discrete-generation framework characterizes a perfectly synchronized parasitoid interacting with a host that has distinct generations, which is frequent in host–parasitoid systems in temperate regions of the world and even some from more tropical regions when parasitism causes generation cycles within the overlapping generations of some multivoltine hosts (Godfray and Hassell, 1987, 1989; Gordon et al., 1991).

When host generations are overlapping, a differential equation framework is more suitable to represent a coupled host–parasitoid system in continuous time:

$$\begin{aligned} \mathrm{d}N/\mathrm{d}t &= g(N)N - h(N,P)P \\ \mathrm{d}P/\mathrm{d}t &= \gamma h(N,P)P - \delta P \end{aligned} \tag{2}$$

where N and P are the population abundance of host (any stage but in general adults) and parasitoid (generally adult female) respectively, $g(N)$ is the per capita net rate of increase, $h(N,P)$ is the per capita functional response of the parasitoid or the rate of host attack, δ the per capita death rate of the parasitoid population, and γ the conversion efficiency of hosts to parasitoids. The differential model, originally developed to explore vertebrate prey–predator interactions (Lotka, 1925; Volterra, 1926), updates both parasitoid and host populations continuously and so includes within-generation dynamics as well as between-generation dynamics (Lotka, 1923; Murdoch, 1990). This is a critical difference between differential and difference equation models.

It is important to note that, in general, biological control has been modelled as a two-species interaction. In reality, a host–parasitoid interaction never occurs in isolation of a host plant and therefore, a tritrophic model should be used to better represent the tritrophic nature of the biological control of insect pests. We will return to this point in a later section.

3. Functional response

The notion of a functional response is central to modelling any type of consumer resource interaction, including host–parasitoid interactions. This notion has been used in various discrete and continuous time models in different but related ways, which has led to some confusion. It will be helpful for us to clarify the notion of a functional response before reviewing the development of host–parasitoid models.

The functional response of a consumer to a change in the density of a resource is generally understood to be the rate at which an individual consumer extracts resources as a function of resource density. Holling (1959) was the first ecologist to investigate this functional relationship in depth, and did so in the context of shrews and deer mice feeding on sawfly cocoons. It is important to note, however, that Holling only considered the per capita response of isolated individuals to prey density: for the case of several consumers competing for resources, interference may reduce the per capita resource extraction rate in response to changes in consumer density at a given resource density (Getz, 1993). More generally, we would expect the functional response to depend on P as well as on N.

The per capita functional response in the continuous-time framework of Eq. 2 is represented by $h(N,P)$, but in the discrete-time framework of Eq. 1, as we will argue, it is the quantity $h(N_t,P_t)=N_t\{1-f(N_t,P_t)\}/P_t$. These expressions are not directly comparable as $h(N_t,P_t)$ is a per capita attack rate per unit time (or generation) whereas $h(N,P)$ is an instantaneous per capita attack rate. The function $h(N_t,P_t)$ in Eq. 1 is derived by considering the number of encounters (N_e) per host, then calculating the mean encounter rate N_e/N and selecting $f(N,P)$ to be the zero term of a probability distribution representing the frequency of 0, 1, 2,..., attacks on individual hosts (as discussed below, the Poisson and negative binomial distributions are the probability distributions used most often).

Holling (1959) presents three different functional response classes, for which the following are nominal forms: type I (linear then constant), type II (decelerating rise to an upper asymptote), and type III (sigmoidal). Canonical forms for these functional response classes in terms of two arbitrary positive constants u and v and the Poisson distribution for the discrete time functions are:

Type I: (linear in N)

continuous:

$$h(N,P)=uN \quad \text{for } N<v/u$$
$$h(N,P)=v \quad \text{for } N\geq v/u$$

discrete:

$$h(N_t,P_t)=N_t\{1-\exp(-uP_t)\}/P_t \tag{3}$$

(note that the constant portion of $h(N_t,P_t)$ is ignored in discrete-time host–parasitoid models)

Type II: (asymptotic in N)

continuous:

$$h(N,P)=uN/(v+N)$$

discrete:

$$h(N_t,P_t)=N_t\left[1-\exp\{-uP_t/(v+N_t)\}\right]/P_t \quad (v \text{ may be } 0) \tag{4}$$

Type III: (sigmoidal in N)

continuous:

$$h(N,P)=uN^2/(v^2+N^2)$$

discrete:

$$h(N_t,P_t)=N_t\left[1-\exp\left\{-uN_tP_t/\left(v^2+N_t^2\right)\right\}\right]/P_t \tag{5}$$

Other forms of type II and type III functional responses abound in the literature (Royama, 1971; Hassell, 1978; Berryman, 1992).

4. Early developments

Thompson (1939) attributes the earliest developments of a mathematical model of the interaction of parasitoids and their hosts to Bellevoye and Laurent (1897), Marchal (1908) and Fiske (1910). However, the first notable model to address the biological control of a host insect by a parasitoid is due to Thompson (1922, 1924) himself. His involvement in some of the earliest biological control programs using parasitoids led him to develop a simple expression for the impact of a parasitoid on its host:

$$Y=N\{1-\exp(-X/N)\} \tag{6}$$

where Y is the number of hosts parasitized, N the number of individuals in the host population and X the total number of eggs laid by the parasitoid population (i.e., $X = \beta P$, where β is the mean number of eggs laid per parasitoid). This expression is notable for two reasons. Firstly, it introduced, for the first time, the notion of using a probability function for parasitoid attack: given that X/N is the mean number of eggs laid per host, then – under the assumption that eggs are randomly or Poisson distributed among hosts – the quantity $\exp(-X/N)$ is the proportion of hosts that escape attack and $\{1 - \exp(-X/N)\}$ is the proportion of hosts that are attacked one or more times. Secondly, it also introduced the assumption that parasitoid attack rate is constrained by egg limitation and that egg limitation imposes an explicit upper bound on the impact of an individual parasitoid in response to host density, generating a type II functional response (Eq. 4 with $v = 0$).

Thompson (1929) further indicated that the dynamics of a host and parasitoid population could be represented by the difference equations:

$$\begin{aligned} N_{t+1} &= \lambda N_t \exp(-\beta P_t/N_t) \\ P_{t+1} &= \lambda N_t\{1 - \exp(-\beta P_t/N_t)\} \end{aligned} \tag{7}$$

which is equivalent to the model of Eq. 1, with the per capita net rate of increase of the host population $d(N_t) = \lambda$, the escape function $f(N_t, P_t) = \exp(-\beta P_t/N_t)$ and the parameter $c = \lambda$. This model assumes that the generations of parasitoid and host are discrete, that the net rate of increase of the host is constant between generations and independent of host abundance and that a single female parasitoid individual always results from an attacked host individual. Because the model identifies the parameter c in Eq. 1 as λ, the implication is that the host population first experiences net reproduction and then parasitism, with the net rate of increase of the parasitoid population being dependent on the abundance of juveniles λN_t. In effect, the model ignores the issues of host and parasitoid survival rates and parasitoid sex ratio. Thompson (1929) interpreted the model as indicating that host–parasitoid interactions are unstable and incorrectly claimed that the parasitoid population will always eliminate the host population either by steady decline or following an initial increase in the host population. As we have shown elsewhere (Getz and Mills, 1996), however, the model has no non-trivial equilibrium ($N^* > 0$, $P^* > 0$) and, depending on initial population densities, predicts that host and parasitoid populations may both crash to zero or may both grow without bound.

A more familiar and influential model of a discrete-generation host–parasitoid interaction was developed by Nicholson and Bailey (1935):

$$\begin{aligned} N_{t+1} &= \lambda N_t \exp(-aP_t) \\ P_{t+1} &= N_t\{1 - \exp(-aP_t)\} \end{aligned} \tag{8}$$

where, in this case, the escape function $f(N_t, P_t) = \exp(-aP_t)$ with a representing the 'area of discovery' or the proportion of the host environment that can be covered by an individual parasitoid in its lifetime. This model implicitly assumes that parameter c in Eq. 1 is 1 and thus that every attacked host gives rise to a single parasitoid female, a situation that is appropriate only for solitary parasitoids in which the male sex is absent (see Gauld and Bolton (1988) for details of parasitoid biology). The importance of the Nicholson–Bailey model lies in the fact that it has served as the basis for the development of more realistic models of discrete-generation host–parasitoid models. However, it is important to note that, like the Thompson model, the Nicholson–Bailey model distributes parasitoid attacks at random among the host individuals but, in contrast to the Thompson model, the functional response is type I (Eq. 3) with mean attack rate driven purely by parasitoid search. This simple model is also unstable and predicts that both host and parasitoid populations will show divergent oscillations (the parasitoid lagging behind the host by one generation) until the parasitoid population is driven to extinction (Hassell and May, 1973).

The linear functional response of the Nicholson–Bailey model was shown to be an inaccurate representation of parasitoid and predator search by Holling (1959), who introduced the hypothesis that the search for hosts is limited by time. The time taken to 'handle' hosts reduces the time available for search generating a type II

functional response for the proportion of hosts attacked (c.f., Eq. 4), with a maximal mean attack rate set by the ratio of the total lifetime of a parasitoid T to the handling time T_h required for an individual host:

$$\begin{aligned} N_{t+1} &= \lambda N_t \exp\{-a' TP_t/(1 + a' T_h N_t)\} \\ P_{t+1} &= N_t[1 - \exp\{-a' TP_t/(1 + a' T_h N_t)\}] \end{aligned} \tag{9}$$

where a' is the rate of parasitoid search. The addition of handling time as a constraint on parasitoid search adds to the instability of the Nicholson–Bailey model, which increases with the ratio T_h/T (Hassell and May, 1973). Nonetheless, handling time was an important concept in the development of host–parasitoid models as it introduced the notion that parasitoid attack is time limited, in contrast to the earlier suggestion of Thompson (1924) that parasitoid attack is egg limited.

Based upon the early observations of population growth by Verhulst (1838) and Pearl and Reed (1920), both Lotka (1925) and Volterra (1926) independently derived a continuous-time differential model for the interaction of a predator and its prey with potential application to host–parasitoid interactions:

$$\begin{aligned} dN/dt &= rN - aNP \\ dP/dt &= \gamma aNP - \delta Y \end{aligned} \tag{10}$$

where $g(N) = r$ is the per capita net rate of increase of the host population, the functional response $f(N, P) = aN$ (the linear portion of Eq. 3), a is the attack rate (assumed to be dependent on search efficiency), γ is the conversion rate of hosts attacked to female parasitoids, and δ is the per capita parasitoid death rate. The model is a more appropriate description of predation rather than parasitism, as parasitized hosts always remain vulnerable to multiple attacks (superparasitism) unless the parasitoid exhibits perfect discrimination against previously parasitized hosts (Van Alphen and Visser, 1990). Despite this, as we elaborate below, the Lotka–Volterra model (Eq. 10) has been used as the basis of many host–parasitoid models. This model predicts constant rather than divergent (Nicholson–Bailey model) oscillations of parasitoid and host populations, the constancy of the cycles resulting from the continuous nature of the growth of host and parasitoid populations (May, 1973).

These early models of host–parasitoid interactions provided an underlying mathematical framework for further development and, to differing degrees, provided conceptual advances for a theory of biological control. In all cases, however, the dynamics of these host–parasitoid models were unable to generate a stable interaction with a low equilibrium host density: the two features that are generally considered characteristic of successful biological control.

5. The search for stability

The firm belief that the persistence of successful biological control results from the direct stabilizing action of the parasitoid (e.g., Huffaker et al., 1976; Murdoch, 1994) spurred a vigorous search for the biological attributes of parasitoids that could induce stability in host–parasitoid models. In fact, the search for parasitoid-induced stability has dominated the development of a theoretical basis for biological control to the exclusion of any other paradigm, and has been a particularly fertile area of research.

Stability can readily be incorporated into the Nicholson–Bailey model by introducing density-dependent self limitation into either the host or the parasitoid population (after Hassell, 1978):

$$\begin{aligned} N_{t+1} &= \lambda N_t^{(1-m)} \exp(-aP_t) \\ P_{t+1} &= N_t^{(1-m)}\{1 - \exp(-aP_t)\} \end{aligned} \tag{11}$$

$$\begin{aligned} N_{t+1} &= \lambda N_t \exp\left(-aP_t^{(1-m)}\right) \\ P_{t+1} &= N_t\left\{1 - \exp\left(-aP_t^{(1-m)}\right)\right\} \end{aligned} \tag{12}$$

where m is a constant representing the severity of density dependence. Unfortunately, these simple power functions of density dependence have some unrealistic properties, as discussed by Hassell (1978), that make them unsuitable as general models. However, Beddington et al. (1975) provide a more elegant demonstration of the stabilizing influence of density-dependent self limitation on the host population of a Nicholson–Bailey model using the discrete form of the logistic expression (or Ricker equation, Ricker (1954), see Eq. 13 below), and Beddington (1975) provides a similar analysis of the stabilizing effect of density-dependent mutual interference between searching parasitoid adults. Hassell et al. (1983) also show that the addition of a density-dependent parasitoid sex ratio has a stabilizing effect on the Nicholson–Bailey model. Leslie (1958) and Murdoch and Oaten (1975) show similar stabilizing properties of density-dependent self limitation in continuous-time Lotka–Volterra models. In each case, the addition of density dependence to either the host or parasitoid population results in population trajectories that show damped oscillations in host and parasitoid abundance.

Murdoch and Oaten (1975) pointed out that a typical asymptotic type II functional response (c.f., Eq. 4) by a parasitoid induces inverse density-dependent mortality on the host population, but that a sigmoidal type III functional response (c.f., Eq. 5) results in density-dependent host mortality over the range of host densities spanned by the accelerating section of the response curve. In the framework of a Lotka–Volterra model a sigmoidal functional response (c.f., Eq. 5) induces stability (Murdoch and Oaten, 1975), but in the context of a Nicholson–Bailey model it does not (Hassell and Comins, 1978). The difference again lies not with the assumptions of the sigmoidal functional response but with the implicit time delay of the Nicholson–Bailey model in contrast to the continuous dynamics of the Lotka–Volterra model. That there are consistent differences in the properties of Nicholson–Bailey (discrete-time difference equation) models and Lotka–Volterra (continuous-time differential equation) models will be a re-occurring theme throughout this review.

Beddington et al. (1978) reviewed a series of modifications of the Nicholson–Bailey host–parasitoid model in the context of a general theory for biological control. In comparing the realism of the various models, they used the criterion that a model must permit persistence of the two populations and must accurately reflect the degree to which the host population is suppressed by the addition of the parasitoid. The base model included self-limiting host population growth to ensure the persistence of the two populations:

$$\begin{aligned} N_{t+1} &= N_t \exp\{r(1 - N_t/K)\} \exp(-aP_t) \\ P_{t+1} &= N_t\{1 - \exp(-aP_t)\} \end{aligned} \tag{13}$$

where $r = \ln(\lambda)$ (c.f., Eq. 8) is the net rate of increase of the host population and K is the carrying capacity of the environment. The base model was then modified in a number of ways which included alteration of the relative generation times of the parasitoid and host, addition of a type II functional response with parasitoid mutual interference (c.f., Eq. 4), addition of a type III functional response (c.f., Eq. 5), addition of a fixed-number refuge from parasitoid attack, and alteration of the distribution of parasitoid attack from random to aggregated. The degree of host suppression is well represented by the ratio q of the host equilibrium population size in the presence and absence of the parasitoid, which for real field-based examples of successful biological control were estimated to vary from $0.002 < q < 0.03$ (Beddington et al., 1978). Of all the model variations considered, only two could match the degree of host suppression observed in successful biological control and both represent forms of environmental heterogeneity: the occurrence of a very small fixed-number host refuge (less than 1% of K), and the aggregation of parasitoid attack. This result spurred a proliferation of models dealing with the consequences of refuges from parasitoid attack that are the subject of the next section.

6. Spatial heterogeneity and the distribution of parasitoid attack

Environmental heterogeneity or patchiness has the effect of providing the host population with a refuge from parasitoid attack and can occur in many different forms (e.g., Crawley, 1992). The main causes of heterogeneity

are either host plant effects, host effects or parasitoid effects. Host plant-induced refuges include the absence of volatile attractants associated with the host (Weseloh, 1976), the presence of parasitoid deterrents such as leaf surface hairs (Woets and Van Lenteren, 1976), and the presence of physical barriers such as the depth of concealment of the host in host plant tissue (Weis et al., 1985). Host-induced refuges include the production of large egg batches that exceed the ovipositional capacity of a parasitoid (Braune, 1982), the selection of host plant parts that are not searched by the parasitoid (Murdoch et al., 1989), and invulnerable classes of individuals (Bailey et al., 1962; Hassell and Anderson, 1984; Murdoch et al., 1987). Finally, parasitoid-induced refuges include parasitoid switching between hosts (Murdoch and Oaten, 1975), and an aggregated distribution of parasitoid attack (Hassell et al., 1991a).

Refuges can be considered explicitly by allowing either a fixed number or fixed proportion of host individuals to be invulnerable to parasitoid attack. Both types of refuge appear to contribute to the stability of Nicholson–Bailey models (Hassell, 1978), but only a fixed number refuge can stabilize a Lotka–Volterra model (Maynard Smith, 1974; Crawley, 1992). However, the most widely explored form of refuge is that generated by the aggregation of parasitoid attack. Considered initially to result from the tendency of parasitoids to aggregate in patches with higher host densities (Hassell and May, 1973, 1974; Murdoch and Oaten, 1975), aggregation was captured in a simple phenomenological model by May (1978) in which the Nicholson–Bailey model assumption of random attack (zero term of the Poisson distribution) was modified to an assumption of aggregated attack (zero term of the negative binomial distribution):

$$\begin{aligned} N_{t+1} &= \lambda N_t \left(1 + aP_t/k\right)^{-k} \\ P_{t+1} &= N_t \left\{1 - \left(1 + aP_t/k\right)^{-k}\right\} \end{aligned} \tag{14}$$

where k is the exponent of the negative binomial distribution describing the degree of aggregation of parasitoid attack (the Poisson model corresponds to the case $k \to \infty$). For $k < 1$, this model provides stability with the extent of host depression q dependent upon both the attack rate a and the degree of aggregation k. As pointed out by Chesson and Murdoch (1986), however, this model represents a limiting case of an earlier model by Bailey et al. (1962), in which aggregation is assumed to be independent of local host density. Subsequent reviews of parasitism in relation to the variation in host density between patches in the field (e.g., Lessells, 1985; Walde and Murdoch, 1988) clearly showed that patterns of parasitism vary from direct dependence on host density, through independence from host density to inverse dependence on host density. In response to this, alternative Nicholson–Bailey models were developed that explicitly incorporated density-dependent (either direct or inverse) aggregation (DDA) and density-independent aggregation (DIA) of parasitoid attack into a linear (type I, c.f. Eq. 3) functional response (Chesson and Murdoch, 1986; May and Hassell, 1988; Hassell et al., 1991a). These models culminated in the '$CV^2 > 1$' rule (Pacala et al., 1990) which states that a host–parasitoid interaction will be stable whenever the coefficient of variation squared (CV^2) of the density of searching parasitoids in the vicinity of each host exceeds unity, making it clear that both these forms of aggregation have a stabilizing influence. However, the assumptions of an unregulated host population and a linear (type I, c.f. Eq. 3) functional response both affect the generality of these results. Hochberg and Lawton (1990) pointed out that the inclusion of self limitation in the host population can lead to circumstances where less aggregation provides greater stability. Ives (1992b) also noted that relaxation of the assumption of a linear (type I) functional response to include an asymptotic (type II, c.f. Eq. 4) curve affected the stabilizing influence of DDA such that low levels of aggregation are destabilizing, while high levels remain stabilizing. The form of the functional response had no effect on the stabilizing influence of DIA.

Although heterogeneity of parasitoid attack (or the existence of refuges from parasitoid attack) became favored as a mechanistic model to account for the success of biological control and could be used to explain case histories of biological control (e.g., Hassell, 1980), there remained a dilemma, termed the paradox of biological control (Arditi and Berryman, 1991). The paradox is that there is a strong trade-off between stability and the success of a parasitoid in depressing the host equilibrium density in discrete-generation models (Luck,

1990; Murdoch, 1990; Ives, 1992b). This trade-off applies not only to the effects of stabilizing heterogeneity on the Nicholson–Bailey model, but also to other stabilizing effects such as a density-dependent sex ratio. A similar trade-off is also apparent in Lotka–Volterra models that incorporate stabilizing influences such as a type III functional response (c.f., Eq. 5) or an invulnerable age class (Murdoch and Oaten, 1975; Murdoch et al., 1987; Murdoch, 1990).

In a very influential paper, Murdoch and Stewart-Oaten (1989) incorporated aggregation of parasitoid attack into a Lotka–Volterra model, using the general form of Eq. 2:

$$\begin{aligned} dN/dt &= rN - g(N)P \\ dP/dt &= \gamma g(N)P - \delta P \end{aligned} \qquad (15)$$

$$\text{with } g(N) = a/P\{NP + \text{cov}(n,p)\}$$

where N and P are interpreted as the average density of hosts and parasitoids per patch, n and p are the corresponding densities on a randomly chosen patch and $\text{cov}(n,p)$ describes the distribution of parasitoids in relation to the local host density. By assuming that the proportion of parasitoids in a patch increases linearly with the proportion of hosts in the patch, the functional response becomes:

$$g(N) = aN + abV(n)/N \qquad (16)$$

where b represents the degree of aggregation and $V(n)$ is the variance in host patch densities. Taylor (1986) compiled considerable evidence to show that the variance in patch density of an organism is a simple power function of its overall mean density $V(n) = AN^x$, where A and x are positive constants, which on substitution into Eq. 16 gives:

$$g(N) = aN + ab'N^{x-1}, \quad \text{where } b' = bA \qquad (17)$$

Using this model, Murdoch and Stewart-Oaten (1989) found that, in contrast to the Nicholson–Bailey models, DDA results in a reduction of the host equilibrium density but that stability is dependent on the value of the exponent x of the power law function. When $x > 2$ the functional response is sigmoidal (type III, c.f. Eq. 5) and provides stability through density dependence acting on the host population. However, when $x = 2$ the functional response is linear (type I, c.f. Eq. 3) and when $x < 2$ the response is asymptotic (type II, c.f. Eq. 4, the most frequent form for the functional response in real systems), neither of which cause density-dependent host mortality and so do not provide stability. They further found that the inclusion of DIA had no effect on either the host equilibrium or stability.

These contrasting effects of aggregation in Nicholson–Bailey and Lotka–Volterra models of local dynamics result from the fact that redistribution of parasitoid attack in discrete-generation models is limited to just once each generation, whereas in differential equation models parasitoids continually respond to changes in the local patch densities of the host (Murdoch, 1990). This constraint in discrete-generation models provides a link between the spatial density-dependent effect of parasitoid aggregation on the host population and the temporal density-dependent effect of parasitoid aggregation on the per-capita efficiency of the parasitoid population (Murdoch, 1990; Taylor, 1993; Rohani et al., 1994). This linkage gives the impression that stability arises from a spatial effect of parasitism on the host population, whereas it in fact results from a temporal effect of the reduced per-capita efficiency of the parasitoid population such that the greater the reduction in parasitoid efficiency, the greater the stability, and the greater the host equilibrium density. In contrast, for continuous-time models, the within-generation response of parasitoids to local host density breaks the linkage between spatial and temporal density dependence. In these models, the per-capita efficiency of aggregated parasitoids is increased, which leads to a reduction in host equilibrium density and removes the temporal density-dependent effect on the parasitoid population.

The phenomenological model of Murdoch and Stewart-Oaten (1989) has generated some controversy in the

literature (Godfray and Pacala, 1992; Ives, 1992a; Murdoch et al., 1992a) since it is based upon a statistical description of the heterogeneity of parasitoid attack rather than on specific movement rules for the parasitoid. However, the inclusion of parasitoid behavior necessitates a change from a local dynamics model to a metapopulation model that represents an ensemble of patches, and any stochastic variation in either host reproduction or parasitoid attack causes sufficient asynchrony in the dynamics of the separate subpopulations of a metapopulation model to induce stability in the ensemble (Reeve, 1988; Taylor, 1990; Ives, 1992a). The explicit inclusion of parasitoid aggregation between patches in continuous time Lotka–Volterra models very often causes stability (Godfray and Pacala, 1992; Ives, 1992a; Murdoch et al., 1992a) because of this asynchrony. The metapopulation stability demonstrated for a Nicholson–Bailey model of patch dynamics with local dispersal also appears to result from asynchrony. More recently, Rohani et al. (1994) have been able to exclude the problem of asynchrony between subpopulations by combining the use of time-lagged differential equations to model the within-generation dynamics of separate patches together with discrete breaks in the generations to model the redistribution of parasitoids and hosts between generations. Parasitoid attack within a host patch is defined by an instantaneous form of the May (1978) functional response (Eq. 14) and parasitoid density in a patch is defined by a specific rate of dispersal between patches. This model confirms that even moderate levels of parasitoid dispersal between patches within generations removes the temporal stabilizing influence generated by the action of DDA on the per-capita efficiency of the parasitoid population. However, in contrast to the model of Murdoch and Stewart-Oaten (1989), within-generation parasitoid dispersal does not destroy the stabilizing influence of DIA. Whether the simplifying assumption of a linear (type I, c.f. Eq. 3) functional response affects the stabilizing influence of DIA has not been tested.

The role of host refuges in the stabilization of host–parasitoid models and the success of a parasitoid in reducing the equilibrium density of the host population is a complex issue that will undoubtedly continue to be debated in the literature. The frequent conflicts between discrete-generation and continuous-time models indicate the potential confounding effects of the structure of the model and suggest that each of the assumptions need to be addressed carefully before any conclusions can be drawn.

One important assumption that has received very little attention in this context is the assumption that parasitoids are search limited over the full range of host densities. We have questioned this assumption (Getz and Mills, 1996), arguing that pro-ovigenic parasitoids (which emerge with a full complement of mature eggs) have an absolute limitation on the number of hosts that they can attack, and that even synovigenic parasitoids (with continuous production of eggs through their adult life) are likely to experience daily egg limitation. Parasitoid attack is inevitably limited by searching efficiency at low host densities but at higher host densities, the condition that prevails initially in biological control programs, it seems more reasonable to assume that parasitoids are egg limited. To address this question we used a discrete-generation model with a (type II) functional response that combines both search limitation (c.f., Eq. 8) and egg limitation (c.f., Eq. 7), and a generalized distribution of parasitoid attack:

$$N_{t+1} = \lambda N_t f(\epsilon)$$
$$P_{t+1} = cN_t\{1 - f(\epsilon)\}$$

where the mean encounter rate

$$\epsilon = a\beta P_t/(\beta + aN_t) \tag{18}$$

a is the search efficiency of the parasitoid, β is the per capita fecundity of the parasitoid, c is the mean fraction of females emerging per parasitized host and $f(\cdot)$ is the proportion of hosts that escape parasitism each generation. Note that the encounter rate function (Eq. 18) reduces to the pure search limited case $\epsilon = aP_t$ as $\beta \to \infty$ and to the pure egg-limited case $\epsilon = \beta P_t/N_t$ as $a \to \infty$. For the case of the negative binomial form of the escape function $f(\epsilon) = (1 + \epsilon/k)^{-k}$, stability arises only if the distribution of parasitoid attack is sufficiently

heterogeneous ($0 \leq k < 1$) and the maximum per capita growth rate of the parasitoid population (βc) is sufficiently greater than the per capita growth rate of the host population (λ). In addition, stability can only be maintained if the per capita host growth rate declines ($\lambda \rightarrow 1$) as the degree of heterogeneity of parasitoid attack increases ($k \rightarrow 0$). Interestingly, the model indicates that the more efficient a parasitoid, the more it is egg limited at moderate to low host densities and the more suppressed the host equilibrium density. This analysis confirms that heterogeneity is not always sufficient for stability in discrete-time host–parasitoid models and that a low stable host equilibrium density can be achieved through biological control.

Arditi and Berryman (1991) also point out that the paradox of biological control can readily be resolved in Lotka–Volterra models by adoption of a ratio-dependent functional response $h(N/P)$ which permits stability at a low host equilibrium density. Although a ratio-dependent form of the functional response is not generally accepted (see Abrams, 1994; Murdoch, 1994) it has some support from an analysis of data on parasitism in the blackberry leafhopper (Pitcairn et al., 1990), and seems worthy of further investigation in the context of the role of host refuges in biological control.

7. Temporal heterogeneity, host age distribution and synchronization

The Nicholson–Bailey and Lotka–Volterra models discussed so far have glossed over the host stage attacked, although it is perhaps generally assumed that the host stage modelled is the stage susceptible to parasitism. Beddington (1974) and May et al. (1981) have addressed the influence of the relative timing of population processes on the stability of the Nicholson–Bailey model. The basic framework of Eq. 1 implies that both parasitism and density dependence act on the initial host population density. If parasitism occurs before density dependence then the latter should act only on the survivors from parasitism and hence Eq. 1a must be modified to:

$$N_{t+1} = g\{N_t, f(N_t, P_t)\} N_t f(N_t, P_t) \tag{19}$$

Alternatively, to reverse the typical sequence of parasitism before density dependence to density dependence before parasitism Eq. 1b must be modified to:

$$P_{t+1} = cg(N_t) N_t \{1 - f(N_t, P_t)\} \tag{20}$$

The widely adopted formulation for a discrete time model (Eqs. 1a and 1b) is therefore a less realistic and general representation of host–parasitoid systems than the modifications of Eq. 19 and Eq. 20. Using the logistic expression (Eq. 13) for $g(N_t)$ and the negative binomial expression (Eq. 14) for the escape function $f(N_t, P_t)$, May et al. (1981) showed that there are only limited differences in the stability properties of models (Eq. 1a and Eq. 1b) and (Eq. 19 and Eq. 1b). However, for the model in which density dependence acts on the survivors from parasitism (Eq. 1a and Eq. 20) the stable equilibrium density of the susceptible host stage may increase above the carrying capacity K set in the absence of parasitism. This has obvious consequences for biological control as the impact of parasitoid species acting early in the life cycle of their hosts, such as egg parasitoids, could theoretically, at least, cause an increase in the mean density of a host, such as a stem-borer, that is heavily affected by density dependent mortality during the larval stage (e.g., Van Hamburg and Hassell, 1984).

More explicit age structure was added to the Nicholson–Bailey model by Wang and Gutierrez (1980). These authors investigated the stability properties of a model with two age classes, immatures and adults, restricting adult parasitoids to attack immature hosts, but allowing the two processes, parasitism and aging to occur in either sequence. When survival from parasitism precedes aging, the model has limited stability (although stability can occur, unlike the corresponding model with no age structure), but when aging occurs before parasitism a host refuge from parasitism is created and the model shows a broader range of stability.

A similar refuge effect was documented by Murdoch et al. (1987) using a delay-differential Lotka–Volterra model, based upon the stage-structure model of Gurney et al. (1983), to examine the effects of age structure:

$$\begin{aligned} \mathrm{d}U(t)/\mathrm{d}t &= E(t) - M_{\mathrm{U}}(t) - aP(t)U(t) - d_{\mathrm{U}}U(t) \\ \mathrm{d}A(t)/\mathrm{d}t &= M_{\mathrm{U}}(t) - d_{\mathrm{A}}A(t) \\ \mathrm{d}J(t)/\mathrm{d}t &= aP(t)U(t) - M_{\mathrm{J}}(t) - d_{\mathrm{J}}J(t) \\ \mathrm{d}P(t)/\mathrm{d}t &= M_{\mathrm{J}}(t) - d_{\mathrm{P}}P(t) \end{aligned} \tag{21}$$

with

$$M_{\mathrm{U}}(t) = E(t - T_1)\exp\left\{-\int_{t-T_1}^{t}\left[aP(x) + d_{\mathrm{U}}\right]\mathrm{d}x\right\}$$

and

$$M_{\mathrm{J}}(t) = aP(t - T_2)U(t - T_2)\exp(-d_{\mathrm{J}}T_2)$$

where $U(t)$, $A(t)$, $J(t)$ and $P(t)$ are the densities at time t and d_{U}, d_{A}, d_{J} and d_{P} are density-independent per-capita death rates at time t of unparasitized immature hosts, adult hosts, juvenile parasitoids and adult parasitoids respectively. $E(t)$ is the density of host eggs produced per day at time t by $A(t)$ adults, $M_{\mathrm{U}}(t)$ and $M_{\mathrm{J}}(t)$ are the density of immature host and juvenile parasitoids maturing to their respective adult stages per day at time t, T_1 and T_2 are constant durations of the immature stage of the host and juvenile stage of the parasitoid respectively, and a is the attack rate of the linear functional response. The linear (type I, cf. Eq. 3) functional response of the parasitoid determines the density of hosts parasitized per day at time t. The model indicates that an invulnerable adult stage of the host is stabilizing over a much broader set of conditions than is an invulnerable juvenile stage, an encouraging result as most hosts tend to be attacked during the juvenile stages (e.g., Clausen, 1962; Gauld and Bolton, 1988). The stability of the model is dependent upon the duration of the invulnerable host adult stage (mean longevity $T_{\mathrm{A}} = 1/d_{\mathrm{A}}$) relative to that of the immature parasitoid stage, but again there is a trade-off with host equilibrium density which increases with T_{A}.

In contrast to the extensive research on the influence of spatial heterogeneity in host–parasitoid models, there has been far less interest in the effects of temporal heterogeneity generated by asynchrony in the phenologies of the susceptible stage of the host and the foraging period of the adult parasitoid. Griffiths (1969) was the first to make a quantitative study of the effects of a temporal refuge or asynchrony in a host–parasitoid model and Münster-Swendsen and Nachman (1978) provided the first direct evidence that temporal asynchrony alone is sufficient to stabilize a host–parasitoid interaction. More recently, Godfray et al. (1994) have examined the effects of a temporal host refuge using an age-structured time-delayed Lotka–Volterra model very similar to those described above. As in the spatial aggregation model of Rohani et al. (1994), this model has a discrete generation component as well as a continuous-time within-generation component. However, in this case the functional response of the parasitoid (that is represented in the within-generation Lotka–Volterra component of the model) combines a type II response to host density with an instantaneous version of May's (Eq. 14) density-independent parasitoid aggregation:

$$h\left[N(t), P(t)\right] = k\ln\left\{1 + aP(t)/k\left[1 + aT_{\mathrm{h}}N(t)\right]\right\} \tag{22}$$

such that k is an inverse measure of the extent to which parasitoid efficiency is dependent on parasitoid density. It is worth noting here that Griffiths (1969) has earlier used a negative binomial expression for the functional response of his discrete-time model of parasitoid asynchrony. In the absence of asynchrony the Godfray et al. (1994) model is stable for sufficient levels of parasitoid density dependence ($k < 1/W$, where W is the duration of the susceptible host stage). However, the degree of density dependence needed to stabilize the model can be reduced to zero depending on the extent to which parasitoid adult emergence is delayed. Only a relatively small window of stability exists in the total absence of direct parasitoid density dependence, but the effect of

asynchrony is apparent over a broader range of delayed parasitoid emergence suggesting that asynchrony in causing a temporal refuge from parasitism may have an important influence on the persistence of host–parasitoid interactions.

8. Parasitoid competition

Intraspecific competition between parasitoids can occur at the adult stage or the larval stage with different consequences for the host–parasitoid interaction. For example, competition between searching parasitoids may cause a direct density-dependent reduction in the parasitoid attack rate function (Hassell and Varley, 1969; Beddington, 1975; Godfray et al., 1994; Eq. 12 and Eq. 22). Alternatively, competition between adult parasitoids may affect the sex ratio (Hassell et al., 1983; Comins and Wellings, 1985) or competition between parasitoid larvae may affect the per capita parasitoid survival within the host (Taylor, 1988), both of which cause direct density dependence in the mean parasitoid reproduction per host (parameter c of Eq. 1). In both cases, intraspecific competition requires the addition of density-dependent self limitation to the parasitoid model and has the result that the interaction is stabilized, at least at moderate levels of density dependence, with the consequence that the host equilibrium density is raised (but see Taylor, 1988).

On the other hand, by biasing its sex ratio towards female progeny, a parasitoid population will invariably increase its competitiveness. As a consequence, a female-biased population may ultimately exclude its competitors or, in extreme cases, exclude its competitors and destroy the host population along with itself (Kaitala and Getz, 1992).

One of the most important questions in biological control has been whether multiple parasitoid species provide better control of the host than a single parasitoid species. In general, both the Nicholson–Bailey and Lotka–Volterra models permit the persistence of only the single most efficient of two parasitoid species (i.e., the parasitoid species with the greatest attack rate a). The addition of parasitoid interference into the Nicholson–Bailey model by Hassell and Varley (1969), (Eq. 12) provided the first instance in which a second parasitoid species could be added to a host–parasitoid model to allow both parasitoid species to persist. A similar result was obtained using a model that included the stabilizing influence of both direct density dependence on the host population (Eq. 13) and temporal density dependence, through a negative binomial distribution of parasitoid attack (Eq. 14), on the parasitoid populations (May and Hassell, 1981):

$$\begin{aligned} N_{t+1} &= N_t \exp\{r(1 - N_t/K)\}(1 + a_P P_t/k)^{-k}(1 + a_Q Q_t/k')^{-k'} \\ P_{t+1} &= N_t\{1 - (1 + a_P P_t/k)^{-k}\} \\ Q_{t+1} &= N_t(1 + a_P P_t/k)^{-k}\{1 - (1 + a_Q Q_t/k')^{-k'}\} \end{aligned} \tag{23}$$

This model includes the simplifying assumption that parasitoid P acts independently of parasitoid Q (i.e., parasitoid P is either the superior larval competitor or parasitoid Q attacks only the hosts that survive parasitism from P) and that the distribution of parasitoid attacks are independent, which implies that the two parasitoids have non-overlapping niches. In general, the model indicates that a second parasitoid can successfully invade and persist in a system when both parasitoids limit their own abundance more than that of the competitor (Briggs, 1993). The successful persistence of a second parasitoid with a greater attack rate generally reduces the host equilibrium density below that achieved by the first parasitoid alone, although the degree of host suppression may not be as great as could be achieved by the second parasitoid acting alone (May and Hassell, 1981, 1988). A very similar model (Nicholson–Bailey with aggregated parasitoid attack) has been extended by Hogarth and Diamond (1984) to include the effects of a variable rather than a fixed outcome of competition with essentially similar results.

However, Kakehashi et al. (1984) pointed out that the model of May and Hassell (1981) allows the two parasitoids to have independent distributions of parasitoid attack within the host population (an assumption also

used by Hogarth and Diamond, 1984), and argue that it may be more realistic to assume that both parasitoids will share the same distribution of attack (i.e., identical niches) in responding to the same set of cues used for host location. This alters the equation for the second parasitoid to give:

$$Q_{t+1} = N_t\left\{(1 + a_P P_t/k)^{-k} - (1 + a_P P_t/k + a_Q Q_t/k)^{-k}\right\} \tag{24}$$

which has little affect on the stability properties of the Nicholson–Bailey model, but does affect the host equilibrium density and the question of multiple introductions of parasitoids. For the situation where both parasitoids have identical niches, the single parasitoid with the greatest attack rate always maximizes the reduction in host equilibrium density. Using a simplified Nicholson–Bailey model with a linear functional response, Kakehashi et al. (1984) were able to introduce a variable level of niche overlap between parasitoids, indicating that single introductions are always the best strategy in biological control unless niches are completely segregated, or when parasitoid Q attacks only a fraction of hosts that have been attacked by parasitoid P and the attack rates of the parasitoids are very different.

Building upon the stage-structured Lotka–Volterra model framework of Murdoch et al. (1987, 1996, Eq. 21), Briggs (1993) and Briggs et al. (1993) consider the interaction of two parasitoids, one of which attacks the host egg stage (P) and the other the host larval stage (Q), in a host population with three age classes:

$$\begin{aligned}
\mathrm{d}E(t)/\mathrm{d}t &= rA(t) - M_E(t) - a_P P(t) E(t) - d_E E(t)\\
\mathrm{d}L(t)/\mathrm{d}t &= M_E(t) - M_L(t) - a_Q Q(t) L(t) - d_L L(t)\\
\mathrm{d}A(t)/\mathrm{d}t &= M_L(t) - d_A A(t)\\
\mathrm{d}P(t)/\mathrm{d}t &= a_P P(t - T_{JP}) E(t - T_{JP}) \exp(-d_{JP} T_{JP}) - d_P P(t)\\
\mathrm{d}Q(t)/\mathrm{d}t &= a_Q Q(t - T_{JQ}) L(t - T_{JQ}) \exp(-d_{JQ} T_{JQ}) - d_Q Q(t)
\end{aligned} \tag{25}$$

with

$$M_E(t) = rA(t - T_E)\exp\left\{-\int_{t-T_E}^{t}\left[a_P P(x) + d_E\right]\mathrm{d}x\right\}$$

$$M_L(t) = M_E(t - T_L)\exp\left\{-\int_{t-T_L}^{t}\left[a_Q Q(x) + d_L\right]\mathrm{d}x\right\}$$

where $E(t)$, $L(t)$ and $A(t)$ are egg, larval and adult host densities, d_i are the density independent death rates of the host stages and r is the per capita birth rate of the host adults. $P(t)$ and $Q(t)$ are the densities of the two adult parasitoids, a_P and a_Q are their respective attack rates, T_{JP} and T_{JQ} are the durations of the juvenile stages in days, and d_{JP} and d_{JQ} the death rates of the juvenile parasitoids. $M_E(t)$ and $M_L(t)$ are maturation functions for the host egg and larval stages with T_E and T_L the durations of the egg and larval stage. The model includes no explicit density dependence acting on the host or either of the parasitoid populations, and none results from the linear parasitoid functional responses.

Briggs (1993) initially retained the assumption of a constant stage duration in this general stage-structured model, to explore the effect of larval competition (termed intrinsic competition by Zwölfer (1971)). By making the assumption that one parasitoid is always intrinsically superior to the other in larval competition, then co-existence is possible only if the later-attacking parasitoid Q is intrinsically superior and shows at least some discrimination against the attack of hosts previously parasitized by parasitoid P. The set of conditions for co-existence is limited and the stability determined by the relative duration of the invulnerable adult stage (as in the single host–parasitoid model of Murdoch et al. (1987, 1992b, discussed above). More generally, the parasitoid that is intrinsically superior is able to exclude its competitor, unless the ratio of relative attack rates (extrinsic competition between adult parasitoids) greatly favors the intrinsically inferior parasitoid. However, in contrast to the earlier non-stage-structured models of May and Hassell (1981) and Kakehashi et al. (1984), that were based solely on extrinsic competition between parasitoids, the single parasitoid species that persists in the

interaction (as a result of both intrinsic and extrinsic competition) does not always provide the best reduction in host density. If the goal is to reduce the abundance of a particular host stage, it is best to use a single intrinsically-superior parasitoid that attacks that particular stage (the result can be reversed if the parasitoid is intrinsically inferior), but the equilibrium density of adult hosts is not always reduced to the greatest extent by the winning parasitoid. In addition, when parasitoids do co-exist in the system, the host density at all stages tends to be intermediate between those levels set by each parasitoid acting alone.

Briggs et al. (1993) later relaxed the assumption of a constant stage duration, to explore the influence of variation in immature stage durations in a model in which parasitoid P is assumed to be intrinsically superior. With a constant stage duration, there is no parasitoid co-existence, as shown by Briggs (1993), but if individuals mature out of a stage at a constant rate (i.e., an exponential distribution of stage durations) the set of conditions under which parasitoid P excludes parasitoid Q is reduced to provide a relatively broad region of parameter space that permits co-existence. In contrast to the constant stage duration model, this variable stage duration model also suggests that under conditions that support the persistence of only a single parasitoid, the winning parasitoid always provides the best reduction in the density of adult hosts (as in non-stage-structured models), and similarly when both parasitoids persist the resultant host equilibrium is also equivalent to the best reduction achievable by either of the parasitoids acting alone. The choice of a constant rate of maturation to represent the variability in immature stage durations is somewhat unrealistic, since it allows some individuals to mature almost immediately from the immature stage. The addition of a minimal stage duration, from which individuals then mature at a constant rate, improves the realism of stage maturation, but drives the resultant behavior of the model towards that of the constant stage duration model. It reduces the parameter space that permits co-existence and reverts to the winning parasitoid not necessarily providing the best reduction in adult host abundance and co-existence providing intermediate host equilibrium densities, unless the mean duration of the variable section of the stage duration greatly exceeds the minimum stage duration.

Building upon an earlier model framework (Hassell et al., 1991b; Comins et al., 1992) in which local dispersal between patches in a two-dimensional grid provided metapopulation persistence of a host–parasitoid model with locally unstable Nicholson–Bailey dynamics, Hassell et al. (1994) demonstrate that these same conditions promote the co-existence of two parasitoids on a single host. If the dispersal rates of the parasitoids are similar only a narrow window of stability exists, but the stable co-existence of the parasitoids occurs over a greater range of conditions if the parasitoids differ markedly in dispersal rate. The less dispersive parasitoid appears to occupy refuges within the grid and to persist at much lower densities than the more dispersive parasitoid. The effects on equilibrium densities of the host metapopulation were not explored.

9. Size-dependent host feeding and sex allocation

Predation of hosts by adult female parasitoids, or host feeding, is frequent among hymenopteran parasitoids (also recorded in the Tachinidae, Nettles, 1987), particularly among synovigenic species, and typically results in the death of the host (Jervis and Kidd, 1986). In general, adult parasitoids feed on the same host population that they use for oviposition, but it is common to find that host feeding is confined to the smaller host individuals that are unacceptable for oviposition (Walde et al., 1989; Kidd and Jervis, 1991a). Similarly, there is mounting evidence that hymenopteran parasitoids that can attack hosts of different size, frequently place male eggs in smaller hosts and female eggs into larger hosts (King, 1987). These host size-dependent features of host–parasitoid interactions have only recently been addressed by models.

The first model to incorporate host feeding as well as parasitism (Yamamura and Yano, 1988) was a non-stage-structured Lotka–Volterra model in which a constant proportion of the attacks by the parasitoid were assumed to result in host feeding and the parasitoid death rate was set to be a complex function of feeding that we will not elaborate here. At intermediate host feeding to oviposition ratios, host feeding was found to stabilize the host–parasitoid model, but it is not clear what feature of the model caused the stabilizing action. Kidd and

Jervis (1989) considered a stabilized Nicholson–Bailey model (with no age structure) in which an empirical relationship between the realized lifetime fecundity of a parasitoid and the number of hosts attacked (Jervis and Kidd, 1986) is replaced by a simplified phenomenological function $K(N_t)$:

$$\begin{aligned} N_{t+1} &= \lambda N_t \exp(-aP_t) \\ P_{t+1} &= KN_t\{1 - \exp(-aP_t)\} \end{aligned} \tag{26}$$

where

$$\lambda = (R - mN_t)$$

and

$$K = 1 - \{1/[\log(N_t) + 1]\}$$

R being the maximum host population growth rate and m a constant representing the degree of host density dependence. Since the destructive host feeding in this model is independent of host and parasitoid density, the choice between host feeding or oviposition has no stabilizing influence on a host population with a density-independent growth rate and it serves to raise the host equilibrium density of the host stabilized model. In a more detailed simulation model that incorporates host and parasitoid age structure, egg resorption and egg limitation, which result from inadequate host feeding, had an important destabilizing effect on the model (Kidd and Jervis, 1989). When stage discrimination (host feeding on younger hosts but oviposition on older hosts) by a parasitoid with an unlimited egg supply was added to the model, a limited degree of stability (dependent on the relative parasitoid generation time) occurred in a continuous generation version of the model but none was apparent in a discrete generation version (Kidd and Jervis, 1991b).

The conflicting evidence that host feeding may either be stabilizing (Yamamura and Yano, 1988) or have no effect on the stability (Kidd and Jervis, 1989, 1991b) of host–parasitoid models with no age structure has been addressed more recently by Briggs et al. (1995). These authors used an analytical Lotka–Volterra framework in which the parasitoid population was structured by egg load (the number of mature eggs stored by a synovigenic female parasitoid at a particular point in time). In this model host feeding had no effect on stability whether the decision to feed on or parasitize a host was a function of parasitoid egg-load or not. A stabilizing effect of host feeding was achieved by introducing the assumption that the mortality rate of parasitoid females is a decreasing function of their egg load, but the further addition of a drain on egg load (egg resorption) to support maintenance resulted in a destabilizing influence.

Murdoch et al. (1992b) have also extended their stage-structured Lotka–Volterra model (Murdoch et al., 1987) to address stage discrimination for both host feeding and sex allocation, such that young immature hosts may either be host-fed or receive a male egg (i.e. young hosts are killed but do not contribute to parasitoid recruitment) and old immatures receive only a female egg. The model has a linear functional response and it is further assumed that host feeding has no effect on parasitoid attack rate or longevity. One clear feature of the model is that the host equilibrium density increases with the relative attack rate of young immatures, the susceptible host stage that fails to contribute to parasitoid recruitment. There is also an inverse relationship between adult parasitoid and old immature host densities, since more adult parasitoids result in the death of more younger immature hosts leaving less old immature hosts for female parasitoid development. This effect, termed 'pseudo-density-dependence', causes delayed density-dependence on the parasitoid population which permits stability under a limited set of conditions (based on the duration of the immature host stage and the development time of the parasitoid) but the typical Lotka–Volterra limit cycles are more prevalent.

10. Case studies in biological control

The models that we have discussed above have been developed as conceptual tools to advance our understanding of host–parasitoid systems in general, the parameters that are most influential in these systems

and the minimal parameter set that is necessary to capture the dynamics of the system. However, it is also important to consider how frequently and how successfully these models have been applied to real case studies in biological control.

The first attempt to use models in the context of real case studies in biological control concerned the winter moth, a defoliator of hardwood trees in eastern Canada (Embree, 1971; Varley et al., 1973). When this program took place in the 1950s the host–parasitoid model in general use was the basic Nicholson–Bailey model (Eq. (8)). This model predicted cyclical outbreaks of the winter moth following the establishment of parasitoids from its native Europe (Varley and Gradwell, 1968), but once implemented, the program was very successful in reducing this pest to low levels of abundance (Embree, 1971). Subsequently, in a retrospective analysis of the program, Hassell (1980), following the prevailing view that parasitoid aggregation was the most likely mechanism for successful biological control (Beddington et al., 1978), used the Nicholson–Bailey model for aggregated parasitoid attack (Eq. 14) to explain the stable reduction of winter moth abundance. More recently, however, Roland (1988, 1994) has re-analysed the program in greater detail to show that although parasitism significantly reduced the abundance of surviving pupae during the population decline of the winter moth, density-dependent predation of unparasitized pupae in the soil, by generalist predators, was able to maintain winter moth at low levels of abundance. Thus in the case of the winter moth, it seems that predation rather than parasitism stabilizes populations under biological control.

As more than 40% of all natural enemy introductions have been targeted against homopteran hosts and the best examples of spectacular control have resulted from this host taxon (Greathead and Greathead, 1992; Mills, 1994), it is not surprising that at least some of these programs have been addressed by host–parasitoid models. The more recent applications of host–parasitoid modelling to biological control programs are based on continuous-time Lotka–Volterra models, either in the form of delay-differential stage-structured models (Godfray and Waage, 1991; Murdoch, 1994; Murdoch et al., 1996), or in the form of simple simulation models with modular age-structured sub-models for each trophic level in the system (Gutierrez, 1992; Mills and Gutierrez, 1996). In general, the programs have been analysed retrospectively; the cassava mealybug in Africa (Gutierrez et al., 1993) and the California red scale in California (Murdoch, 1994; Murdoch et al., 1996). However, Waage (1990) introduced the idea of prospective modelling to examine the dynamic consequences of different combinations of parasitoid species, from the parasitoid assemblage that exists in the region of origin of a pest, in order to select the most effective parasitoid species for introduction in a biological control program. Prospective modelling has subsequently been applied to the mango mealybug in west Africa (Godfray and Waage, 1991) and the silverleaf whitefly in California (Mills and Gutierrez, 1996).

These applications of host–parasitoid models to case histories and prospective projects in biological control have revealed several important considerations. Firstly, in both the cassava mealybug (Hammond et al., 1991) and the California red scale (Reeve and Murdoch, 1985) systems there is very little evidence of an aggregation of parasitoid attack. It is also apparent from these case studies that some form of age structure is essential in host–parasitoid models to be able to correctly interpret the dynamic consequences to the host population of competing parasitoid species that attack different stages of the host life cycle (Godfray and Waage, 1991; Gutierrez et al., 1993; Mills and Gutierrez, 1996; Murdoch et al., 1996). The role of a refuge from parasitism, either as physical refuges (Murdoch et al., 1989) or as host quality (i.e., size) effects (Gutierrez et al., 1993; Murdoch, 1994), appears to have a strong influence on the local dynamics of the host populations in these case studies. Lack of synchronization, or temporal refuges, were not apparent in these systems but may nonetheless be another important refuge factor in real examples of biological control, particularly those in which there are discrete host generations. Despite the fact that host feeding is common to the parasitoids of the Homoptera, this aspect of the interaction has either been ignored (Godfray and Waage, 1991; Murdoch, 1994; Murdoch et al., 1996) or considered of little consequence to the overall dynamics of the interaction (Gutierrez et al., 1993; Mills and Gutierrez, 1996) in these case studies. Finally, host plant effects are apparent in the California red scale system (Murdoch, 1994) but such 'bottom-up' effects have only been explicitly explored for the cassava mealybug system (Gutierrez et al., 1993, 1994).

11. Conclusions

The pursuit of a theoretical basis for biological control, through the development of mathematical models to enhance our understanding of the mechanisms for successful control of a pest population by an introduced parasitoid, has had a checkered history. Starting from a very simple assumption that parasitoids are egg limited (Thompson, 1924) the majority of subsequent models have all focused, perhaps erroneously, on host limitation. Similarly, the early notion that biological control results when the parasitoid induces density-dependent mortality on the pest population (Smith and van den Bosch, 1967; Huffaker et al., 1976), focused attention on the stabilizing action of various parameters of the parasitoid host interaction, whereas with the realization that the local dynamics of successful programs may not be stable (Murdoch et al., 1985) the degree to which a parasitoid population can suppress the regional abundance of a pest has become, quite rightly, a more important question in biological control models.

The discrete time formulation of the Nicholson–Bailey model appears to be of declining interest for providing a theoretical framework for biological control, even in situations where the host has discrete generations (e.g., Godfray et al., 1994) due to the overriding importance of stage structure and developmental delays on the attack rate of the parasitoid population. However, in dealing with broader issues of host–parasitoid interactions a Nicholson–Bailey structure may still be valuable (e.g., Hassell et al., 1994) and simpler to analyze than differential equation models. In this context, it seems long overdue to re-appraise the basic structure of the Nicholson–Bailey model (Eq. 1) with its implicit assumption that reproduction, parasitism and self limitation all act upon the same host stage, or at least the same level of host abundance. The Thompson (1929) model (Eq. 7) included parasitism acting on the abundance of the host after reproduction, but in reality the host population will experience reproduction, parasitism and density dependence as a sequence of events acting at the different levels of abundance that occur through a generation. A more general model that accounts for this effect requires the form:

$$\begin{aligned} N_{t+1} &= \lambda N_t g(D_{t+1}) f(S_{t+1}, P_t) \\ P_{t+1} &= cS_{t+1}\{1 - f(S_{t+1}, P_t)\} \end{aligned} \tag{27}$$

where D_{t+1} and S_{t+1} are the densities of the host population at the particular points in time when self-regulation and parasitoid attack are in effect. These densities will themselves be functions of adult host N_t and female parasitoid P_t densities, such that:

$$\begin{aligned} D_{t+1} &= F_D(N_t, P_t) \\ S_{t+1} &= F_S(N_t, P_t) \end{aligned}$$

As before, the parameter λ is the net reproductive rate of the host taking into account the host sex ratio, the mean per capita fecundity of female hosts and all density independent mortality and c reflects the mean number of female parasitoids emerging from a parasitized host (i.e., includes parasitoid sex ratio and host mortality after parasitoid attack but before the parasitoid exits the host). This formulation is a more realistic representation of the sequence of population events that occur both prior to, during, and after the host reaches the stage susceptible to parasitism and thus embodies some basic elements of stage structure without the complexity of using a series of separate host equations for the three distinct stages.

The same concern applies to the continuous time Lotka–Volterra model, unless stage structure is added explicitly (as in the models based upon Gurney et al., 1983). In addition, in contrast to the Nicholson–Bailey models, the instantaneous parameters of Lotka–Volterra models do not explicitly incorporate the effects of superparasitism. These models therefore assume that any host individual can only be parasitized once or that parasitoids show perfect discrimination against previously parasitized hosts. Neither assumption fits the observed behavior of most parasitoids (Godfray, 1994) and the extent to which this feature may influence the predictions of such models, as far as we are aware, has never been explored.

The continued use of a linear (Nicholson–Bailey and Lotka–Volterra) functional response in models of biological control would also seem to be in need of revision. Since all parasitoids must experience egg limitation at higher host to parasitoid ratios, an asymptotic type II response (with its known destabilizing influence, Hassell and May (1973)) must be considered a minimal representation of the functional response, particularly in view of the fact that the shape of the functional response can cause very different predictions from host–parasitoid models (e.g., Ives, 1992b).

It must also be argued that a two-species host–parasitoid relationship never takes place in isolation, as assumed in almost all models. In reality, all arthropod pests must at minimum represent an intermediate trophic level in a tritrophic system in which the variable plant resource may have as great an influence on the pest population as the population of parasitoids (Gutierrez et al., 1994). In addition, it is very seldom that only a single parasitoid species is used in a biological control program, although one parasitoid species is often much more abundant than its competitors, and consequently a multiparasitoid tritrophic model (such as that used by Gutierrez et al., 1993; Mills and Gutierrez, 1996) needs to be more widely employed in the analysis of biological control.

Thus, in conclusion, with greater attention to the basic assumptions of the host–parasitoid models used in biological control, it seems that significant advances can be made in our understanding of the mechanisms that lead to the successful suppression of the abundance of a pest through the introduction of parasitoids. Such models provide an experimental framework to examine the reasons for past biological control successes and failures and to develop more successful programs in the future. The notion of prospective modelling, in contrast to retrospective modelling, is both intuitively appealing and seems likely to offer important contributions to the development and implementation of future programs, although as of yet it has seldom been used. It must be remembered, however, that real biological systems are complex and that the application of simple models to specific pest problems may not generate realistic predictions without consideration of the more detailed biological relationships pertinent to that situation. Nonetheless, there is much to be gained from using models to assess the generalities of biological control programs and to continue to develop a sound theoretical framework that can be used to improve the implementation of parasitoid introductions for particular pest taxa.

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