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Population Dynamics and Biological Control [and Discussion]

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Population dynamics and biological control

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Using simple models for host–parasitoid and host–pathogen interactions, we present a basic framework for examining the outcome of releasing natural enemies against a target pest population in a classical biological control programme. In particular, we examine the conditions for the initial invasion and establishment of a natural enemy species, for the maximum depression of the host population, and for the persistence of the populations in a stable interaction. In these conditions there are close parallels between parasitoids and pathogens.

The practice of augmenting an existing natural enemy population by regular mass releases has been widely practised, especially with parasitoids. The conditions for eradication of the pest are very similar in host–parasitoid and host–pathogen models, namely that releases must be greater than the equilibrium production of natural enemies in the absence of releases. Any additional density dependence acting on the host population after the stage attacked by parasitoids can influence the effectiveness of augmentative releases. This is particularly the case with over-compensating density dependence when additional releases can actually lead to an increase in the host population.

A theoretical basis for biological control cannot be properly developed simply by considering the dynamics of releasing single natural enemy species. Biological control often involves the interplay among different types of natural enemies affecting the same host population. As a step in the direction of producing more complex, multispecies models, we examine the dynamics of three situations: (1) where the host is attacked by two parasitoid species; (2) by a generalist predator and a specialist parasitoid; or (3) by a parasitoid and a pathogen. The dynamics of these three-species systems can be complex, and with properties not easily foreseen from the separate pairwise interactions. These results caution us against formulating biological control strategies purely in terms of two-species systems.

For the main part we examine host–parasitoid interactions with discrete, synchronized generations. These would appear to be less suitable to tropical insects where continuous generations and life cycles of the host and parasitoid of different length are to be expected. We show, however, that cycles (with a period of one host generation) can be obtained from an age-structured simulation model, and that these are promoted by the parasitoids having a life cycle half as long as that of the host. Some implications for biological control are discussed.

Finally, we turn briefly to the dynamics of host–parasitoid and host–pathogen interactions where pesticides are also applied, and we discuss the evolution of pesticide resistance within the context of these models.

Introduction

The broad objective of biological control is to reduce the average abundance of a pest by using one or more populations of natural enemies, and in so doing to reduce the chance of future outbreaks. These are issues that go to the very heart of population dynamics, involving the determinants of population abundance (measured by long-term average levels), the role of

natural enemies in promoting the persistence of their hosts or prey, and the 'invasion' of new, natural, enemy species into established guilds. Biological control should thus be viewed as an application of the principles of population ecology; only in this way can it escape from its current state of *ad hoc* practices based mainly on poorly documented past experiences.

This does not mean, however, that such a basic ecological approach to biological control will lead to confident pronouncements on the outcome of particular programmes. Understanding the dynamics of populations in the field requires lengthy and intensive study, on time scales that are usually incompatible with the urgency imposed by heavy pest-inflicted losses. Predictions in population biology are, moreover, almost invariably probabilistic rather than deterministic. We should, therefore, look to population ecology to provide insights on general strategic problems (such as the relative merits of generalist versus specialist natural enemies, or of single versus multiple introductions), rather than detailed predictions on the use of particular natural enemies.

In this paper we shall consider for the main part the use of parasitoids and predators in biological control, although we shall also in several places draw parallels with the use of pathogens. It is encouraging that several principles apply irrespective of the type of natural enemy. We begin by considering basic components of the interactions between hosts and parasitoids, and between hosts and pathogens. This leads to a simple framework for discussing the dynamical properties of such interactions. We then extend the discussion to situations with more than one natural enemy species, with complex host and parasitoid life cycles, or with natural enemies regularly augmented by further releases or supplemented by the use of insecticides.

Basic frameworks for the dynamics of host–enemy interactions

(*a*) *Hosts and parasitoids*

More than 10% of all metazoan species are insect parasitoids, a term first applied by Reuter (1913) to describe insects that develop as larvae on the tissues of other arthropods, which they ultimately kill. Many previous studies have focused on the importance of particular attributes of parasitoids to biological control (see, for example, Hassell & May 1973; Beddington *et al.* 1978; Waage & Hassell 1982), drawing conclusions from simple models for the interaction of the population of a host and its coupled parasitoid:

$$N_{t+1} = Fg(fN_t)\, Ntf(N_t, P_t), \tag{1a}$$

and

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

Here, N and P are the host and parasitoid populations, respectively, within successive generations, t and $t+1$; $Fg(fN_t)$ is the per capita net rate of increase of the host population (which is density dependent when the function g is less than 1); c is the average number of adult female parasitoids emerging from each parasitized host (c therefore includes the average number of eggs laid per host parasitized, the survival of these progeny, and their sex ratio); and the function f defines the fraction of hosts that are not parasitized. A feature of such interactions with discrete generations is the different dynamics that can occur depending upon the sequence of mortalities and reproduction in the host's life cycle (Wang & Gutierrez 1980; May *et al.* 1981; Hassell & May 1986). Equation (1) is for the case of parasitism acting first, followed by the density dependence defined by g (see May *et al.* (1981) for a discussion of alternatives). In

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effect, therefore, the model represents an age-structured host population with pre- and post-parasitism stages. The implications of this are discussed in a number of the contexts below.

Throughout this paper we shall use a particular form for the function f in equation (1), namely the zero term of a distribution of parasitoid attacks on hosts that is assumed to be negative binomial (May 1978) (see figure 3):

$$f(N_t, P_t) = \left[1 + \left\{\frac{aP_t}{k(1 + aT_h N_t)}\right\}\right]^{-k}. \tag{2}$$

Here, a is the *per capita* searching efficiency of the parasitoids, T_h is their handling time (Holling 1959 *a*, *b*) and k is the parameter from the negative binomial determining the degree of contagion in the distribution of attacks. Contagion increases as $k \to 0$, whereas in the opposite limit, $k \to \infty$, the Poisson distribution is recovered to give the Nicholson–Bailey expression for independently random parasitoid attacks (Nicholson 1933; Nicholson & Bailey 1935). The use of (2), with the clumping parameter k constant, enables us to explore the dynamical effects of parasitoid searching behaviour that is non-random or aggregated, without being engulfed in a proliferation of parameters characterizing the details of such behaviour; that is, (2) is no more than one among several ways in which some of the essentials of non-random search by parasitoids may be introduced in a simple way (Hassell & May 1973, 1974; Hassell 1978; May 1978; Perry & Taylor 1986). In reality, the outcome of a parasitoid's searching behaviour cannot usually be characterized so simply (Hassell & May 1974; Chesson & Murdoch 1986; Kareiva & Odell 1987; Perry & Taylor 1986). For example, the clumping parameter k may depend explicitly on the overall host density, as shown in figure 1*a*. In this case the decrease in clumping as host density rises reflects the more even distribution of parasitism from patch to patch as the host population gets more abundant (Hassell 1980). Such changes in k with host population size have only a minor effect on the dynamical properties of the interaction (see below).

In the simplest form of the model, defined by (1) and (2), we ignore density dependence

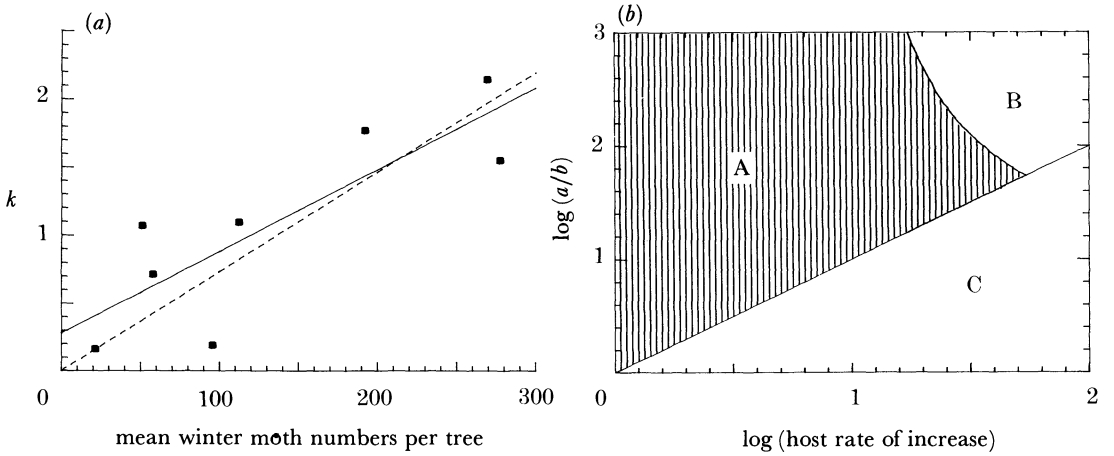

FIGURE 1. (*a*) The relation between the degree of clumping of parasitoid larvae per winter moth host (expressed by the parameter k from the negative binomial distribution), and the mean winter moth numbers per tree. Solid line $Y = 0.28 + 0.006X$; b_{yx} (slope) ± 0.004. Broken line: regression constrained through the origin, $Y = 0.007X$. (*b*) Local stability boundaries for the model in equations (1) and (2), in terms of the parameter combination a/b (where a is the parasitoid searching efficiency and b is the slope (equal to 0.006) of the regression in (*a*). The model is stable in region A and shows expanding oscillation in regions B and C. (With k constant, the model is locally stable in both regions A and B, which corresponds to the condition $k < 1$.) (After Hassell (1980).)

and handling time ($g(fN_t) = 1$ and $T_h = 0$), whereupon the population dynamics are easily understood. The equilibrium populations are given by $N^* = P^*/(1-1/F)$ and $P^* = (k/a)[F^{1/k}-1]$, and the system is stable, with disturbances damping back to the equilibrium state, for all k less than 1. In short, increasing the contagion in the distribution of parasitoid attacks promotes stability. Interestingly, values of k have been estimated for several species of insect parasitoid (see, for example, papers by Broadhead & Cheke 1975; Hassell 1980; Elliott 1982, 1983; Perry & Taylor 1986), and in almost all cases the mean value is less than unity. Making k a function of host density, as in figure 1*a*, does not affect this conclusion; it merely makes the condition for stability a little more restrictive, as shown in figure 1*b*.

More generally, if we take account of other density-dependent factors (apart from parasitoids) influencing host abundance, the parasitoid population may not be able to maintain itself. Specifically, suppose that the host population in the absence of parasitoids fluctuates about some long-term average density or 'carrying capacity', K (given from (1) by $Fg(K) = 1$). The parasitoids will be unable to invade and persist in this system (that is P_t will not increase from low levels) if K is below a threshold value K_T, given (for $T_h = 0$) by

$$K_T = 1/ac. \tag{3}$$

Note that, as expected, this threshold host density decreases (so that parasitoid persistence is easier) as parasitoid searching efficiency, a, increases.

Comparable models framed as differential equations, and thus appropriate to situations with completely overlapping generations, have also been developed (see, for example, Leslie & Gower 1960; Pielou 1969; Murdoch & Oaten 1975). We shall, however, restrict ourselves here to interactions in which generations are effectively discrete and non-overlapping. Not only are these common in temperate regions (e.g. univoltine insect pests attacking fruit and timber trees), but also they can arise in much less seasonal, uniform conditions, as a direct consequence of the host–parasitoid interaction itself (Auslander *et al.* 1974; and see below).

Classical biological control involves the introduction of a natural enemy against a pest

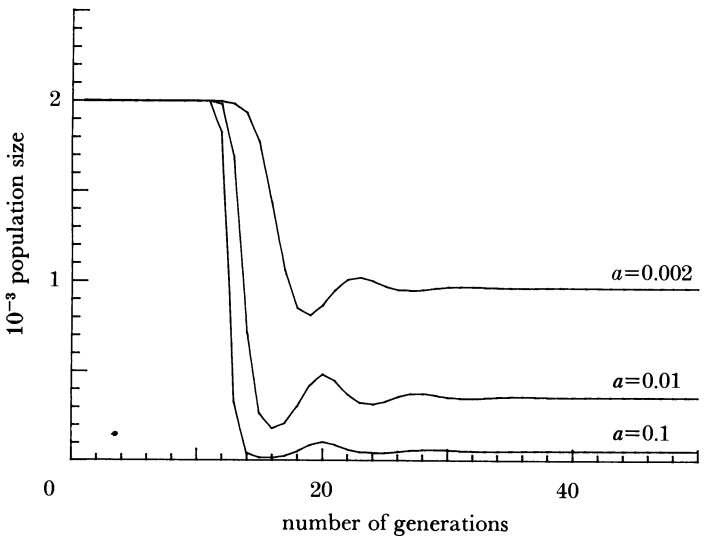

FIGURE 2. Numerical example from the model in equations (1) and (2), showing a host population reduced from its carrying capacity by the introduction of parasitoids in generation 10. The three lines show the different outcomes with parasitoids of different searching efficiencies as shown. Other parameters: $F = 3$, $k = 0.5$.

population fluctuating around its carrying capacity. Whether the parasitoid can establish itself and, if so, whether the host population is unstable or persists closely around a new lower level (as in the examples in figure 2) will depend upon several components of the interaction, such as the nature of the function f, whether the host rate of increase is density dependent, and whether c is a function of parasitoid density (e.g. due to density dependence in parasitoid sex ratios (Hassell *et al.* 1983)). The degree of 'depression' of the host population below its carrying capacity was defined by Beddington *et al.* (1975) as the ratio, q, of host average abundances before and after the introduction of parasitoids (i.e. $q = K/N^*$, where K is the carrying capacity in the absence of the parasitoid; see figure 2). The magnitude of q, and therefore the success of the parasitoid as a biological control agent, depends upon the balance between:

(1) the host's net rate of increase ($Fg(fN_t)$ in (1)), and

(2) the various factors affecting overall parasitoid performance, such as the *per capita* searching efficiency (a) and maximum attack rate of adult females, the spatial distribution of parasitism in relation to that of the host, and the sex ratio and survival of parasitoid progeny.

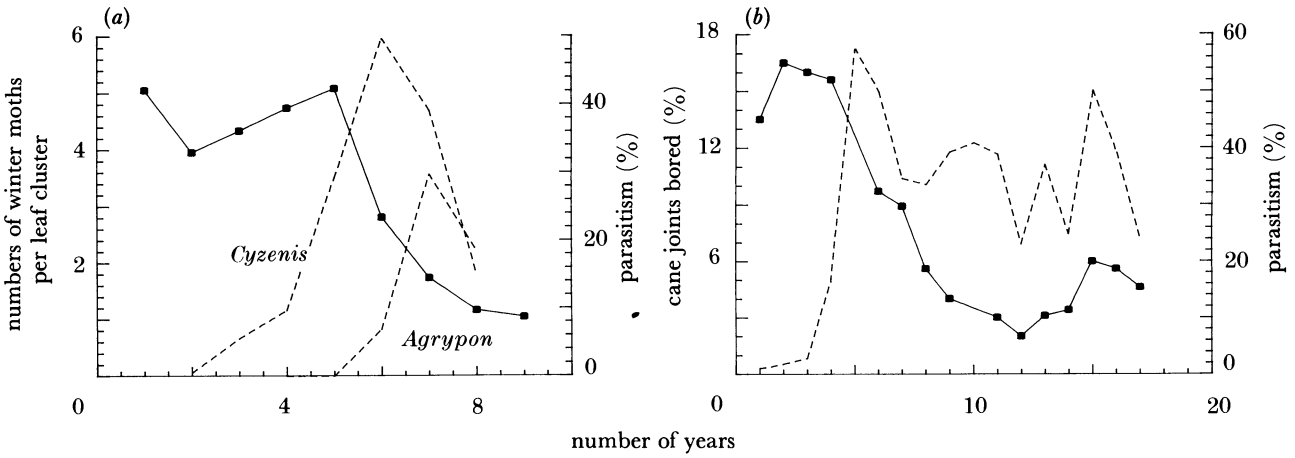

FIGURE 3. The biological control of two insect pests after the introduction of parasitoids. (*a*) The winter moth (*O. brumata*) in Nova Scotia by the tachinid *C. albicans*, and the ichneumonid *Agrypon falveolatum* (after Embree 1966). (*b*) Sugar cane stem borers (*Diatraea* spp.) by the tachinid *Lyxophaga diatraeae*, and the braconid *Apanteles falvipes* (after Anon 1980). Broken lines indicate percent parasitism (From Waage & Hassell (1982).)

Cases of successful biological control have never been monitored sufficiently before, during and after the release of natural enemies to enable convincing pictures to be drawn in the manner of figure 2. Figure 3*a*, *b* shows two of the best examples. In the case of the winter moth in Nova Scotia, although not properly quantified, it is known that the populations have persisted at very low levels since accurate sampling ceased (see below). A much clearer example of what natural enemies can do is given in figure 4, showing a resource-limited host population in the laboratory declining after the introduction of parasitoids, and then persisting in a relatively stable host–parasitoid interaction.

In one case, at least, the parameters in (1) and (2) have been estimated for a successful biological control programme; that of the winter moth (*Operophtera brumata*) in Nova Scotia, which was successfully controlled in hardwood forests after the establishment of the two parasitoids from Europe (figure 3*a*). The information comes from two independent studies on

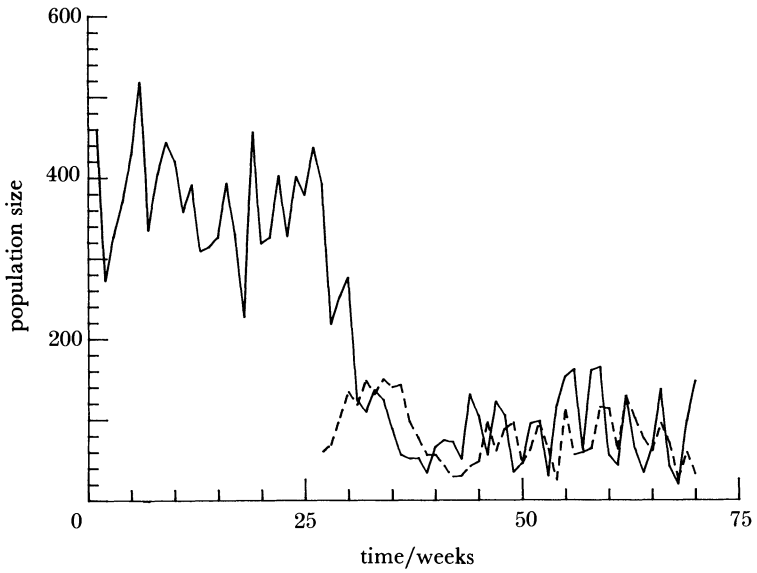

FIGURE 4. The depression in population size of the bruchid beetle *Callosobruchus chinensis* (—) after the introduction of the pteromalid parasitoid *Anisopteromalus calandrae* (----) in week 26 of the interaction. Populations maintained in the laboratory at 30 °C and 70% relative humidity, with black-eyed beans as the host resource (V. A. Taylor & M. P. Hassell, unpublished results).

the winter moth and its natural enemies: one from a natural habitat in Wytham Wood, Oxford, U.K., (Varley *et al.* 1973; Hassell 1980), and the other in Nova Scotia where the winter moth was accidentally introduced in the 1930s (Embree 1965, 1966). By using the winter moth data from Nova Scotia and the detailed information on one of the parasitoids (the tachinid fly *Cyzenis albicans*) from Wytham Wood (Hassell 1968, 1969*a*, *b*), the dynamics of the Nova Scotia interaction can be described by

$$N_{t+1} = FN_t s_e s_p \left\{1 + \left(\frac{aP_t}{k}\right)\right\}^{-k}, \tag{4a}$$

and

$$P_{t+1} = 0.65N_t \left[1 - \left\{1 + \left(\frac{aP_t}{k}\right)\right\}^{-k}\right]. \tag{4b}$$

Here, $F = 89$; s_e (equal to 0.02) is the mean fractional survival of winter moth between the estimated egg and prepupal stages; and s_p (equal to 0.65) is the mean survival of winter moth pupae in the soil, probably due mainly to generalist predators (the same affects the *C. albicans* pupae, as shown in (4*b*)). Finally, a (0.14 m^2 per generation) is the searching efficiency of *C. albicans* and k $(0.28 + 0.006N_t)$ is the degree of clumping of parasitism within hosts, both determined at Wytham Wood (Hassell 1980); note the explicit dependence of the clumping parameter k upon host density (see figure 1*a* above).

The predictions of this model are shown in figure 5. The small equilibrium populations are the result of the balance between (*a*) the low net rate of increase of the winter moth once it is discounted by the mortalities other than parasitism (i.e. $Fs_e s_p = 1.16$) and (*b*) the high overall *per capita* efficiency of the parasitoids (a combination of a high value of a (equal to 0.18) and the relatively high survival of parasitoid progeny (0.65)). The persistence of the populations at these low levels is entirely due to the clumped distributions of parasitoid progeny among the total host population resulting from the small values of k in (4). Without these clumping effects

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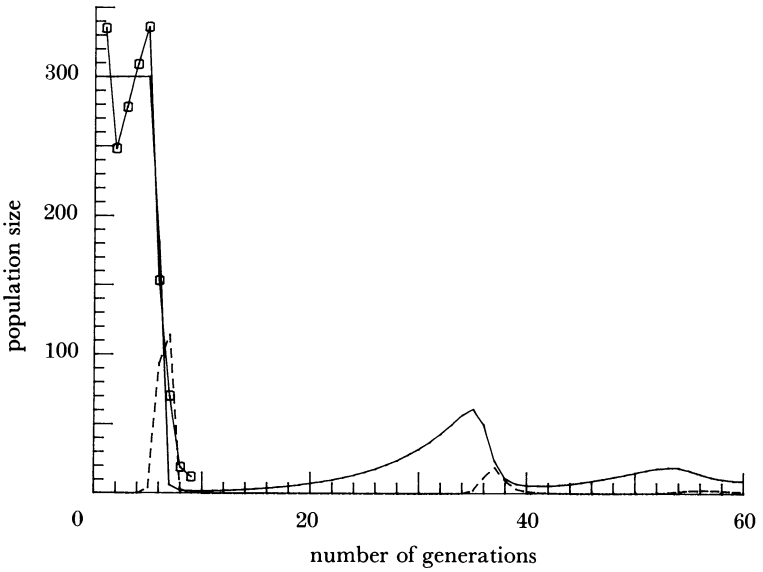

FIGURE 5. Biological control of the winter moth in Nova Scotia. Solid and broken lines give the host and parasitoid populations per square metre of canopy area, respectively, predicted from the model in equation (4). Parameter values: $F = 89$, $s_e = 0.02$, $s_p = 0.65$, $a = 0.14$ m^2 and $k = 0.28 + 0.006Nt$. Solid points give the observed winter moth larval populations (data from Embree 1966). (For further details see Hassell (1980).)

(i.e. assuming random parasitism), there is no regulatory mechanism and the model is unstable.

The values of k in figure 1*a* were obtained by fitting the negative binomial distribution to frequency distributions of *C. albicans* larvae within the salivary glands of dissected winter moth larvae collected at Wytham Wood (figure 6*a*). Behaviourally, these patterns arise from the tendency of adult *C. albicans* to oviposit preferentially in areas of high leaf damage, thus causing higher probabilities of parasitism in areas with higher winter moth density, as shown in figure 6*b* (Hassell 1968). Examples of such spatial density dependence are matched in the literature by as many others that are inversely density dependent, and by those that show no relation at all. They may all be explained, however, in the same terms of different allocations of foraging time between patches and differing constraints on the maximum attack rate within a patch (Hassell 1982*a*; Lessells 1985). Interestingly, both the direct *and* inverse patterns can contribute markedly to population regulation, with the details of the host distribution determining which is the more important in any particular case (Hassell 1984).

Such variation in the fate of host individuals in different patches is only one of several different ways in which within-generation heterogeneity can arise. Alternatively, some hosts may be protected from parasitism within some form of explicit physical refuge (Maynard Smith 1974; Hassell 1978), or there may be some degree of temporal asynchrony between parasitoids and hosts (a refuge in time; Griffiths (1969); Hassell (1969*a*)), or hosts may vary in some phenotypic character(s) that make(s) some individuals more able to resist parasitism than others (Hassell & Anderson 1984). All these mechanisms, however, share the property of rendering some individuals more susceptible to parasitism than others, giving different probabilities of parasitism for different host individuals or groups of individuals. This in turn creates a partial refuge for the host population, which promotes the stability of the interaction (Bailey *et al.* 1961; Hassell & May 1973).

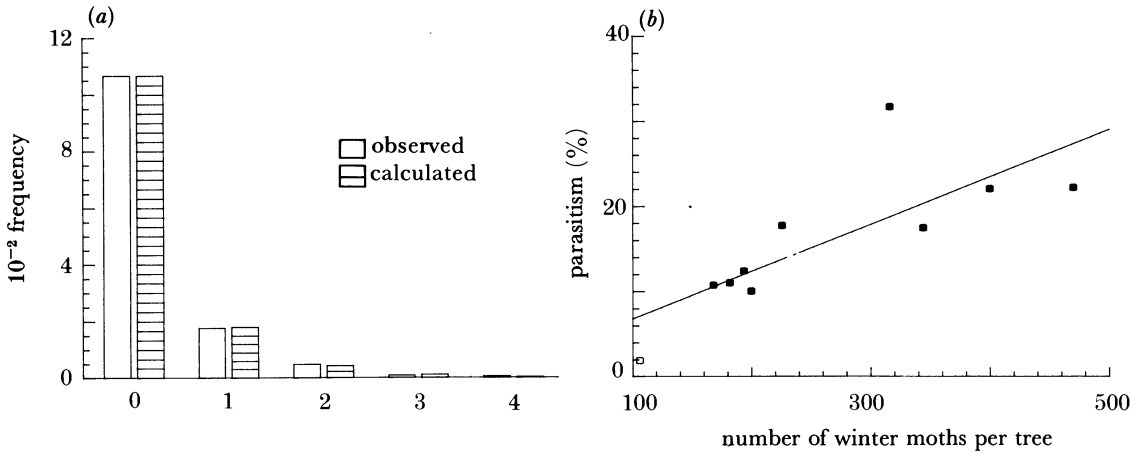

FIGURE 6. (*a*) The observed frequencies of winter moth larvae containing different numbers of *C. albicans* larvae, compared with the expected values from the negative binomial distribution with $k = 0.55$. (*b*) The same data as in (*a*), but now partitioned to show the different levels of parasitism on trees of different local winter moth density ($Y = 1.250 + 0.055X$; $r = 0.772$, $P < 0.01$). (After Hassell & May (1985).)

We have, therefore, a picture in which specialist, synchronized parasitoids can make effective biological control agents provided that their overall *per capita* efficiency (in relation to the discounted host's net rate of increase) is high enough to cause a marked depression of the host equilibrium. In general, however, this 'overall efficiency' is not simply the usual instantaneous measure, a, of equations (2) and (4a, b). Also included should be any constraints on parasitoid performance, such as a low maximum attack rate, male-biased sex ratios, mortalities of parasitoid progeny, and distributions of parasitism that are not well correlated with the distribution of hosts (i.e. course-grained). Including these factors gives more complex measures of parasitoid efficiency, such as

$$A = \frac{1}{n}\sum_{n=1}^{n}\left\{\frac{1}{P_i T_i}\ln\left(\frac{N_i}{N_i - P_e}\right)\right\}. \qquad (5)$$

Here, n represents the number of patches, $P_i T_i$, the number of adult parasitoids and the average time they spend searching for N_i hosts, and P_e the number of adult parasitoids emerging, all within patch i (Hassell 1982*b*). Along with such high parasitoid efficiency, successful biological control also requires some regulatory mechanism(s) that minimize(s) the chance of future outbreaks. Obvious density-dependent processes, such as resource limitation on host density or mutual interference between parasitoids, are unlikely candidates when populations are sparse. The various mechanisms generating heterogeneity in the distribution of parasitoid attacks are, however, as marked, or more so, when populations are sparse, and so provide a plausible and potentially powerful means of regulating interactions associated with biological control (Beddington *et al.* 1978).

This conclusion stems from models in which there is complete redistribution of host and parasitoid individuals within the habitat in each generation. All patches are thus recolonized afresh each generation, and this is clearly appropriate for many, more mobile species. At the other extreme, however, there are interactions in which hosts and parasitoids can interact within a patch over several generations, with only a certain amount of mixing and recolonization of patches in each generation. Scale insects and their natural enemies, or predator–prey interactions involving mites, provide good examples. In such cases, local

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extinction of hosts and parasitoids within patches can easily occur (Murdoch *et al.* 1985; Chesson 1981), and the persistence of the system as a whole is dominated by the degree of asynchrony between the state of the different patches rather than by the spatial distribution of parasitism *per se* (Sabelis & Laane 1986; Sabelis & van der Meer 1986). There is thus a continuum from cases of more or less complete mixing of the subpopulations each generation (e.g. winter moth) where heterogeneity in the distribution of parasitism is likely to be very important for regulation, to cases in which large fractions of the host and parasitoid populations do not move from their patches and where persistence is largely governed by the temporal refuge effect of host patches being colonized at different times by the natural enemies (J. K. Waage, M. Sabelis & M. P. Hassell, unpublished results; see Waage (this symposium) for further details). The ideal attributes of a successful biological control agent in the latter case have yet to be explored, but clearly they will include the ability of the natural enemies to locate host patches and, once there, to exploit the host sub-population rapidly by the natural enemies' combined functional and numerical responses.

(*b*) *Hosts and pathogens*

Viruses, bacteria, Protozoa and fungi can kill insects, and such pathogens have been deliberately used in efforts to control particular insect pests. The use of the bacterium *Bacillus thuringiensis* against the gypsy moth, *Lymantria dispar*, in North America is probably the best-known example. A variety of other case studies are reviewed by Tinsley & Entwhistle (1974), Tinsley (1979) and Falcon (1982). Unlike the work involving parasitoids, such empirical efforts to use pathogens as agents of biological control have, until recently, not been accompanied by analysis of the underlying dynamics of insect host–pathogen systems. Such a theoretical framework has recently been explored by Anderson & May (1981), who indicate applications to specific interactions between some insect pests and viral or microspordian protozoan pathogens. What follows is a brief summary of this work, which is still in its formative stages.

Consider first a host population with discrete, non-overlapping generations, whose average density is regulated by a lethal pathogen that spreads in epidemic fashion through each generation before reproduction. The dynamics of this population is again described by equation (1*a*) (where $g = 1$ in the absence of other density-dependent effects):

$$N_{t+1} = FN_t f(N_t). \tag{6}$$

The difference between this and (1*a*) is that $f(N_t)$ now represents the fraction escaping infection (rather than escaping parasitoid attack). This fraction escaping infection as an epidemic spreads through a population of density N_t is given implicitly by the Kermack–McKendrick expression, $f = \exp\{-(1-f)\,N_t/N_T\}$ (Kermack & McKendrick 1927); here N_T is the threshold host density (which depends on the virulence and transmissibility of the pathogen). For N less than N_T, the epidemic cannot spread ($f = 1$), whereas the infected fraction, f, falls to ever smaller values as N increases above N_T. This simple, natural and purely deterministic model exhibits completely chaotic behaviour: the system has no stable equilibrium point, nor any stable cycles, but rather fluctuates (tending to alternate between relatively high and relatively low population densities) in such a way that the deterministic dynamics are effectively indistinguishable from the sample function of some random process (for a more detailed discussion, see May (1985)).

Equation (6), with its bizarre dynamics, is the analogue (for regulation by a pathogen) of the familiar Nicholson–Bailey equation (for regulation by a parasitoid). Had (6) been studied sooner, chaotic dynamics may have forced themselves upon our attention earlier, and the history of several subjects might be different. As it is, the dynamical properties of (6) have been appreciated too recently for them yet to have motivated any systematic study of field or laboratory populations. Most of the insect host–pathogen systems that have received attention differ from the model of (6) in that transmission is via free-living infective stages of the pathogen (rather than by direct contact between susceptible and infected hosts, as in the Kermack–McKendrick assumption), and/or host generations to some extent overlap (so that differential, rather than difference, equations are appropriate). Largely for these reasons, studies of insect host–pathogen dynamics have mainly used differential equations, representing the approximation that populations of hosts and pathogens change in a continuous way.

In the simplest such model, the host population is assumed to have a *per capita* birth rate a and a *per capita* death rate b from all causes other than the pathogen, both of which rates are density independent. The total population $N(t)$ is partitioned into uninfected and infected hosts, with population densities $X(t)$ and $Y(t)$, respectively; $N = X+Y$. Although invertebrate species are usually able to mount cellular or humoral responses to infection, current evidence suggests they are not able to develop acquired immunity to agents of infectious disease (Anderson & May 1981). Thus the basic model is lacking the class of recovered-and-immune hosts found in conventional epidemiological models for vertebrates. The basic model further assumes that infections are transmitted directly from infected to uninfected hosts, so that the net rate at which new infections appears is βXY, where β is a transmission parameter. This description of the infection process accords with some data (Anderson & May 1981). Infected hosts either recover (at a rate γ, so that the characteristic recovery time is $1/\gamma$), or are killed by the disease (at a rate α).

When all this is put together, the dynamics of the model population are given by

$$\mathrm{d}X/\mathrm{d}t = a(X+Y)-bX-\beta XY+\gamma Y, \tag{7a}$$

and

$$\mathrm{d}Y/\mathrm{d}t = \beta XY-(\alpha+b+\gamma)\,Y\,. \tag{7b}$$

New susceptibles appear by birth (from either infected or uninfected individuals) or by recovery from the infected state; infectious individuals appear at the rate βXY, and remain infectious for an average time of $1/(\alpha+b+\gamma)$ before they die of the disease or of other causes, or recover. The overall population density obeys

$$\mathrm{d}N/\mathrm{d}t = rN-\alpha Y. \tag{7c}$$

Here we have defined r equal to $a-b$ as the *per capita* rate of population growth in the absence of the pathogen.

In the absence of infection (Y equal to 0, N equal to X), this model population will grow exponentially at the rate r. If a small number of infectious individuals are introduced into such a disease-free population (corresponding to X approximately equal to N, Y much less then N), the infection will spread and establish itself provided the right-hand side of (7b) is positive; this will be the case if $N > N_T$, where the threshold host density N_T is defined as

$$N_T \equiv (\alpha+b\ +\gamma)/\beta. \tag{8}$$

Given the exponential growth of this host population in the absence of the disease, the threshold will always be attained eventually, and thus the infection can always establish itself sooner or later. More generally, if other density-dependent factors regulate the host population around some average value K in the absence of disease, then the pathogen can invade only if $N_T < K$; this result is directly analogous to the earlier threshold results for parasitoid establishment.

Once established, the virus, bacterium, or other infectious agent can, in the absence of any other density-dependent effects, regulate the host population density, provided it is sufficiently pathogenic:

$$a > r. \tag{9}$$

If this regulatory criterion is met, then in the simplest model the host population is regulated to a constant equilibrium value, N^*, given by $N^* = [\alpha/(\alpha - r)]\, N_T$, with N_T given by (8). The fraction of this equilibrium host population that is infected at any one time is simply $Y^*/N^* = r/\alpha$; perhaps surprisingly, the more virulent the infection (the larger α), the smaller the equilibrium fraction infected. On the other hand if (9) is not satisfied, the host population continues to grow exponentially at a diminished *per capita* rate, $r' = r - \alpha$, until other kinds of regulatory effect can no longer be ignored.

Table 1 lists some estimates of *per capita* mortality rates induced by pathogens, α, and from other natural causes, b, for a miscellany of invertebrate hosts and viral, bacterial, protozoan, and fungal infections. Table 1 lacks corresponding estimates of the *per capita* birth rates, a, and consequently lacks comparisons between α and r; there are, unfortunately, very few field studies that give estimates of all three parameters. Thus it is hard to say how often α exceeds r in the field. The data compiled in table 1 do no more than indicate that many disease agents of invertebrates are highly pathogenic, with the most pronounced pathogenicity tending to arise among viral and bacterial agents. For a more full discussion, both of the theory and the data, see Anderson & May (1981).

Notice that, just as self-maintaining parasitoids cannot in general eradicate their hosts, so too pathogens will not in general be capable of eradicating the target pest species; once the host population is driven to sufficiently low densities, it will be below threshold for maintenance of the pathogen. Exceptions can arise by some of the complications discussed below; eradication could, for instance, be made possible by extreme cases of vertical transmission. Given that a self-sustaining pathogen is unable to eradicate the pest, the aim is usually to reduce the host population significantly below its disease-free average level, K (around which the pristine population may have exhibited a greater or lesser degree of density-independent fluctuation). The question thus arises, what is the degree of pathogenicity, α, producing maximum depression of the pest population?

First thoughts might suggest that the greater the pathogenicity, the better. Certainly, if $\alpha \to 0$ the pathogen has no effect on host population growth. But, at the other extreme, too large a value for α leads to too large a value of N_T (essentially because the disease kills hosts so fast that a very large host population is needed to perpetuate the infection), and thence to the regulated population having high density. As emphasized by Anderson & May (1981), the optimum self-sustaining control, in the sense of lowest equilibrium levels of pest population density, will usually be attained from an infectious disease agent with intermediate pathogenicity. For the simple model of equation (7), we can see this explicitly from the expression $N^* = \alpha(\alpha + b + \gamma)\,/\{(\alpha - r)\beta\}$, which is minimized for the intermediate value

TABLE 1. NATURAL AND PATHOGEN-INDUCED MORTALITY RATES FOR INSECT HOSTS OF SOME VIRAL, BACTERIAL, PROTOZOAN AND FUNGAL INFECTIONS

(From Anderson & May (1981), where references are given. Ewald (1987) has in some cases given independent assessments of these mortality rates, by using a somewhat different estimation procedure; his estimates are within factor-of-two agreement.)

pathogen	host	natural mortality rate, b (per week)	pathogen-induced mortality rate, α (per week)	ratio α/b (one figure accuracy)
Viruses				
sack brood virus	*Apis mellifera*	0.170	1.200	7
nuclear polyhedrosis virus	*Cadra cautella*	0.061	0.540	9
nuclear polyhedrosis virus	*Hyphantria cunea*	0.003	0.800	300
A.B.P. virus	*Apis mellifera*	0.250	1.900	8
nuclear polyhedrosis virus	*Porthetria dispar*	0.060	0.630	10
nuclear polyhedrosis virus	*Malacosoma americanum*	0.070	0.370	5
Bacteria				
Bacillus thuringiensis	*Simulium vittatum*	0.035	2.400	70
B. thuringiensis	*Choristoneura fumiferana*	0.001	4.000	4 000
Aeromonas punctata	*Anopheles annulipes*	0.360	2.900	8
Erwinia spp.	*Colladonus montanus*	0.031	0.170	5
Protozoa				
Nosema stegomyiae	*Anopheles albimanus*	0.230	0.410	2
Pleistrophora schubergi	*Hyphantria cunea*	0.003	0.036	10
Herpetomonas muscarum	*Hippelates pusio*	0.170	0.430	3
Tetrahymena pyriformis	*Culex tarsalis*	0.260	0.660	3
Fungi				
Beauveria tenella	*Aedes siemensis*	0.026	0.500	20
B. tenella	*Culex tarsalis*	0.110	0.840	8
B. bassiana	*Musca domestica*	0.270	0.740	3
B. bassiana	*Hylemya antiqua*	0.300	0.550	2
B. bassiana	*Phormia regina*	0.240	0.560	2
Metarrhizium anisopliae	*Musca domestica*	0.270	0.380	1
M. anisopliae	*Hylemya antiquia*	0.300	0.480	2
M. anisopliae	*Phormia regina*	0.240	0.420	2
Aspergillus flavus	*Culex peus*	0.020	0.170	9
A. flavus	*C. tarsalis*	0.061	0.220	4
Fusarium oxysporum	*C. pipiens*	0.027	0.620	20

$\alpha = r + \{r(a+\gamma)\}^{\frac{1}{2}}$ (and which becomes infinite for $\alpha \to \infty$ or $\alpha \to r$). This is an important point, because many control programmes assume that the most virulent pathogens are necessarily the best. This may to some extent be true for inundative release programmes, but it is not true for programmes that aim at a degree of self-perpetuation of the pathogen.

The basic model of (7) omits many biological features that can complicate host–pathogen systems. Briefly, pathogens may reduce the reproductive output of infected hosts (making it more likely that the pathogen will regulate the host population), may be transmitted 'vertically' from parent to unborn offspring (which decreases N_T, making it easier to maintain the infection in relatively low density populations of hosts), and may undergo an incubation or latent period within the host, during which time the host is infected but not yet infectious (this process tends to increase N_T, and also makes it less likely the infection will regulate the host population). The pathogenicity of the infection may, moreover, depend on the nutritional state of the host, which in turn is likely to depend on the host population density; under these circumstances, the host population may have two alternative stable states, and pass from one

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TABLE 2. SUMMARY OF THE WAY IN WHICH SOME REALISTIC COMPLICATIONS CAN AFFECT THE ABILITY OF A PATHOGEN TO REGULATE ITS HOST POPULATION, AND TO PERSIST WITHIN A POPULATION OF HOSTS THAT EITHER IS OF LOW DENSITY OR FLUCTUATES WIDELY IN ABUNDANCE

complicating factor	effect on regulatory capacity of pathogen (relative to basic model defined by equation (7))	effect on threshold host density to maintain pathogen (relative to basic model)
diminution in reproductive capacity of infected hosts	regulation is easier; regulated host population density is lower	no effect on threshold criterion
vertical transmission	no effect on regulatory condition; regulated host population density is lower	threshold host density lower (can be arbitrarily small)
latent period of infection	regulation is harder; regulated host population density is higher	threshold host density is higher
pathogenicity is stress related	can always regulate if pathogenicity is sufficiently strongly enhanced by stress	pathogen can persist provided transmission efficiency is high enough
other density-dependent constraints on host population growth	disease-related depression of host population density maximized for an intermediate degree of pathogenicity	pathogen cannot persist if threshold host density is too high (if $N_T > K$)
free-living infective stages of pathogen	regulatory criterion as for basic model; regulated state may be a stable point or cyclic oscillations	easier to maintain pathogen, especially if infective stages are long-lived

to the other discontinuously. The effects of these complications are summarized in table 2, and discussed in detail elsewhere (Anderson & May 1981).

A major complication arises when the free-living transmission stages of the pathogen are long-lived, as happens for many pathogens of insects. Such free-living infective stages include the spores of many bacteria, protozoans and fungi, and the capsules, polyhedra or free particles of viruses (Tinsley 1979). In particular, baculoviruses of univoltine insects of temperate forests (principally of lepidopteran, hymenopteran and dipteran species) tend to have long-lived infective stages, partly because the soil environment of temperate forests affords relative protection from the ultraviolet components of sunlight (Jacques 1977). In the event of such complications, we define the population of free-living infective stages of the pathogen to be $W(t)$ at time t; in (7b) the transmission term is now proportional to the rate of encounters between susceptible hosts and infective stages of the parasite, νWX:

$$\mathrm{d}Y/\mathrm{d}t = \nu WX - (\alpha + b + \gamma)\, Y. \tag{10}$$

Equation (7c) for $N(t)$ remains unchanged. Infective stages are produced at a rate λ from infected hosts, and are lost by death (at a rate μ) or by absorption in hosts (at a rate νN), which gives

$$\mathrm{d}W/\mathrm{d}t = \lambda Y - (\mu + \nu N)\, W. \tag{11}$$

The three equations (7c), (10) and (11) give a complete description of the dynamical behaviour of the variables $N(t)$, $Y(t)$ and $W(t)$. Provided infected hosts produce transmission stages of the parasite at a sufficiently fast rate (specifically, $\lambda > \alpha(\alpha + b + \gamma)/(\alpha - r)$), the pathogen will again regulate its host population so long as $\alpha > r$. However, the regulated state may be a stable point, or it may be a stable cycle. The cyclic solutions tend to arise for infections of high pathogenicity that produce large numbers of long-lived infective stages. In effect, the 'seedbank' of long-lived transmission stages can induce oscillations by the time lags it introduces into the system; the longer the life of transmission stages in relation to the characteristic regulatory time-scale (set largely by $1/\alpha$), the greater the propensity to oscillation.

Many microsporidian protozoan and baculovirus infections of insects appear to possess the combination of a relatively large α and a small μ that produces cyclic changes in host

Table 3. Cyclic variations in the abundance of forest insect species (from Anderson and May (1981), where references are given)

host insect species	locality	period of cycles in population abundance (years)	pathogen
Orgyia pseudotsugata (Douglas-fir tussock moth)	North America	7–10	nuclear polyhedrosis virus
Acleris variana (black-headed budworm)	eastern Canada	10–15	nuclear polyhedrosis virus
Bupalus piniarius (pine looper)	Europe	5–8	nuclear polyhedrosis virus
Zeiraphera diniana (larch bud moth)	Europe	9–10	granulosis virus
Diprion hercyniae (spruce sawfly)	North America	8	nuclear polyhedrosis virus
Malacosoma disstria (tent caterpillar)	North America	8–12	nuclear polyhedrosis virus

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abundance. For the forest insect pests listed (with associated pathogens) in table 3, insertion of reasonable estimates of α, μ, and r in (7c), (10) and (11) suggests cycles with periods in the general range 3–30 years, in approximate accord with the observed periods of 5–12 years for these pests. Only for the larch budmoth, *Zeiraphera diniana*, and an associated granulosis virus could Anderson & May (1980) find sufficient data to estimate all parameters in the relevant model. In this one instance they did find an encouraging fit between the observed and theoretically estimated periods of the oscillations in host density, and in the cyclic patterns of prevalence of infection (although the agreement between the observed and theoretically estimated amplitudes of the cycles in host density were less encouraging, with the observed amplitude being an order of magnitude larger than the theoretical estimate). McNamee *et al.* (1981) have refined the analysis, but there remains much scope for further research, particularly in field studies of forest insects and their pathogens.

Augmentative releases

(*a*) *Release of parasitoids*

The regular, inundative release of mass-reared parasitoids, most often *Trichogramma* spp. that attack the egg stage of a wide range of insect species, has been practised since early in this century as an alternative to classical biological control, particularly against lepidopterous pests (DeBach & Hagen 1970; Ables & Ridgeway 1981). The efficacy of such programmes is limited by a number of factors (Stinner 1977), which are now listed.

(1) Mass rearing can often decrease the fecundity, longevity and searching efficiency of parasitoids (Stinner *et al.* 1974).

(2) Released parasitoids may rapidly disperse away from the crop in question. Possible remedies for this are to 'pretreat' parasitoids with host kairomones to stimulate search after release (Gross *et al.* 1975), or to apply the kairomones directly to the crop to act as arrestants for the released parasitoids (Lewis *et al.* 1975; Waage & Hassell 1982).

(3) The released individuals may act as a kind of biological insecticide in the sense that none of the parasitoid progeny survive or remain in the area to parasitize future pest generations. This differs in spirit somewhat from programmes that aim to augment an established population of natural enemies of the same or different species.

(4) Lepidopterous pests generally suffer high mortality among early larval stages. This has led to controversy and conflicting reports on the potential impact of released egg-parasitoids in relation to other later occurring mortalities (see, for example, Myers 1929; Box 1932; Pickles 1936; Metcalfe & Breniere 1969). In addition, in the case of graminaceous stalk borers, this early larval mortality is likely to be density dependent owing to competition for the limited space within a stem, as shown by the example in figure 7 for the sorghum stalk borer, *Chilo partellus*, (van Hamburg & Hassell 1984). This raises additional problems for parasitoid release, as discussed in relation to the models below.

A theoretical treatment for several models of periodic mass release of parasitoids has been given by Barclay *et al.* (1985). Before this the only theoretical discussion of such inundative releases is that of Knipling & McGuire (1968) (see also Knipling 1972), who used simple numerical examples of the release of Nicholson–Bailey parasitoids to investigate how levels of parasitism were affected by the density at which parasitoids were released and by their searching efficiency. The examples considered by Barclay *et al.* (1985) are all for discrete

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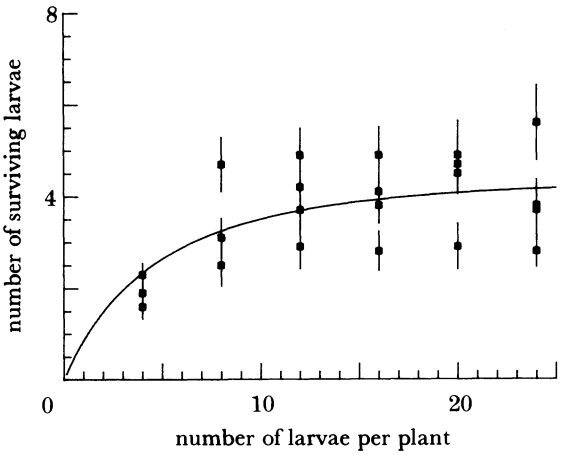

FIGURE 7. The density dependent relationship between the number of surviving larvae (S) of the sorghum stalk borer (*Chilo partellus*) and the first instar larvae per plant in a glasshouse experiment. Means ($\pm$standard errors (s.e.)) of 15 replicates are shown, and the data are described by the model, $S = N(1+dN)^{-b}$, fitted by nonlinear least squares ($d = 0.15 \pm 0.09$, $b = 1.16 \pm 0.41$ (s.e.)). (After van Hamburg & Hassell (1984).)

host–parasitoid interactions, as in equation (1). In what follows, we revisit some of their results, and also discuss the impact of augmentative releases where the pest is already attacked by a different parasitoid species (see also Appendix 1). The analogous studies of criteria for eradicating pests by pathogen release are presented elsewhere (Anderson & May 1981), and are only briefly summarized here.

Consider a simple extension of the model in (1):

$$N_{t+1} = FN_t g(fN_t) f(P_t) \tag{12a}$$

and

$$P_{t+1} = cN_t\{1 - f(P_t)\} + R. \tag{12b}$$

Here, R represents the population of the released parasitoids in each generation, and the function f is given by (2). If we neglect for the moment the host density dependence in (12a) (i.e. put $g = 1$), our conclusions from this model are straightforward (the results are derived in Appendix 1).

(1) There is a linear decline in the host equilibrium level as R increases, leading to eradication when $R > P^*$ (where P^* is the pristine equilibrium density of parasitoid adults, before any programme of releases); see figure 8a.

(2) Parasitoid releases insufficient to eradicate the host promote stability, to the point where even a Nicholson–Bailey interaction (random search) can be stabilized if the releases are large enough; see figure 8b.

Equations (12a, b) are for the particular case of parasitism acting first in the host's life cycle, followed by an additional density-dependent mortality acting at a later host stage, as in the sorghum stalk borer example above. The degree of depression in the host population, caused by the releases of parasitoids, now depends critically on whether the host density dependence is under-, exactly or over-compensating (figure 9). Releases will always complement under-compensating density dependence ($b < 1$) and lead to lower equilibria than if either acted alone (figure 10a). However, as the density dependence becomes more severe, there is a growing risk that any parasitoid releases will be valueless (i.e. the density dependence compensates for the additional parasitism) or, worse still, may actually lead to higher pest

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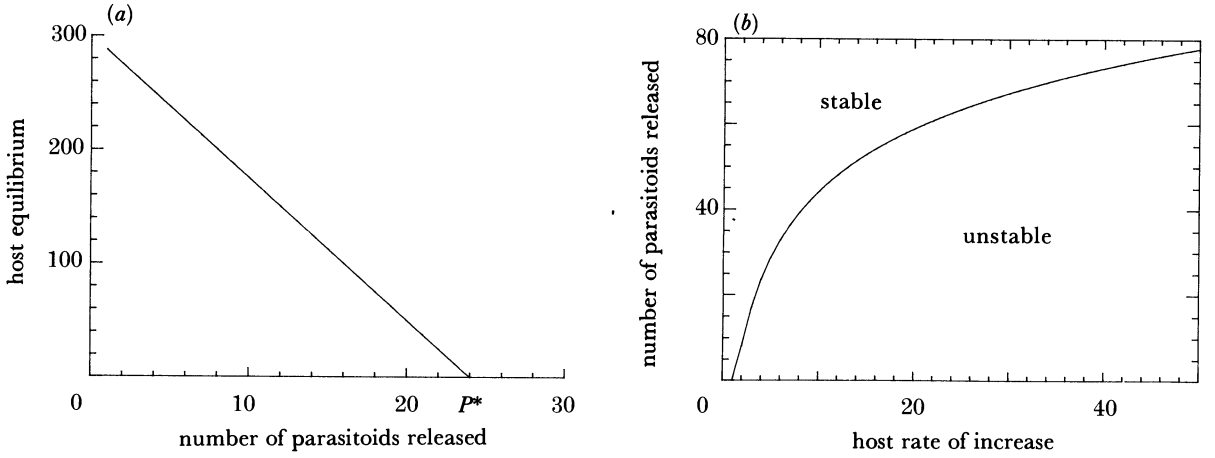

FIGURE 8. Equilibrium and stability properties of the model in equation (12). (*a*) The decline in host equilibrium levels (N^*) as R, the number of parasitoids released, increases. Eradication occurs when $R > P^*$, where $P^* = (k/a)(F^{\frac{1}{k}-1})$, $N^* = (P^*-R)/(1-1/F)$ and $a = 0.05$, $k = 0.5$ and $F = 5$. (*b*) Stable and unstable regions in relation to the number of parasitoids released, R, and the host rate of increase, F, with parameters as in (*a*) except $k \to \infty$ (i.e. random parasitism).

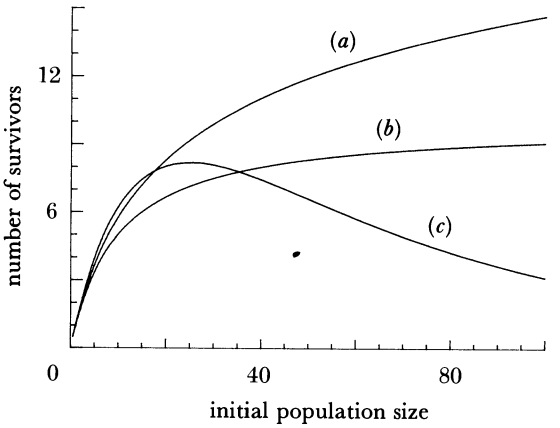

FIGURE 9. Examples of (*a*) under-compensating, (*b*) compensating and (*c*) over-compensating density dependence from the model $S = N(1+dN)^{-b}$ (see also figure 7). Parameter values: (*a*) $d = 0.1$, $b = 0.8$, (*b*) $d = 0.1$, $b = 1$; (*c*) $d = 0.01$, $b = 5$.

populations if the density dependence is over-compensating (b greater than 1), as shown by the example in figure 10*b*. It may well be that some of the conflicting results after releases of *Trichogramma* against stem borers, such as *Diatrea saccharalis* (see, for example, Box 1932; Tucker 1934*a*, *b*; Metcalfe & van Whervin 1967) result from differences in the degree of larval density dependence in different situations and areas.

Often, augmentative releases are used where the pest is already attacked by parasitoids of a different species. Once again, the order of parasitism by the different species is important to the outcome (May & Hassell 1981). With species P already present and Q released augmentatively, the possibilities are:

(*a*) Q acts first:

$$\left.\begin{aligned} N_{t+1} &= FN_t g(Q_t) f(P_t), \\ Q_{t+1} &= R + N_t\{1-g(Q_t)\}, \\ \text{and} \quad P_{t+1} &= N_t g(Q_t)\{1-f(P_t)\}. \end{aligned}\right\} \qquad (13)$$

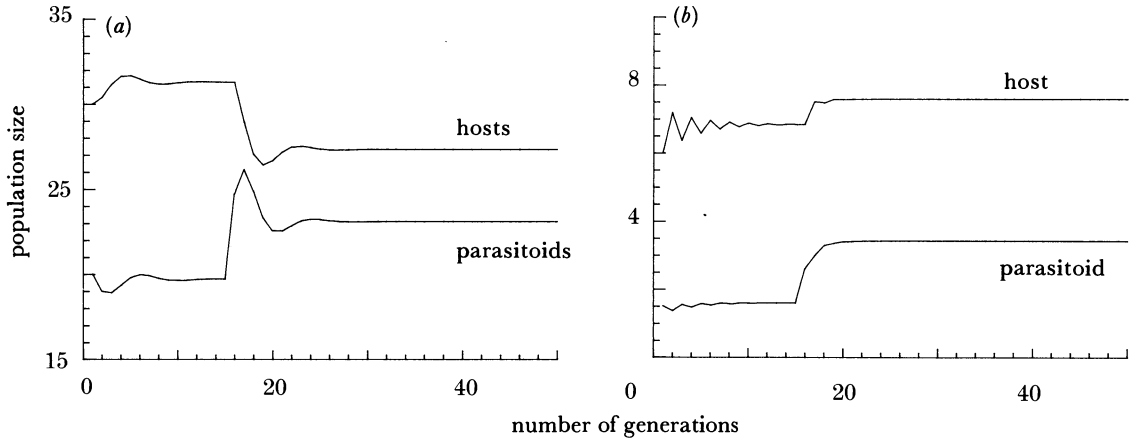

FIGURE 10. Numerical examples from the model in equation (12) with different levels of density dependence, g, defined by the expression used in figures 7 and 8. Parasitoid releases commence in generation 20. (a) The resulting depression in the host equilibrium when the density dependence is under-compensating ($d = 0.1$, $b = 0.8$, $F = 5$, $a = 0.1$, $k = 0.8$, $R = 5$). (b) The increase in host population level with over-compensating density dependence ($d = 0.05$, $b = 10$, $F = 10$, $a = 0.3$, $k = 0.8$).

(b) P acts first:

$$\left.\begin{aligned} N_{t+1} &= FN_t g(Q_t) f(P_t), \\ P_{t+1} &= N_t\{1-f(P_t)\}, \\ \text{and} \quad Q_{t+1} &= R + N_t f P_t\{1-g(Q_t)\}. \end{aligned}\right\} \tag{14}$$

(c) P and Q act simultaneously, with proportions attacked depending on their relative abundance and relative attack rates. Assuming Nicholson–Bailey attack rates,

$$\left.\begin{aligned} N_{t+1} &= FN_t \exp(-a_P P_t - a_Q Q_t), \\ P_{t+1} &= N_t\{a_P P_t/(a_P P_t + a_Q Q_t)\}\{1-\exp(-a_P P_t - a_Q Q_t)\}, \\ \text{and} \quad Q_{t+1} &= Nt\{a_Q Q_t/(a_P P_t + a_Q Q_t)\}\{1-\exp(-a_P P_t - a_Q Q)\} + R. \end{aligned}\right\} \tag{15}$$

In all three cases, augmentative releases reduce host abundance leading to eradication when $g(R) > 1/F$ (figure 11). The picture is complicated, however, by the effect of releases on the coexistence of the two parasitoid species. For cases (a) and (c) above, which are most appropriate for the release of egg-parasitoids, releases of Q will always promote to some degree the instability of the established species P. Indeed, if there are no regulatory processes in the interaction other than the releases themselves (i.e. both P and Q are Nicholson–Bailey parasitoids) species P will always become extinct unless acting before Q in the host's life cycle (case b), in which case P and Q can coexist if $aN^* = (\ln F - R)/(1-1/F) > 1$.

The success of the augmentative release of egg-parasitoids clearly depends on a number of factors, including the efficiency of the parasitoids, the survival of parasitoid progeny for the next generation, any dispersal from the target area, the presence of other parasitoid species and any density-dependent processes affecting the host population. The mix of these will undoubtedly vary in importance in different systems, and will need to be carefully evaluated for each case to avoid costly and time-consuming investments where the likelihood of success is small.

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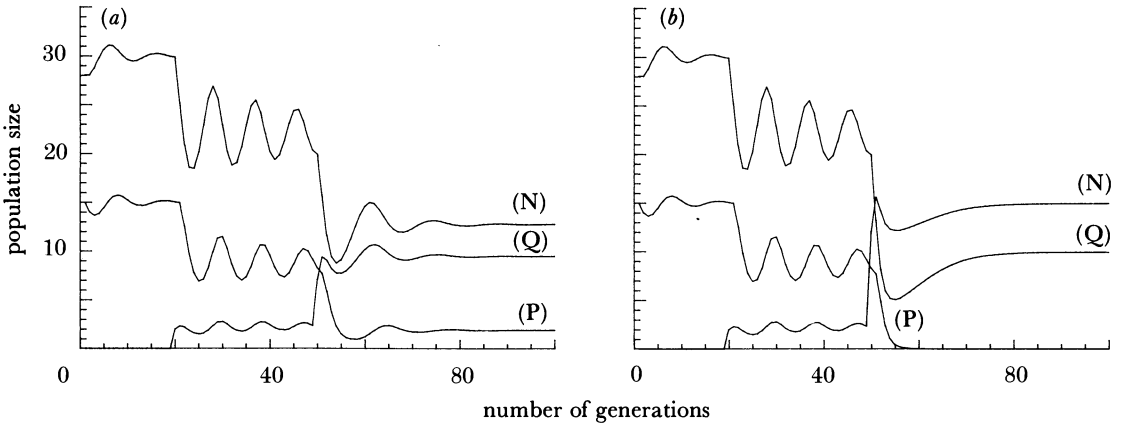

FIGURE 11. Numerical examples from the model in equation (14) showing the introduction and establishment of a second parasitoid species, Q, in generation 20. Augmentative releases of Q begin in generation 50, leading to a further reduction in the host population with (*a*) both P and Q coexisting ($R = 5$) or (*b*) Q replacing P ($R = 10$). Other parameters: $a_P = a_Q = 0.1$, $k_P = k_Q = 0.5$, $F = 2$, $g = 1$ (i.e. no additional host density dependence).

(*b*) *Release of pathogens*

For the reasons outlined above, eradication of a pest species by a pathogen will usually require continual introduction of transmission stages. We summarize here the results of Anderson & May (1981) for the critical rate, above which such transmission stages must be introduced, to achieve extinction of the pest.

We established above that for the pathogen to be capable of regulating or eradicating the target-insect pest population we require α to be greater than r (or some appropriately modified version of this relation if the infection decreases reproductive capacity, if pathogenicity is stress-related, or if other complications enter; table 2). If this criterion is obeyed, then the host population will be extinguished if free-living transmission stages of the pathogen are introduced at a rate A in excess of a critical rate Ac given by

$$Ac = \mu r(\alpha + b + \gamma) / \{\nu(\alpha - r)\}. \tag{16}$$

Here the parameters α, b, γ, and r have their usual meanings; μ is the death rate of free-living infective stages (whence their life expectancy is $1/\nu$); and ν is the rate at which infective stages successfully infect hosts (that is, ν is the transmission coefficient of free-living infective stages). Eradication efforts of this kind are more likely to be successful if they require relatively low rates of introduction of the pathogen. Thus, the criterion $A > Ac$, with Ac defined by (16), is more likely to be met for infectious agents that are highly pathogenic (α large), have long-lived transmission stages (μ small), and have high transmission efficiency (ν large). In accord with common sense, both (16) and the overriding constraint $\alpha > r$ state that pest species with high population growth rates (large r) are relatively difficult to control.

With the exception of ν, all the parameters in (16) can be measured, although there are very few host–pathogen systems for which all such measurements have been made. Direct assessment of the transmission parameter ν is, however, exceedingly difficult. It is therefore helpful to note that the critical introduction rate, Ac, can alternatively be expressed as $Ac = \lambda Y_0^*$. Here Y_0^* is the equilibrium population of infected hosts, and λY_0^* is the equilibrium net rate of production of infective stages by infected hosts, in a natural system when no pathogens are being artificially

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introduced ($A = 0$). Thus if we are using a pathogen found in natural systems and believed to have α greater than r, we have only to introduce infective stages at a net rate in excess of the rate at which they are produced by the pristine host–pathogen association, to eradicate the host population. This criterion is in direct correspondence to the result $R > P^*$ for eradication of a pest species by augmentative release of parasitoids. It seems likely that this critical rate can, for example, be estimated (and attained in practice) for some known baculoviruses of forest insect pests.

It is to be emphasized that eradication of an insect pest population by release of parasitoids or transmission stages of pathogens will usually require maintaining rates of release above the eradication threshold (R greater than P^* or A greater than Ac) for many years. Even then, constant surveillance and reintroductions are likely to be needed to prevent resurgence of the pest species.

Multispecies interactions

Many of the successes in biological control have involved the release of single parasitoid species (DeBach 1974; Messenger 1976), and of all predator–prey systems in the field, such examples come closest to the theoretician's delight of a closed two-species interaction. It is not possible, however, to develop a theoretical basis for biological control exclusively within such a simple framework. Many biological control programmes have led to the establishment of more than one parasitoid species (Clausen 1978), in others indigenous natural enemies have also played a part (Griffiths *et al.* 1984), and in yet others there has been the interplay between natural enemies and pathogens (Magasi & Syme 1984). Such interactions require the development of more complex, multispecies models involving mixes of parasitoids, predators and pathogens. These can show if the dynamics of simple multispecies systems are just the expected blend of the separate pairwise interactions, or if they have unexpected properties of their own. In biological control, the study of such systems may shed light both on how easily an introduced parasitoid can invade a community in which natural enemies of different types are already present and on how effective the introduced species (if it is indeed established) could be in lowering host populations and promoting persisting control.

(*a*) *Multiparasitoid introductions*

A longstanding debate in biological control has centred on whether preliminary screening should be aimed at identifying the single most efficient parasitoid before release, or whether several species of parasitoids should be introduced for greatest effect. Some have suggested that interspecific interference between introduced parasitoid species may actually result in less depression of host abundance than achieved by introducing a single species (Turnbull & Chant 1961; Watt 1965; Kakehashi *et al.* 1984); others have suggested that multiple introductions will often provide a greater degree of host depression, or at least are a good way of identifying the best species without any attendant risks of diminished control (see, for example, van den Bosch & Messenger 1973; Huffaker *et al.* 1971; May & Hassell 1981; Waage & Hassell 1982).

The dynamics of multiparasitoid introductions can be conveniently considered within the framework of (13) and (14) (but without the augmentative releases; i.e. $R = 0$), and either with or without a density-dependent host rate of increase (May & Hassell 1981). In this case, it is assumed that species P is already established before the introduction of species Q, which

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either acts on the surviving hosts left by P or, if attacking the same host stage, is intrinsically inferior as a larval competitor whenever multiparasitism occurs. Three principle predictions emerge.

(1) As expected, the two species of parasitoids are most likely to coexist if each contributes to the stability of the interaction (e.g. if k has a small value in (2)).

(2) Coexistence is also more likely if species Q has the higher searching efficiency (figure 12*a*). If the efficiency is not high enough, Q will simply fail to become established (figure 12*b*); if too efficient, however, Q will cause the replacement of P (figure 12*c*). An apparent example

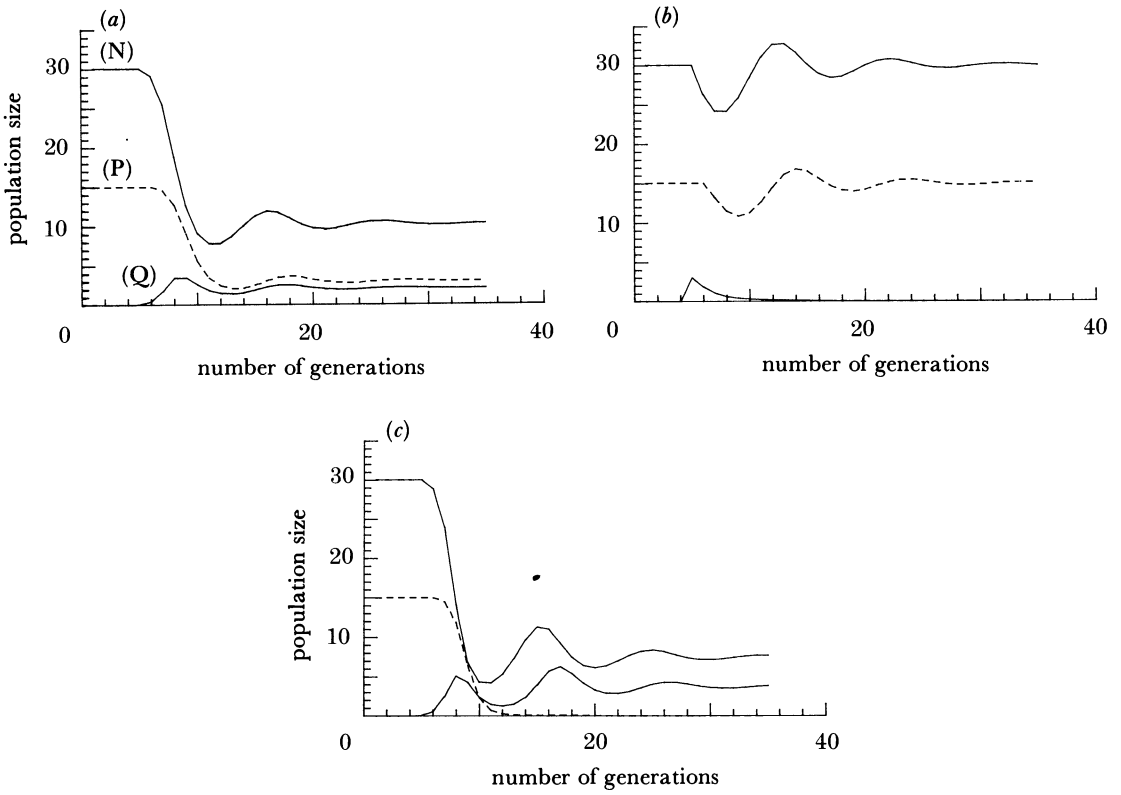

FIGURE 12. The effect of multiple parasitoid introductions from the model in equations (13) or (14), but with $g = 1$ and $R = 0$. Three possible outcomes are shown, after the introduction in generation 5 of a second parasitoid species, Q, where a stable host–parasitoid equilibrium already exists ($N^* = 30$, $P^* = 15$). (*a*) P and Q coexist; $a_P = 0.25$, $a_Q = 0.35$, $k_P = k_Q = 0.25$, $F = 2$. (*b*) Q fails to become established; $a_P = 0.1$, $a_Q = 0.05$, $k_P = k_Q = 0.5$, $F = 2$. (*c*) Q replaces P; $a_P = 0.1$, $a_Q = 0.4$, $k_P = k_Q = 0.5$, $F = 2$. (After Hassell (1978).)

of such 'competitive replacement' comes from the successive introductions of *Opius* parasitoids to control the fruitfly *Dacus dorsalis* in Hawaii (figure 13). If both species P and Q attack the same host stage rather than act in sequence, these criteria imply that coexistence is favoured if the intrinsically inferior species (Q) is also extrinsically superior to P in having the greater searching efficiency. A good natural example of this situation comes from the study of Zwolfer (1979) concerning the coexistence of two species of eurytomid parasitoids, *Eurytoma serratulae* and *E. robusta*, attacking a common host species, the knapweed gall fly, *Urophora cardui*, on creeping thistle. *E. serratulae* is the extrinsically superior species, being the more efficient searcher and having a preference for larger thistleheads containing more hosts. On the other

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hand, *E. robusta* is intrinsically superior in that its larvae will often kill those of *E. serratulae* if both occur within the same host individual. A model of the system predicts coexistence of both species due to just this balance between adult and larval competitive abilities.

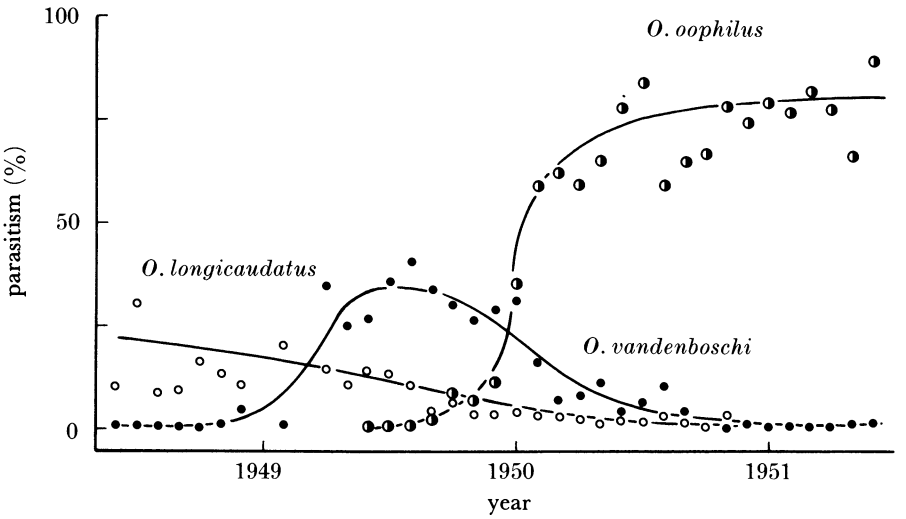

FIGURE 13. Changes in the percentage parasitism of the fruitfly *Dacus dorsalis* by three species of *Opius* parasitoids. Note that each successive parasitoid species causes a higher maximum level of parasitism. (Data from Bess *et al.* (1961), after Varley *et al.* (1973).)

(3) In general, the successful establishment of a second parasitoid species (Q) will further reduce the host equilibrium (figure 14). Not always, however, will this depression be greater than could have been achieved by Q alone, in the absence of P. In particular, if Q tends to parasitize hosts at random (k much greater than 1) rather than showing marked clumping of its attacks (k less than 1), the host depression with both P and Q present will be less than that with Q alone. This effect, however, is slight (dotted line in figure 14) and, given the difficulties in precisely estimating these parameters before introduction in a biological control programme, the analysis points to multiple introductions as a sound biological control strategy.

More recently, Kakehashi *et al.* (1984) have pointed out that the use of the negative binomial for the distribution of parasitism in these models represents a special case of each species causing quite independent contagious distributions of parasitism within the total host population. They then consider the other extreme where both parasitoid species respond in exactly the same way to host cues such as odour, density, etc., making a negative polynomial model with a common index of contagion, k, more appropriate. This difference has little effect on the stability of the models, but does change the equilibrium properties. In particular, it bears on the multiple introduction controversy argument because a single introduction of the superior searcher is now clearly the better strategy for maximizing the depression in host abundance. In the real world such complete covariance in the distributions of parasitism is probably less likely than more or less independent distributions (Hassell & Waage 1984), but in any event this is a cautionary example where general, strategic predictions can be affected by changes in the detailed model assumptions.

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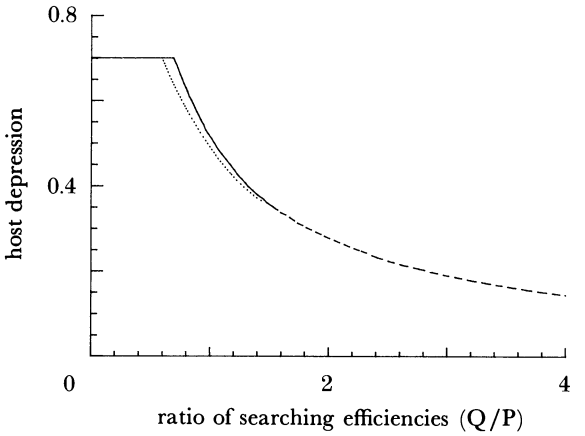

FIGURE 14. The depression in the host equilibrium, relative to the parasitoid-free carrying capacity, from the model in equation (13) with $R = 0$ and $g = \exp(-dNt)$. At very low searching efficiences of Q relative to that of P, Q cannot invade and there is a stable interaction with only the host and P present (*NP*). As the searching efficiency of Q increases, Q invades and the host equilibrium is further reduced (*NPQ*). Finally, at higher searching efficiencies, Q replaces P to give even lower host populations (*NQ*) (which for the main part, however, are locally unstable). Dotted lines show the additional depression if only the host and Q were present. Parameter values; $d/(a_P \ln F = 0.4$, $F = 2$, $k_P = 0.25$, $k_Q = 3$. (After May & Hassell (1981).)

(*b*) *Generalists and specialists*

Introductions in biological control will often be made where the pest is already significantly affected by one or more generalist natural enemies, be they polyphagous parasitoids or generalist predators such as carabids, staphylinids or small mammals. Strategies for introduction should thus be based on a fundamental understanding of

(1) the general conditions necessary for a specialist to 'invade' an existing host–generalist interaction, and

(2) once established, how the specialist interacts with the generalist to affect the dynamics of the host population, and how this is altered if it acts before or after the generalist in the host's life cycle.

We commence by assuming that the generalists have a type II functional response based on a negative binomial distribution of encounters with the hosts (cf. equation (2)), and a simple numerical response given by

$$Gt = h\{1 - \exp(-N_t/b)\}. \tag{17}$$

Here, h is the saturation number of predators and b determines the typical prey density at which this maximum is approached (Southwood & Comins 1976) (figure 15). Such a numerical response might arise from reproduction on a much shorter time scale than that of the hosts or, more likely, from a behavioural response involving 'switching' from feeding elsewhere (Royama 1970) or on other prey species (Murdoch 1969). Combining these functional and numerical responses gives a density-dependent mortality over at least a range of host densities (figure 16), defined by the model

$$N_{t+1} = FN_t g(N_t), \tag{18}$$

where

$$g = [1 + a'G_t/\{k'(1 + a'T'_h N_t)\}]^{-k'}. \tag{19}$$

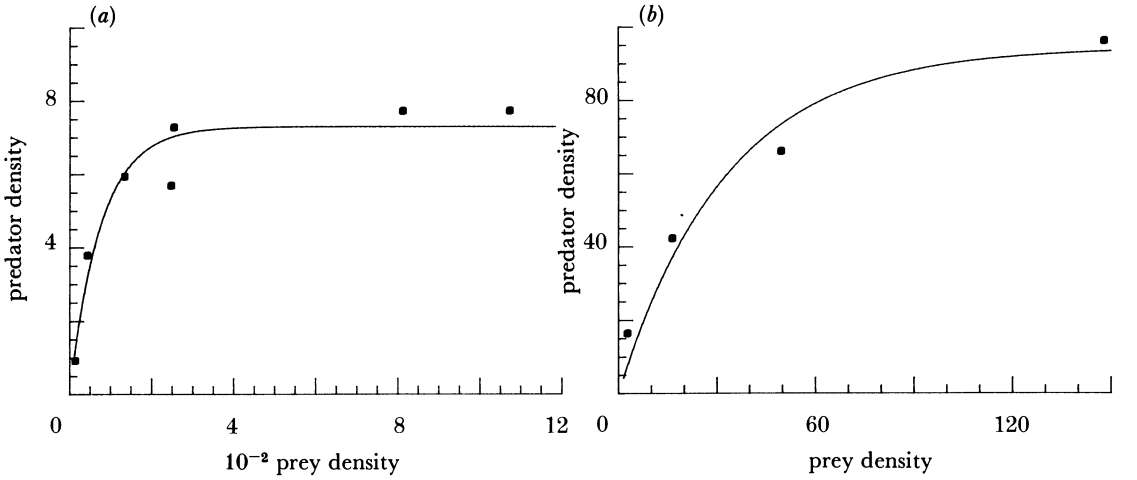

FIGURE 15. Numerical responses of two generalist predators described by equation (17), fitted by nonlinear least squares. (*a*) *Peromyscus maniculatus* in relation to the density of larch sawfly (*Neodiprion setifer*) cocoons (per thousand acres) (1 acre = 4046.8564 m^2) (data from Holling 1959*a*). (*b*) The bay-breasted warbler (*Dendroica fusca*) (nesting pairs per 100 acres) in relation to larvae of the spruce budworm (*Choristoneura fumiferana*) numbers per 10 square feet (1 foot = 0.3048 m) of foliage (data from Mook 1963). Estimated parameter values (±95% confidence limits); (*a*) $h = 7.30 \pm 1.07$, $b = 76.87 \pm 49.16$. (*b*) $h = 94.32 \pm 42.21$, $b = 32.97 \pm 42.27$. (After Hassell & May (1986).)

Here a', T_h' and k' are the parameters of the generalists' type II functional response, as in (2). Locally stable equilibria can occur (figure 17) owing to the density dependence in equation (20*a*), and are made more likely by small handling times, high searching efficiency, a 'strong' numerical response (large h, small b) and low net host rates of increase. The same parameters also maximize the degree of depression in the host equilibrium.

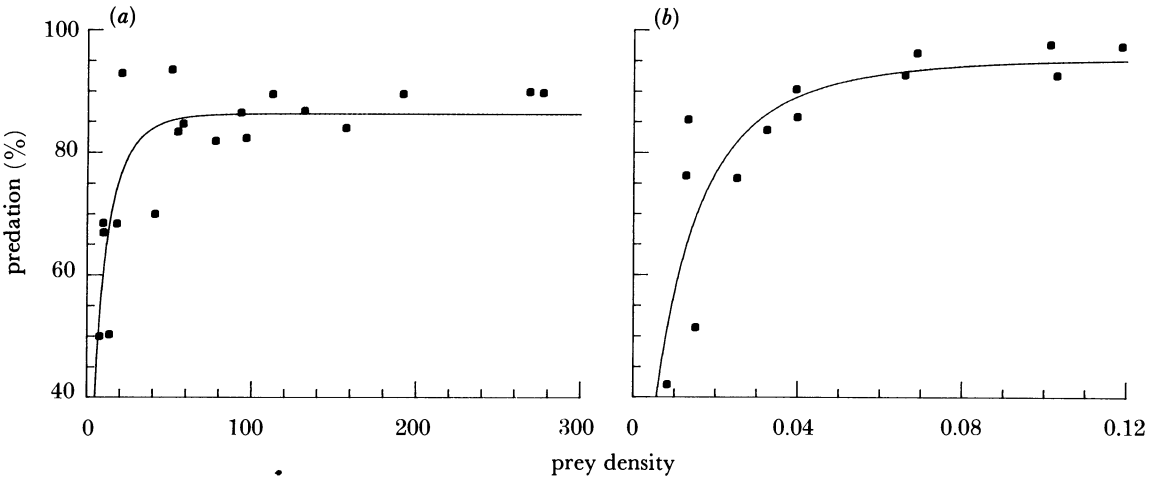

FIGURE 16. Examples of density-dependent predation of soil-pupating Lepidoptera, ascribed primarily to predation by carabid and staphylinid beetles. Data described by equations (18) and (19), fitted by nonlinear least squares. (*a*) Mortality of winter moth pupae per square metre; $a'h = 1.99 \pm 0.18$ (s.e.), $b = 15.94 \pm 3.17$, $k \to \infty$ (data from Varley *et al.* 1973). (*b*) Mortality of *Pardia tripunctatana* pupae per 0.18 m^2; $a'h = 3.08 \pm 1.0$, $b = 0.03 \pm 0.01$, $k \to \infty$ (data from Bauer (1985)). (After Hassell & May (1986).)

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We now add to this picture by the introduction of a specialist parasitoid (P) as part of a biological control programme:

$$N_{t+1} = FN_t f(P_t)\, g(N_t f), \tag{20a}$$

and

$$P_{t+1} = cN_t\{1-f(P_t)\}. \tag{20b}$$

Here, the function f is again given by (2). As discussed above, with more than one mortality acting, we need to be explicit on their relative timing in the host's life cycle. Equations (20a, b) are for the case of generalists acting after the specialist; if they act first the model becomes

$$N_{t+1} = FN_t f(P_t)\, g(N_t), \tag{21a}$$

and

$$P_{t+1} = cN_t g(N_t)\,[1-f(P_t)]. \tag{21b}$$

The following conclusions emerge from an analysis of these models (see Hassell & May (1986) for further details).

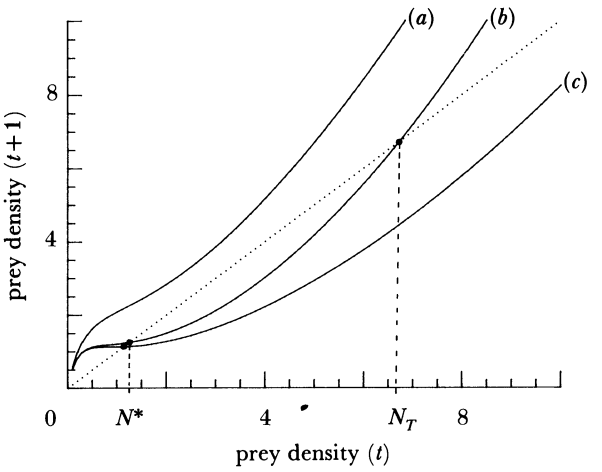

FIGURE 17. Map of prey densities in successive generations, t and $t+1$, from equations (18) and (19). A locally stable equilibrium occurs at N^* and an unstable, 'release' point at N_T. (*a*) $a'h = 3$, $a'bT_h' = 0.04$. (*b*) $a'h = 2$, $a'bT_h' = 0.04$. (*c*) $a'h = 3$, $a'bT_h' = 0.08$. $F = 7$ and $k \to \infty$, throughout. (After Hassell & May (1986).)

(1) The introduced parasitoid, P, can invade and coexist more easily if it acts before the generalist in the host's life cycle (figure 18*a*). This is simply due to the larger pool of hosts available. Conversely, if the host rate of increase, F is too low, or if the generalist's overall efficiency, $a'h$, is too high relative to P, the specialist will be unable to invade and a persistent three-species interaction is impossible. This is broadly the same conclusion as reached by Holt (1977). Specialist egg-parasitoids may thus 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.

(2) Once again, if the specialist acts before generalists, and if the generalists cause strong over-compensating density dependence, there is the risk of higher host populations than existed before the introduction with only the generalist acting on its own (figure 18*b*).

(3) After successful establishment, there can either be only one equilibrium point (or stable cycle), or there may be more complex situations with a variety of possible alternative stable

states. Thus there may be two alternative persistent states, one with only the generalist and the other with all three species present, or two alternative three-species states in which the interaction may 'flip' between high and low levels if sufficiently perturbed. These possibilities are illustrated in figure 19.

(4) A stable system with all three species can exist when one or both of the two-species interactions alone would be unstable.

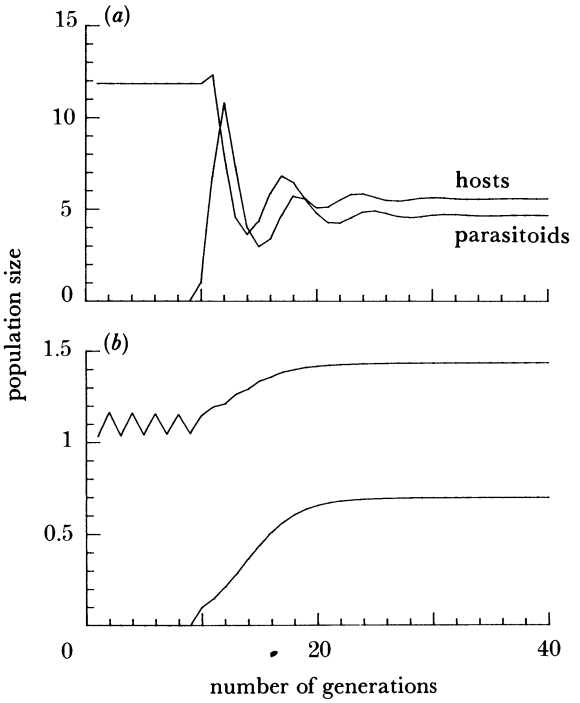

FIGURE 18. Numerical examples from the model in equation (20), where the specialist parasitoids 'invade' the host–generalist interaction in generation 10. Parameter values; $F = 8$, $T_h' = T_h = 0$, $h = 20$, $b = 10$, $k' \to \infty$, $k = 0.8$, $a = 0.8$, and (*a*) $a' = 0.15$ and (*b*) $a' = 1$.

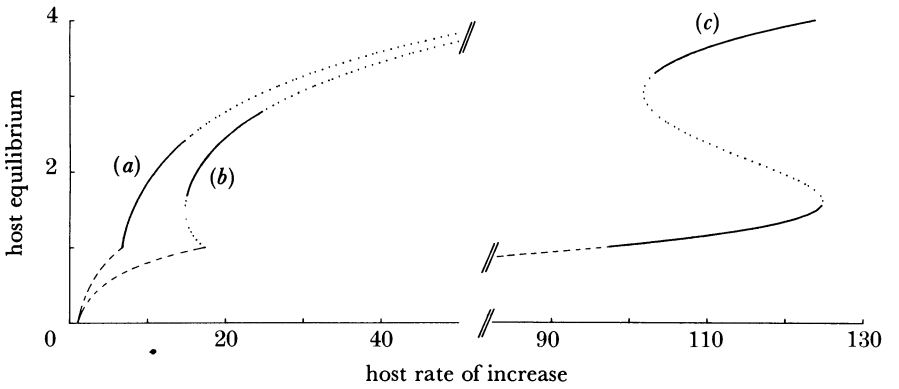

FIGURE 19. Three examples of the dependence of the host equilibrium, N^*, on the host rate of increase, F, for the model in equation (20). (*a*): $h = 20$, $b = 10$; (*b*): $h = 30$, $b = 10$; (*c*): $h = 5$, $b = 0.4$, $a' = a = 1$ and $k' = k \to \infty$ throughout. The broken lines indicate host–generalist interactions, the solid lines indicate locally stable host–generalist–specialist interactions, and the dotted lines indicate locally unstable ones.

In short, unlike the separate pairwise interactions each of which have rather straightforward dynamics, the combined three-species system presents a wider, and in some respects unexpected, range of properties. Not only are such studies of fundamental interest in showing how population dynamics can affect community structure, but also they caution us against formulating biological control strategies purely in terms of two-species interactions.

(*c*) *Pathogens and parasitoids*

Various models for the combined action of a pathogen and a parasitoid or predator upon a target host population have been considered by Carpenter (1981) and Anderson & May (1986).

In general, all such systems may be viewed as specialized studies in two-species competition, in which the pathogen and the parasitoid compete for a common resource (the host species). It is therefore not surprising that basically four situations can arise, depending on the parameters characterizing the interactions (searching efficiency, aggregation, etc. for the parasitoid; virulence, transmissibility, etc. for the pathogen): parasitoid and pathogen may coexist; either parasitoid or pathogen species may regulate the host population to densities below the threshold for maintenance of the other species, thus 'competitively excluding' it; or there may be two alternative stable states, one with parasitoid only and the other with pathogen only, with the initial conditions determining which state is attained in any particular instance. Moreover, given the oscillatory propensities of the individual components of these systems, any one of the above states may be a steady equilibrium, or a stable cycle, or even (in some models) chaotic fluctuations.

It is pedagogically interesting to consider the very simplest such model, for the case of a host population with discrete, non-overlapping generations (R. M. May & M. P. Hassell, unpublished results). Combining the models in (1) and (6), we suppose the adult population in generation t, N_t, is first attacked by a lethal pathogen spread by direct contact, and then the survivors are attacked by randomly searching parasitoids:

$$N_{t+1} = FN_t S(N_t) f(P_t), \tag{22a}$$

and

$$P_{t+1} = cN_t S(N_t)\{1-f(P_t)\}. \tag{22b}$$

Here $S(N)$ is the fraction surviving the epidemic, given earlier (see (6)) by the implicit relation $S = \exp[-(1-S)N_t/N_T]$, and f has the Nicholson–Bailey form $f(P) = \exp(-aP)$. For $acN_T(\ln F)/(F-1)$ less than 1, the pathogen excludes the parasitoid (essentially, by maintaining the host population at levels too low to sustain the parasitoid, with its relatively low searching efficiency, (a). For values of acN_T significantly in excess of $(F\ln F)/(F-1)$, a linear analysis might suggest the parasitoid would exclude the pathogen for similar reasons. But a pure Nicholson–Bailey system gives diverging oscillations, and the pathogen can always invade as the hosts cycle to high values. A typical outcome is shown in figure 20, where the 'noisy' curves derive from the simple and purely deterministic equation (22). Figure 20 has a moral: the basic period of the oscillations in host abundance is determined primarily as the Nicholson–Bailey period for parasitoid dynamics; but the stable – if somewhat ragged (chaotic) – oscillations, rather than diverging Nicholson–Bailey cycles, derives from the pathogen. It is thus meaningless to ask whether the dynamics of this system are determined mainly by the parasitoid or by the pathogen. Both are important in different ways. The

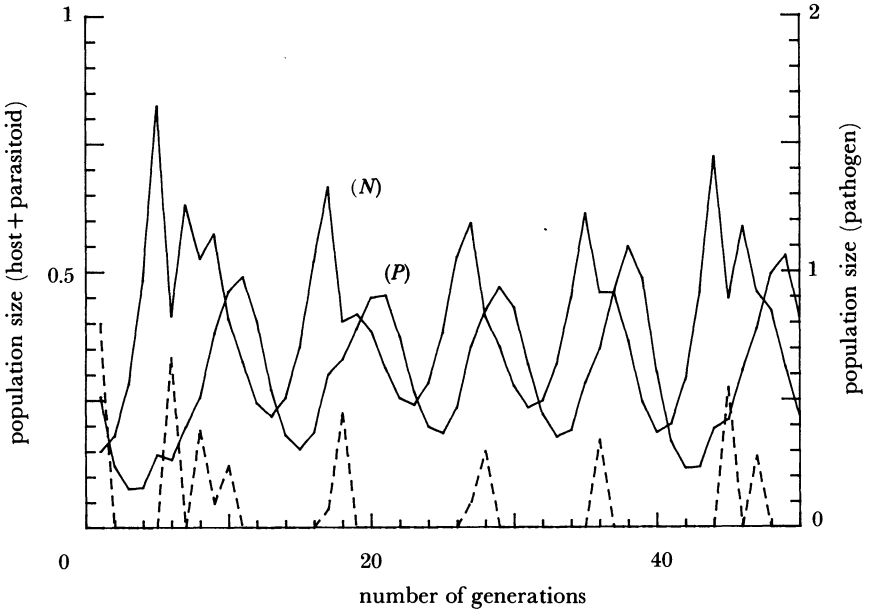

FIGURE 20. A numerical example from the model in equation (22), where $a = F = 2$ (for further details see text). Pathogen populations drawn to half scale.

parasitoid sets average host abundances and oscillatory periods, but the pathogen provides long-term stability, preventing overshoot or crash.

COMPLEX LIFE CYCLES

For parasitoids, our discussion so far has focused on non-linear difference equations as a means of representing coupled, synchronized interactions with discrete generations. These correspond well to many temperate zone situations, where seasonality often synchronizes the populations and provides a natural interval between the appearance of successive generations. Such models, however, would not seem appropriate to the many host–parasitoid interactions in more tropical climes, where the life cycles of hosts and parasitoids may be of quite different lengths and where one would expect continuous generations with all stages present at the same time. These call for models in continuous time, with the age structure of the populations being explicitly included. A classic study of this kind, using host–parasitoid models framed as delay-differential equations, is that of Auslander *et al.* (1974). Interestingly, they found that discrete generation 'waves' could easily occur in such interactions with suitable parameter combinations.

Recently, Godfray & Hassell (1987) have also demonstrated how cycles whose period is approximately equal to the duration of one host generation period can unexpectedly arise, in this case depending in large measure on the ratio of the lengths of the host and the parasitoid life cycles. They developed a simulation model with a host population having four stages – pre-parasitism (e.g. eggs), susceptible (e.g. larvae), post-parasitism (e.g. pupae) and adults – and with the adults having a constant fecundity per unit time. The parasitoids have a life cycle divided into only two stages: the adults that search for and parasitize hosts, and the immature stages developing on or in parasitized hosts. All the immature stages of host and parasitoid have distributed developmental periods, drawn from an inverse quadratic distribution with specified

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mean and variance (Sharpe *et al.* 1977). The adults have an exponential survivorship curve. Finally, the rate of parasitism is given again by equation (2). (Note that the model collapses to (1) if the total host and parasitoid life cycles are of the same length, the adult hosts and parasitoids live for only one time unit and if there is no variance in the duration of the immature stages.)

Numerical simulations of this model suggest two distinct types of population behaviour at equilibrium:

(1) constant population sizes with all host and parasitoid age classes present (continuous generations) (figure 21*a*), and

(2) stable cycles of host and parasitoid populations with periods of both being approximately equal to the average duration of one host generation ('discrete' generations) (figure 21*b*).

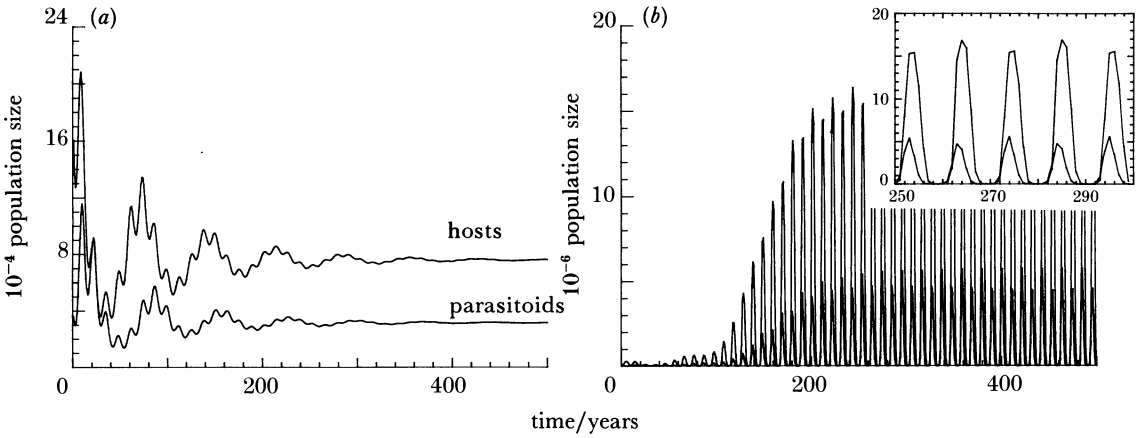

FIGURE 21. Simulations from the age-structured model described in the text. (*a*) Complete overlap of stages at equilibrium; host and parasitoid life cycles of equal length. (*b*) Stable cycles of host and parasitoids with periods of both approximately equal to the duration of one host generation (see inset); parasitoid life cycle half as long as that of the host. Other parameters the same for both examples. (From Godfray & Hassell (1987), in which further details are given.)

The two most important parameters affecting which of these behaviours occurs are (*a*) the degree of aggregation in the distribution of parasitism (k) (very small values of k promote continuous generations), and (*b*) the ratio of the host and parasitoid life-cycle lengths. It is particularly interesting that discrete generations are most likely when the parasitoid's life cycle is about 0.5 or 1.5 times that of the hosts. Conversely, parasitoid life cycles that are very short, or about the same length as the host's, or about twice as long, promote continuous generations. In a recent theoretical study of a broadly related problem, MacDonald (1986) has shown that delays in regulatory mechanisms affecting prey or predator populations separately, usually cause oscillatory dynamics, but that a stable equilibrium can ensue when such delays are present for both populations and are of approximate equal duration (as is the case in the above models when host and parasitoid life cycles are of similar length).

Some support for these theoretical suggestions comes from examples of time series of laboratory and natural host–parasitoid systems. Interestingly, a tendency for discrete generations in these systems generally occurs when the ratio of the lengths of host and parasitoid life cycles is approximately two to one (figure 22). Such unequal generation times appear to be typical of many lepidopteran host–parasitoid systems in the tropics.

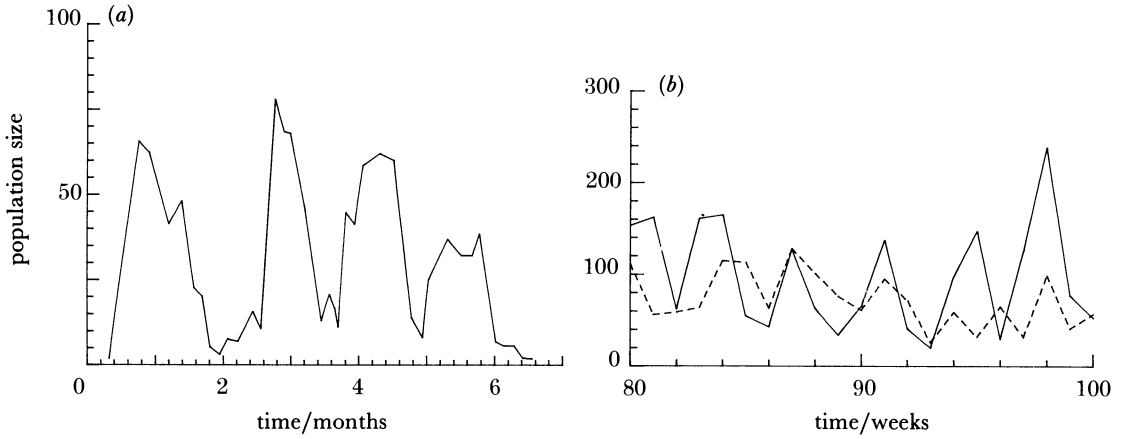

FIGURE 22. Examples of more or less discrete generations in insect populations. (*a*) Part of a time series of emergences of the adult coffee leaf miner *Leucoptera caffeina* from field samples of pupae (after Notley 1956). (*b*) A segment of the interaction in figure 4, taken from weeks 80–100, showing a tendency for parasitoid population cycles of one host generation period.

The relative generation times of hosts and parasitoids have been largely neglected in appraising parasitoids for release. Yet they are attributes that are easy to measure under natural conditions and they can have important implications for biological control. In particular, lower average densities of hosts are obtained from continuous interactions than from comparable ones with discrete generations. On the other hand, there could be good reasons for sacrificing this gain and preferring discrete generations if, for example, synchronized host and parasitoid populations are needed for the optimal timing of supplementary chemical control. Some problems of integrating chemical and biological methods of control are considered in the next section.

PESTICIDES AND NATURAL ENEMIES

Modern pest management aims at integrating a variety of chemical, biological and cultural methods of pest control. A recurrent problem in this, however, has been the antagonistic effects of biological and chemical control methods, because pesticides in general have an adverse effect on natural enemies, often to a greater degree than on the target pest population. In some cases this has even led to 'resurgence' of the pest population to levels higher than occurred before the application of the pesticide (DeBach 1974).

(*a*) *Pesticides and parasitoids*

In this subsection we explore the effects of a regular application of insecticide to the host–parasitoid system described by equation (1) (with $g(fN_t)$ equal to 1), focusing in particular on the timing of insecticide application relative to parasitism. A much fuller treatment, with details of the model, is given in Hassell (1984) and Waage *et al.* (1985). Some comparable models in continuous time are discussed by Barclay & van den Driessche (1977) and Barclay (1982).

There are four obvious possibilities for the relative timing of insecticides and parasitism:

(1) insecticides act after parasitism and only kill hosts;

(2) insecticides act before parasitism and only kill hosts;

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(3) insecticides act after parasitism and also kill parasitized hosts, or

(4) insecticides act before parasitism and also kill adult parasitoids.

The effects of these different strategies can be viewed as the depression (q) of the host equilibrium caused by the insecticides:

$$q = N^*_{(+I)}/N^*_{(-I)}. \tag{23}$$

Here $N^*_{(+I)}$ and $N^*_{(-I)}$ are the equilibrium host populations with and without insecticides, respectively. Values of q less than 1 thus indicate a net benefit from insecticide application (figure 23*a*), whereas q greater than 1 indicates a perverse increase in host abundance compared with the insecticide-free level (figure 23*b*).

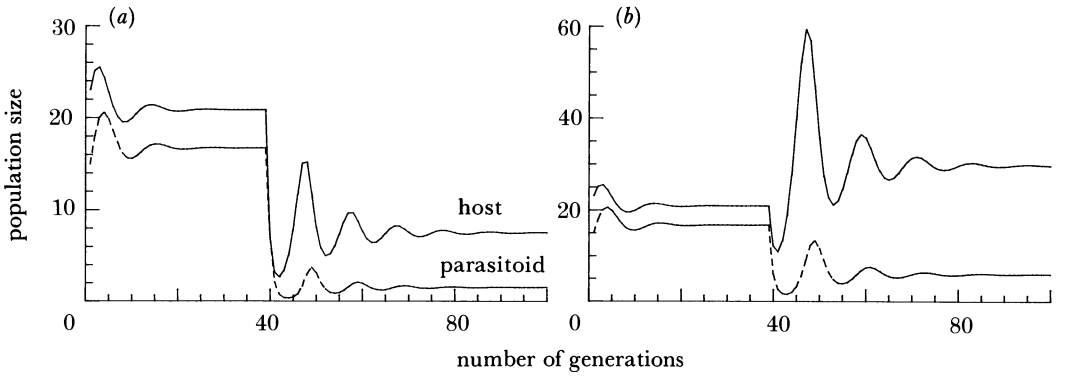

FIGURE 23. Examples of supression and 'resurgence' from the model in equations (1) and (2) with the addition of insecticides in generation 40 onwards, affecting both hosts and adult parasitoids (I_h and I_p are the fraction of hosts and parasitoids surviving insecticides, respectively). (*a*) Insecticides compliment the parasitoids in reducing host populations; $I_h = 0.4$, $I_p = 1$. (*b*) Insecticides also affecting the parasitoids and causing resurgence; $I_h = 0.4$, $I_p = 0.35$. Other parameters the same for both examples; $a = 1$, $T_h = 0.05$, $k = 0.6$, $F = 5$. (See Waage *et al.* (1985) for further details.)

An obvious conclusion from the models is that insecticides that have no effect on the parasitoids always contribute to reduced host population levels (q less than 1). However, if the insecticides also kill adult or immature parasitoids this depression is reduced to the point that pest resurgence may occur (q greater than 1). This problem is accentuated by adult parasitoids often being more susceptible to insecticides than the host stage which they attack (Abdelrahman 1973; Croft & Brown 1975; Croft 1977), and this only increases the likelihood of pest resurgence.

(*b*) *Pesticides and pathogens*

Carpenter (1981) has documented the fact that 'disease is implicated in the natural regulation of invasions of the aquatic nuisance *Myriophyllum spicatum* (L.), yet harvested and herbicide-treated stands sometimes persist long after decimation of neighbouring stands'. He has gone on to explore the combined effects of pathogens and control measures (such as pesticides or harvesting) upon the dynamics of the target pest population, by using the basic framework developed above for the host–pathogen interactions.

Carpenter (1981) concludes that pesticides, or other interventions that increase the pest death rate can, on the one hand, permit a less virulent pathogen to control the population (the criterion $\alpha > r$ is easier to meet if r is reduced by an elevated death rate). On the other hand, such pesticides will usually increase the threshold pest population necessary for the pathogen

to spread, and also will lengthen the time required for the pathogen to reduce pest population levels. In contrast, Carpenter (1981) notes that supplementary control measures that decrease the pest birth rate (such as release of sterile males) do not usually exhibit such antagonistic interactions with control by pathogens.

Population dynamics and the evolution of pesticide resistance

Up to this point, we have dealt exclusively with dynamical aspects of the interactions between pests and the pathogens, parasitoids, and/or pesticides used in control programmes. In reality, the introduction of such agents of control inevitably alters the selective forces acting on the genetic variation found in natural populations, so that resistance to pesticides and pathogens, or behavioural changes that reduce predation levels, are likely to evolve. Thus, for example, Levin & Pimentel (1981) and May & Anderson (1983) have discussed the influence of population genetics and population dynamics upon the coevolution of host–pathogen systems; the latter authors give a fairly detailed discussion of the way the predominant genotype of myxoma virus has changed in Australia since it was first introduced to control rabbit populations in the early 1950s. In the final analysis, all attempts at pest control are aimed at a moving target.

Although there has been much work on biochemical and genetic aspects of pesticides resistance (see, for example, National Academy of Sciences (N.A.S.) (1986)), there has until recently been little work on the way population genetics and nonlinear ('density dependent') aspects of population dynamics combine to affect the rate at which pesticide resistance evolves. Comins (1977*a*, *b*, 1984) has studied some of these problems in the absence of natural enemies, and Tabashnik & Croft (1982) and Tabashnik (1986) have made numerical studies of the characteristically different rates at which pesticide resistance evolves among orchard pests and their natural enemies. May & Dobson (1986) have recently reviewed this subject, and outlined a more general analysis of the evolution of pesticide resistance in interacting systems of pests and natural enemies. What follows is a brief summary of their work.

It is helpful to begin by considering the population genetics of pesticide resistance for a pest species in a region subject to repeated application of the pesticide, but where immigration from untreated regions occurs in each generation. We consider resistance to involve a single diallelic locus (as is indeed sometimes the case), with resistant, heterozygote, and susceptible genotypes denoted by *RR*, *RS*, and *SS*, respectively. Under pesticide application, the three genotypes are assumed to have relative fitnesses $1:1-hs:1-s$, respectively. Here s represents the 'selection strength' of the pesticide ($1>s>0$), and h measures the degree to which the resistance allele, R, is dominant (for R recessive, $h=1$; for R dominant, $h=0$). Suppose that, in each generation, a fraction m of the total gene pool in the treated area derives from susceptible, *SS*, immigrants, with the remaining fraction $(1-m)$ deriving from the previous generation in the treated region. The gene frequency of R in generation $t+1$, p_{t+1}, is then given by a standard calculation as

$$p_{t+1}=\frac{(1-m)\{p_t^2+p_tq_t(1-hs)\}}{(1-m)\{p_t^2+2p_tq_t(1-hs)+q_t^2(1-s)\}+m}. \qquad (24)$$

Here, as usual, q is the gene frequency of S; $q_t=1-p_t$. Equilibrium solutions of (24) are obtained by putting p_{t+1} equal to p_t equal to p, and solving the resulting algebraic equation.

One solution is always $p = 0$. Other solutions (with 1 less than q less than 0) are given by the quadratic equation

$$(1-2h)\,q^2+hq-\varphi = 0. \tag{25}$$

Here φ is a parameter characterizing the relative strengths of migration and selection, $\varphi \equiv m/\{s(1-m)\}$. For h greater than $\frac{2}{3}$, (25) can have two distinct solutions, providing φ is small enough (specifically, $\varphi < h^2/\{4(2h-1)\}$); for $h < \frac{2}{3}$, (25) always has at most one sensible solution with 1 less than or equal to q less than or equal to 0.

This simple model gives an illuminating (and surprisingly unfamiliar) metaphor for what can happen when selection is opposed by gene flow. The possibilities are illustrated in figure 24, which shows equilibrium values of the gene frequency of the resistance allele, R, as a function of the migration-selection parameter, $\varphi \equiv m/\{s(1-m)\}$, for various values of h. If R is not too dominant ($h > \frac{2}{3}$), there are two alternative stable states when migration is neither too large nor too small in relation to the selection strength. The possibility of alternative stable states disappears if resistance is sufficiently dominant ($h < \frac{2}{3}$), in which case the resistance allele always predominates at low enough migration levels. As emphasized by Comins (1977*b*), in a somewhat more complex treatment, the time taken for resistance to appear can be significantly prolonged by deliberate efforts to keep immigration from untreated regions relatively high, particularly if resistance is a recessive character.

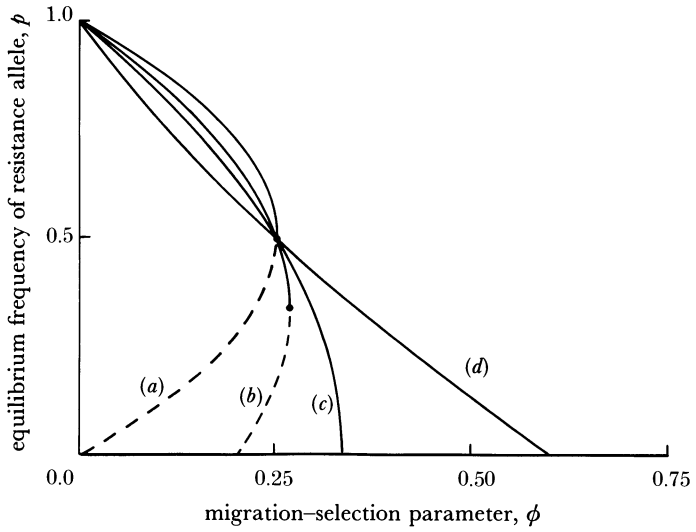

FIGURE 24. The equilibrium frequency, p, of the resistant allele, R, plotted as a function of the parameter characterizing the relative strengths of migration and selection, $\varphi = m/\{s(1-m)\}$, for the simple model defined by equation (24). The curves labelled (*a*), (*b*), (*c*), (*d*) are for h (the dominance parameter) equal to 1.00, 0.80, 0.67, 0.40, respectively. The curves labelled (*a*) and (*b*) can exhibit two alternative stable states (p large and p equal to 0), divided by an unstable state (dashed curves); for sufficiently large φ, p equal to 0 is the only stable state.

These remarks about the evolution of resistance in the presence of gene flow assume new significance when they are combined with consideration of the population dynamics of the pest. By definition, pests with undercompensating density dependence tend to recover steadily and monotonically to their original densities after a disturbance (such as application of a pesticide) that reduces population density. Conversely, pest populations with overcompensating density

dependence tend to manifest a perverse response to pesticide application: if driven to a low level by pesticide application, such a population tends in the next generation to bounce back to a higher level than would otherwise have been the case. It follows that, for pests with undercompensating density dependence, the population densities of the next generation on average will be lower in treated regions that in untreated ones, making the effects of migration from untreated regions relatively more significant. Conversely, for pests with overcompensating density dependence, the next generation on average will be at higher densities in treated regions than in untreated ones, whence migration is relatively less significant. But we have just seen that resistance tends to evolve faster when migration is relatively unimportant, which prompts the qualitative conclusion that undercompensating density dependence tends to retard the evolution of pesticide resistance, and overcompensating density dependence to accelerate it. These ideas are developed more fully by May & Dobson (1986), who note, *inter alia*, that a pest exhibiting a most pronounced degree of overcompensation in field studies is the colorado potato beetle (*Leptinotarsa decemlineata*), which (perhaps coincidentally) is notorious for the speed with which it has developed resistance to a wide range of pesticides.

The propensity for pest species to evolve resistance more quickly than their natural enemies do has often been noted (Tabashnik 1986; Roush & Croft 1986). One reason might be that the coevolution between plants and phytophagous insects has preadapted the latter to the evolution of detoxifying mechanisms, whereas this is much less the case for the natural enemies of such insects. Laboratory studies do not, however, indicate general patterns of this kind, and it seems likely that under controlled conditions the rate of evolution of pesticide resistance in prey and in predator populations depends on the detailed molecular mechanisms underlying detoxification (Mullin *et al.* 1982). This has prompted a search for pesticides that are less lethal for natural enemies than for pests (Roush & Plapp 1982), or even the release of natural enemies that have been artificially selected for resistance to specific pesticides (Roush & Hoy 1981).

An alternative explanation for the typically swifter evolution of resistance by pests than by their natural enemies lies in the population dynamics of prey–predator associations. Suppose a pesticide kills a large fraction of all prey and all predators in the treated region. For the surviving prey, life is now relatively good (relatively free from predators), and the population is likely to increase rapidly. Conversely, for the surviving predators life is relatively bad (food is harder to find), and their population will tend to recover slowly. This argument can be elaborated by a standard phase plane analysis of Lotka–Volterra or other, more realistic, prey–predator or host–parasitoid models. Such analysis shows that, in the aftermath of application of a pesticide affecting both prey and predator, prey populations will tend to exhibit overcompensating density-dependent effects, whereas predator populations will tend to manifest undercompensation. From the arguments developed earlier in this section, we would expect that, for a given level of migration and pesticide application, pest species (which effectively have overcompensating density dependence) will tend to develop resistance faster than will their natural enemies (which effectively have undercompensating density dependence).

In short, both population genetics and population dynamics must be considered in projecting the long-term consequences of control programmes, particularly if natural enemies are combined with pesticides or other 'artificial' control methods.

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Conclusion

Fundamental studies of host–parasitoid and host–pathogen interactions reveal several factors affecting the dynamics both of natural systems, and of manipulated ones arising from biological control. Simple mathematical models can provide a potent tool for gaining rapid insights into population behaviour: insights that would otherwise only accrue gradually, and piecemeal, as examples slowly accumulate where natural and biological control 'case histories' have been analysed in some detail. Unfortunately, almost none of the 300 or so 'successes' in biological control have been studied in the field beyond the relatively short period when natural enemies are actually being released, and then only in a rather superficial way. This is a great opportunity being wasted. Biological control programmes are, in effect, manipulation experiments on a grand scale involving relatively few species. If properly monitored, they would provide invaluable information, for example, on population regulation, on the dynamics of interactions involving predators, parasitoids and pathogens, and on species invasions; ecology and biological control would both benefit greatly.

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Appendix 1

This appendix derives the dynamical properties of equation (12), as summarized in the text.

Possible equilibrium values of the host and parasitoid populations are obtained by putting $N_{t+1} = N_t = N^*$ and $P_{t+1} = P_t = P^*$, respectively. For g equal to 1, (12a) gives P^* from

$$Ff(P^*) = 1. \tag{A 1}$$

Equation (A 1) says that the equilibrium parasitoid density is equal to that obtained in the absence of any inundative release (i.e. for R equal to 0). If $f(P)$ has the form of equation (2), with T_h equal to 0, (A 1) gives

$$P^* = (k/a)\,(F^{\frac{1}{k}}-1). \tag{A 2}$$

Equation (12b) gives simply

$$N^* = (P^*-R)/(1-1/F). \tag{A 3}$$

Clearly, no equilibrium is possible, and the host population will be eradicated, if sustained release of parasitoids is made at a rate R greater than P^*.

For R less than P^*, the linearized stability properties of the system may be obtained in the usual way by writing $N_t = N^* + x_t$ and $P_t = P^* + y_t$, neglecting nonlinear terms, and writing $x_t, y_t \sim \Lambda^t$, to obtain for the eigenvalues Λ the quadratic equation

$$\Lambda^2 - (1+\zeta/F)\,\Lambda + \zeta = 0. \tag{A 4}$$

Here ζ is defined in general by

$$\zeta = -FN^*(\mathrm{d}f/\mathrm{d}P)^*. \tag{A 5}$$

The equilibrium state will be locally stable iff both eigenvalues Λ have modulus less than unity, which is to say iff

$$\zeta < 1. \tag{A 6}$$

This criterion (A 6) is, in general, increasingly easy to satisfy as R increases from zero to the critical value P^*.

In particular, if $f(P)$ is given by (2) (with $T_h = 0$), we have

$$\zeta = F^{\frac{-1}{k}}\{k(F^{\frac{1}{k}}-1) - aR\}/\{1-1/F\}. \tag{A 7}$$

That is, the equilibrium state is stable to small disturbances iff

$$aR > F^{\frac{1}{k}}\{k(1-F^{\frac{-1}{k}}) - (1-F^{-1})\}. \tag{A 8}$$

This requirement is always satisfied if $k < 1$, and for $k > 1$ it is increasingly easily satisfied as aR increases. Even for the randomly-searching parasitoids of the original Nicholson–Bailey model (which corresponds to $k \to \infty$ in (A 8)), the familiar diverging oscillations can be replaced by a stable equilibrium if inundative release rates are large enough that $aR > (\ln F - 1 + 1/F)$; the critical rate of release for eradication is yet higher at $aR > \ln F$.

Discussion

P. Neuenschwander (*International Institute of Tropical Agriculture, Ibadan, Nigeria*). Professor Hassell's presentation implies that on the basis of simulation models simple introduction of the most efficient parasitoid is superior over multiple introductions, under conditions where strong density-dependent factors are already at work. Practical experience does not bear this out. Could you tell us what differences in host densities are involved in the different examples. This answer is of considerable importance for the practitioner in the field who has to justify introduction without being able to document efficiency beforehand.

M. P. Hassell. On the contrary, our analytical models suggest that multiple introductions of parasitoids are a sound biological control strategy, even in the presence of additional density dependence affecting the host population. Additional releases will usually lead to further depression of the host population, and where this is not the case, the effect is very slight, so that two parasitoid species perform almost as well as the most efficient on its own. As discussed in our paper, Kakehashi *et al.* (1984) show that there are situations where a single introduction of the most efficient parasitoid is the better strategy for reducing the host population. Their model, however, requires all the parasitoid species to respond in the same behavioural way to the spatial distribution of hosts, which we believe to be unrealistic.

D. J. Rogers (*Department of Zoology, University of Oxford, U.K.*). Professor Hassell's paper mentions an inverse relation between a host's effective rate of increase and the degree of suppression that a parasitoid can achieve (i.e. lower rates of increase are associated with greater degrees of suppression).

Bearing in mind the large amount of suppression recorded by Beddington *et al.* (1978) in cases of successful biological control would Professor Hassell like to comment on the very low rates of increase of the pest species that this result implies, i.e. do pests really have such low rates of increase? If so, why do they become pests?

Could this explain the mediocre success rate of 'classical biological control' and are failures attributable to host species having relatively higher rates of increase?

M. P. Hassell. The examples that I showed of parasitoids reducing host populations, but not to the low levels characteristic of classical biological control successes, come from laboratory interactions where parasitism is the only major mortality suffered by the host population. Insect populations in the field will normally have a net rate of increase very much lower than their *per capita* fecundity due to the many other factors affecting their natality or mortality. Experience has shown that insects with very low net rates of increase can easily become pests if divorced from their usual complement of natural enemies. A pest species with a high net rate of increase will clearly require a more efficient natural enemy to cause a given level of depression. I know of no good evidence indicating that the failures in classical biological control are correlated with high rates of increase.

T. R. E. Southwood, F.R.S. (*Department of Zoology, University of Oxford, U.K.*). In the previous paper Dr Wagge and Dr Greathead indicated how the specificity of potential biological control agents could be tested by a 'centrifugal technique': testing them against other potential hosts

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of decreasing relatedness to the target species. Does Professor Hassell think, in the light of his emphasis on search pattern, that this and other behavioural characters should (or could) also be taken into account in assessing potential control agents before release?

M. P. Hassell. Several behavioural characters of natural enemies that are highly relevant to biological control could well be studied before release, particularly when comparing a number of potential biological control agents. For example, the ability of natural enemies to locate isolated patches of hosts, their maximum attack rates within such patches, whether or not they host feed (therefore affecting longevity), all affect the overall efficiency of a natural enemy and thus its ability to depress the host population. Behavioural characters such as these are all amenable to careful study under controlled conditions as part of evaluating candidates for a biological control programme. They may also provide useful pointers to the specificity of a natural enemy, because highly evolved behavioural adaptations for locating host individuals are much more likely to occur in relatively specific natural enemy species than in polyphagous ones.

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