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Heterogeneity and the dynamics of host–parasitoid interactions

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SUMMARY

This paper is concerned with the dynamical effects of spatial heterogeneity in host–parasitoid interactions with discrete generations. We show that the dynamical effects of any pattern of distribution of searching parasitoids in such systems can be assessed within a common, simple framework. In particular, we describe an approximate general rule that the populations of hosts and parasitoids will be regulated if the coefficient of variation squared (CV^2) of the distribution of searching parasitoids is greater than one. This criterion is shown to apply both generally and in several specific cases. We further show that CV^2 may be partitioned into a density-dependent component (direct or inverse) caused by the response of parasitoids to host density per patch, and a density independent component. Population regulation can be enhanced as much by density independent as by density-dependent heterogeneity. Thus the dynamical effects of any pattern of distribution of searching parasitoids can be assessed within the same common framework. The paradoxical impact of density-independent heterogeneity on dynamics is especially interesting: the greater the density independence, and thus the more scattered the data of percent parasitism against local host density, the more stable the populations are likely to be. Although a detailed analysis of host–parasitoid interactions in continuous time has yet to be done, evidence does not support the suggestion of Murdoch & Oaten (1989) that non-random parasitism may have quite different effects on the dynamics of continuous-time interactions. There appears to be no fundamental difference in the role of heterogeneity in comparable discrete- or continuous-time interactions.

A total of 65 data sets from field studies have been analysed, in which percentage parasitism in relation to local host density have been recorded. In each case, estimated values of CV^2 have been obtained by using a maximum likelihood procedure. The method also allows us to partition the CV^2 into the density dependent and density-independent components mentioned above. In 18 out of the 65 cases, total heterogeneity was at levels sufficient (if typical of the interactions) to stabilize the interacting populations (i.e. $CV^2 > 1$). Interestingly, in 14 of these it is the host-density-independent heterogeneity that contributes most to the total heterogeneity.

Although heterogeneity has often been regarded as a complicating factor in population dynamics that rapidly leads to analytical intractability, this clearly need not necessarily be so. The $CV^2 > 1$ rule explains the consequences of heterogeneity for population dynamics in terms of a simple description of the heterogeneity itself, and provides a rough rule for predicting the effects of different kinds of heterogeneity on population regulation.

INTRODUCTION

A relatively recent and major concern in population ecology has been to determine the effects of heterogeneity on population dynamics. Earlier, theoretical work tended, for convenience of analysis, to assume completely homogeneous populations in which each individual has the same chance of reproducing, and the same risk of dying. The wealth of evidence to the contrary has prompted the development of theoretical frameworks for most types of species interactions that take explicit account of heterogeneity between individuals. The common conclusion, whether one considers competing species (see, for example, Atkinson & Shorrocks (1981); Hanski (1981); Ives & May (1985)), plant–herbivore interactions (see, for example, Crawley (1983)), hosts and pathogens (see, for example, Anderson & May (1984) or predators and prey (Comins & Hassell (1987)), is that heterogeneity promotes the persistence of the interacting populations. This paper considers the effects of heterogeneity on the dynamics of one particular kind of predator–prey system, that of insect parasitoids and their hosts.

Within the broad class of predatory metazoans, the parasitoid lifestyle predominates; one recent estimate of the number of parasitoid species is as high as 15×10^6 (Hochberg & Lawton 1990). Parasitoids occur in a number of different insect groups (but mostly in the Hymenoptera). They are recognized by their characteristic life cycle which has aspects in common with that of both predators and parasites. The adult female lays her eggs on, in or close to the body of another

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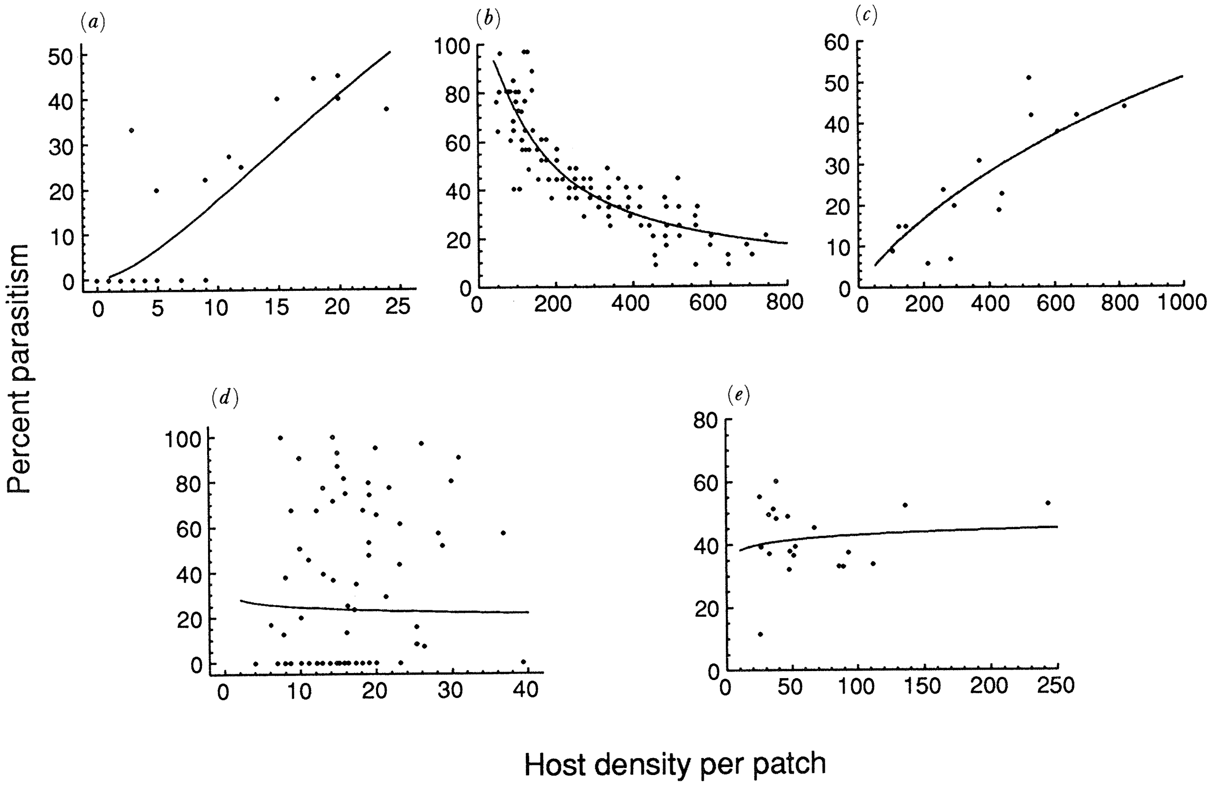

Figure 1. Examples of field studies showing percentage parasitism as a function of host density. Curves are means predicted from equation (3) evaluated at maximum likelihood estimates. (*a*) Direct density dependence from data set 25 in tables 2 and 3; (*b*) inverse density dependence from data set 9; (*c*) direct density dependence from data set 33; (*d*) density independent heterogeneity from data set 14; (*e*) density independent heterogeneity from data set 39. (From Pacala & Hassell 1990.)

arthropod (usually an insect), which is eventually killed by the feeding parasitoid larva. In their effect on a prey population, they are, in effect, predators where the act of 'predation' involves oviposition rather than direct consumption of the prey.

The two attributes of: (i) only the females searching for hosts, and (ii) parasitism defining reproduction, combine to make parasitoids particularly appropriate subjects for the development of generalized predator–prey models such as those of Lotka (1926) and Volterra (1926) and Nicholson & Bailey (1935). In recent years, the emphasis in this work has increasingly been to determine how various forms of heterogeneity can affect host–parasitoid population dynamics. Such heterogeneity can arise in many ways, but most often has been viewed in terms of a patchily distributed host population with different probabilities of parasitism from patch to patch (where the patch may be an arbitrary sampling unit or some clearly recognizable discrete unit of the habitat, such as a leaf or plant).

The widespread interest in the effects of heterogeneity on host–parasitoid dynamics has led many workers to record the distribution of parasitism in the field in relation to the local density of hosts per patch. Of 194 different examples listed in the recent reviews of Lessells (1985), Stiling (1987) and Walde & Murdoch (1988), 58 show variation in attack rates among patches depending directly on host density (figure 1*a*, *c*), 50 show inversely density-dependent relations (figure 1*b*) and 86 show variation uncorrelated with host density (density independent (figure 1*d*, *e*).

A popular interpretation of these data, guided by the earlier theoretical literature (see, for example, Hassell & May (1973, 1974); Murdoch & Oaten (1975)), has been that only the direct density-dependent patterns promote the stability of the interacting populations. This, however, is not the true picture. Both inverse density-dependent patterns (Hassell 1984; Walde & Murdoch 1988), and variation in parasitism that is independent of host density (Chesson & Murdoch 1986; Hassell & May 1988; Pacala *et al.* 1990; Hassell *et al.* 1991), can in principle also be just as important to population regulation. The reasons for this are described below, but arise essentially because any variation in levels of parasitism from patch to patch has the net effect of reducing the mean parasitoid searching efficiency (measured over all hosts) as average parasitoid density increases (the so-called 'pseudointerference' effect of Free *et al.* (1977)). This has obvious implications for the design of field studies on host–parasitoid systems, because no longer can the effects of such heterogeneity be inferred simply from the shape of the relations between percentage parasitism and local host density.

Our aims in this paper are threefold: (i) to show that one can describe the effects of heterogeneity in levels of parasitism using relatively simple criteria; (ii) that this heterogeneity can be broken down into constituent parts that are density dependent and density independent, and (iii) that these measures can be readily applied to the kinds field data that are normally available.

HOST–PARASITOID MODELS IN PATCHY ENVIRONMENTS

Let us consider a habitat which is divided into discrete patches (e.g. food plants for an herbivorous insect) among which adult insects distribute their eggs. The immature stages of these insects are hosts for a specialist parasitoid species whose adult females forage across the patches according to some as-yet-unspecified foraging rule. We also assume that parasitism dominates host mortality such that the hosts are on average kept well below their carrying capacity.

Quite different modelling approaches have developed around this scenario, depending on whether the generations of host and parasitoid are discrete, or continuous with all stages overlapping. Difference equations, stemming back to the Nicholson–Bailey model, have been traditionally used to represent discrete-generation interactions, while differential equations in the Lotka–Volterra tradition have been used for interactions with overlapping generations. The principal difference between the two is that the continuous-time models should exhibit more stable dynamics than the corresponding models in discrete time (May 1974). This view has recently been challenged by Murdoch & Oaten (1989), and is further discussed below.

Yet a third approach to modelling host–parasitoid interactions has recently been developed by R. M. Nisbet and W. S. C. Gurney and colleagues by using systems of time-lagged differential equations incorporating age-structure and developmental rates (Nisbet & Gurney 1983; Gurney, Nisbet & Lawton 1983; Gurney & Nisbet 1985; Murdoch *et al.* 1987). An interesting property of such continuous models with time lags, first shown by Auslander *et al.* (1974), is the appearance of population cycles with a period of roughly one host generation interval. This has recently been more fully discussed by Godfray & Hassell (1987, 1989) who showed that, despite the continuous interaction, such models can show either stable populations with all stages overlapping, or cycles more-or-less recovering the discrete generation interactions described above. Which of these outcomes occur depends largely on the ratio of the lengths of the host and parasitoid life cycles.

(*a*) *Interactions in discrete time*

In this paper, we focus principally on discrete-time interactions and commence with a familiar framework for such interactions (Hassell 1978):

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

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

Here N and P are the host and parasitoid populations in successive generations t and $t+1$, λ is the host's finite rate of increase in the absence of the parasitoid (the fecundity per adult discounted by the average of all mortalities other than parasitism), and w is the average number of female parasitoids emerging from each host parasitized (henceforth assumed to be one). Finally, $f(N_t, P_t)$ is a function giving the average fraction of hosts that escape parasitism, and whose form depends upon all the factors that affect the rate of parasitism of hosts by the P_t searching adult parasitoids. An internal equilibrium of (1*a*, *b*) is defined by: $\lambda f(N^*, P^*) = 1$ and $P^* = N^*(1-1/\lambda)$, and is locally stable if

$$-\lambda^2/(\lambda-1)\, P^* \partial f(N^*, P^*)/\partial P_t < 1, \tag{2a}$$

$$(\lambda-1)/\lambda(-\partial f(N^*, P^*)/\partial P_t) > \partial f(N^*, P^*)/\partial N_t. \tag{2b}$$

In a habitat composed of discrete patches, the term $f(N_t, P_t)$ in (1*a*) and (1*b*) represents the average, across all patches, of the fraction of hosts escaping parasitism. The distribution of hosts in such a patchy setting can either be random or vary in some other prescribed way. Similarly, the density of searching parasitoids in each patch can either be a random variable independent of local host density or a deterministic function of local host density. We call these patterns of heterogeneity in parasitoid distribution host density-independent heterogeneity (HDI) and host density-dependent heterogeneity (HDD), respectively (Pacala *et al.* 1990; Hassell *et al.* 1991). Comparable terms have been coined by Chesson & Murdoch (1986) who labelled models with randomly distributed parasitoids as pure error models and those with parasitoids responding to host density in a deterministic way as pure regression models.

A more biological interpretation of the stability criteria (2*a*) and (2*b*) is as follows. The process of parasitism generates negative covariance between the local (within-patch) abundance of parasitoids and the local abundance of surviving hosts. This is simply because high densities of parasitoids result in high levels of parasitism and correspondingly low densities of surviving hosts. Let C_e be the covariance, at equilibrium and at the end of a growing season, between the local densities of parasitoids and unparasitized hosts divided by the product of the equilibrium mean densities of parasitoids and unparasitized hosts. The division by the product of the means produces a scaled covariance in the same way that dividing a variance by the square of the mean produces a scaled variance (the square of the coefficient of variation). Similarly, let C_b be the scaled covariance of the local parasitoid and total (parasitized and unparasitized) local host densities at equilibrium. C_b may be thought of as the scaled covariance at the beginning of the growing season (before any parasitism takes place).

In the Appendix, we show, under quite general assumptions, that condition (2*a*) is equivalent to $C_e + 1 < (C_b+1)\,Z(\lambda)$, where $Z(\lambda)$ decreases from one to zero as λ increases from one to infinity (λ must be greater than one for the host to be able to persist even in the absence of parasitism). In the vicinity of $\lambda = 1$, $Z(\lambda)$ is approximately equal to $1/\lambda \approx 1$. Thus, as λ increases from one to infinity, condition (2*a*) changes

Table 1. *A range of discrete-time models in which the $CV^2 > 1$ rule is either the exact or an approximate stability criterion*

model no.	HDD or HDI	brief description	$f(N_t, P_t)$	$CV^2 > 1$ rule
I	HDI	any host distribution and parasitoids uncorrelated with hosts (gamma distributed). Reduces to model of May (1978)	$f(P_t) = \int_0^\infty g(\epsilon)\, \mathrm{e}^{-aP_t\epsilon}\, \mathrm{d}\epsilon$	exact
II	HDD	gamma distributed hosts per patch and local parasitoid density a deterministic function of local host density	$f(P_t) = \int_0^\infty g(x)\, x\, \mathrm{e}^{(-aP_t(x)^{\mu})} \mathrm{d}x$	approx.
III	HDI or HDD	a general HDI- and HDD-model of which I, II, and most other published discrete-generation models are special cases. Arbitrary host distribution and parasitoid distribution (either HDI or HDD) given by general functional forms	$f(N_t, P_t) = \int_0^\infty \phi(n) \int_0^\infty \gamma(\epsilon) \sum_{p=0}^{\infty} \frac{\mathrm{e}^{-P_t g(n)\epsilon}[P_t g(n)\, \epsilon]^p}{p!} \mathrm{e}^{-ap}\, \mathrm{d}\epsilon\, \mathrm{d}n$ where $\phi(n)$ is the host distribution, $\gamma(\epsilon)$ is the unit mean distribution of the residual ϵ, and $g(n)$ is the function governing density	approx.
IV	HDI	no spatial structure in the habitat but variation between hosts in their ability to encapsulate parasitoid progeny	$f(P_t) = \int_0^1 q(r)\, \mathrm{e}^{(-aP_t\{1-r\})} \mathrm{d}r$	approx.
V	HDI	evenly distributed hosts. Only a fraction of the parasitoid progeny emerging within a patch enter a 'pool' for subsequent redispersal according to a negative binomial distribution. The remainder stay on in the patches and reproduce there	(see text)	approx.

from $C_e < C_b$ to $C_e < -1$. For dynamics to be stable, the process of parasitism must reduce C_e to a level sufficiently below C_b. In other words, the process of parasitism must sufficiently spatially segregate parasitoids and unparasitized hosts. Obviously, small C_e implies that, at the end of a growing season, parasitoids are found primarily in patches that contain small numbers of uninfected hosts. This spatial segregation reduces the efficiency of the parasitoids and so acts to stabilize the host–parasitoid interaction. The level by which C_e must be reduced relative to C_b is an increasing function of the host's intrinsic rate of increase (λ). Host populations with large λ's rebound quickly and so are more difficult to stabilize than host populations with small λ's.

Stability condition (2*b*) is discussed fully in the Appendix. Suffice it to say here that the condition is always likely to be satisfied and (2*a*) is thus usually necessary and sufficient for stability.

Hassell & May (1988) suggested a very simple approximation of condition (2*a*) which states that interactions of the form of (1*a*, *b*) are stable if the distribution of parasitoids from patch to patch (measured as the square of the coefficient of variation, CV^2) is sufficiently heterogeneous. In particular, if the density of searching parasitoids in the jth patch is q_j ($j = 1, 2, \ldots, z$), the CV^2 of the q_j across the z patches should exceed one. More recently, Pacala *et al.* (1990) and Hassell *et al.* (1990) have extended this work and showed that a very similar criterion applies across a range of models in discrete time. Their criterion differs in that the density of searching parasitoids per patch is now weighted by the number of hosts in that patch. Thus, if p_i is the density of searching parasitoids in the vicinity of the ith host ($i = 1, 2, \ldots, y$), the stability criterion now becomes that the CV^2 of the p_i measured across all y hosts should exceed one. In what follows, 'CV^2' refers exclusively to the coefficient of variation squared of the p_i.

To show the generality of this criterion for discrete-generation host–parasitoid systems, we now survey a range of models in all of which the '$CV^2 > 1$ rule' applies either exactly or approximately. A brief summary is given in table 1 and full details in Hassell *et al.* (1990).

Model I

Consider a specific situation in which the host distribution across patches is arbitrary, and the distribution of parasitoids is unrelated to that of their hosts. It is, therefore, an HDI-model as the heterogeneity is host density independent, local densities of searching parasitoids are determined by chance and by responses to environmental cues that are uncorrelated with host densities per patch. More specifically, let us assume that parasitoid density varies as a gamma distributed random variable from patch to patch, such that the fraction of hosts that escape parasitism in (1*a*, *b*) is given by:

$$f(P_t) = \int_0^\infty g(\epsilon)\, e^{-aP_t\epsilon}\, d\epsilon. \tag{3}$$

Here $g(\epsilon)$ is the gamma probability density function for parasitoids per patch with unit mean and variance $1/\alpha$, where α is a positive constant governing the shape of the density function and a is the usual term for the per capita searching efficiency of the parasitoid. The term $\exp(-aP_t\epsilon)$ is thus the zero-term of a Poisson distribution with mean $aP_t\epsilon$. It gives the probability of a host being attacked zero times by parasitoids that search randomly within a patch containing searching parasitoids at density $P_t\epsilon$.

Hassell *et al.* (1990) show that equation (3) reduces to

$$f(P_t) = [\alpha/(\alpha + aP_t)]^\alpha.$$

A host–parasitoid equilibrium always exists if $\lambda > 1$, and will be locally stable if

$$\alpha[1 - 1/\lambda^{1/\alpha}] < 1 - 1/\lambda, \tag{4}$$

which will always be true if $\alpha < 1$. The CV^2 for gamma distributed parasitoids in this model is simply $1/\alpha$, so that the condition $\alpha < 1$ is identical to $CV^2 > 1$. Notice, incidentally, that this model is formally identical with the phenomenological model proposed by May (1978), in which parasitoid attacks are effectively distributed in a negative binomial manner with clumping parameter, $k (k = \alpha)$.

Model II

From the extreme of no correlation between the spatial distributions of parasitoids and hosts, the second example goes to the opposite extreme of a perfect correlation between the two. Local parasitoid density now deterministically tracks patch-to-patch variation in host density, presumably due to some deterministic foraging rule dominating parasitoid distribution. Specifically, let us suppose it is now the local host density (n) that varies from patch to patch as a gamma distributed random variable with mean N_t. Local parasitoid density is given by a regression function, $P_t(n/N_t)^\mu$, where μ is a constant governing the degree to which parasitoids aggregate in patches of high host density. The aggregation is directly density dependent if $\mu > 0$ and inversely density dependent if $\mu < 0$ (Hassell 1984). This expression has been widely used in host–parasitoid models (see, for example, Hassell & May (1973); Hassell (1984); Kidd & Mayer (1983), and provides better fits than an analogous linear function to the kind of data in figure 1, particularly in accounting for the curvature of inverse density dependence (figure 1*b*). The average fraction of hosts surviving parasitism can now be written as

$$f(P_t) = \int_0^\infty g(x)\, x\, e^{(-aP_t(x)^\mu)}\, dx, \tag{5}$$

where $g(x)$ is the unit mean gamma density and $x = n/N_t$.

Stability in model II is only affected by three parameters λ, μ and α. The relation of these to the CV^2 is shown by the numerical examples in figure 2. The $CV^2 > 1$ rule is now only the approximate condition for stability, but the approximation is good for values of λ near one and for highly aggregated host distributions (small values of α) (figure 2).

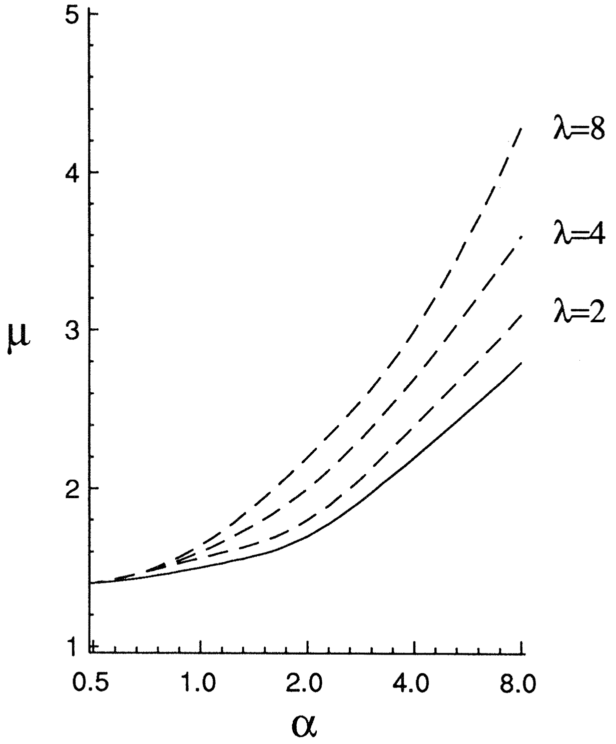

Figure 2. Stability criterion for model II. The internal equilibrium is stable for values of μ and α above the appropriate dashed curve, and is unstable for values below. The solid curve shows values of μ and α at which $CV^2 = 1$. $CV^2 > 1$ above the curve and $CV^2 < 1$ below. (From Hassell *et al.* 1990.)

Model III

Models I and II represent end-points of the continuum between HDI and HDD. They also include restrictive, though reasonable, assumptions about the different functional forms (e.g. gamma distributed populations or power-law dependence of local parasitoid on local host densities). We now consider a much more general model that relaxes these assumptions: spatial distributions are left unspecified and any degree of spatial covariance between parasitoid and host is allowed.

Specifically, we assume that the distribution of the relative numbers ($n = N/N_t$) of hosts in patches does not change with host density, but otherwise can be of any distribution whatsoever. We also assume that the mean density of parasitoids in a patch is given by the function $P_t g(n) U$. Here, g is an arbitrary function of host density that determines host density dependent aggregation by parasitoids. In contrast, U is a random variable with a mean of one that determines the level of HDI. This random variable creates heterogeneity among patches in their relative attractiveness to parasitoids that is independent of the host abundances in the patches. Finally, we assume that the actual number of parasitoids visiting a patch is Poisson distributed about the patch-specific mean, $P_t g(n) U$.

With these assumptions, the fraction of parasitoids that escape parasitism, $f(N_t, P_t)$, is given by $E(e^{-ap})$, where E is the expectation across all hosts and p is Poisson distributed about a mean, $P_t g(n) U$, which is itself a random variable (because of U) and a function of host density. It is important to note that between-patch variation in $P_t g(n) U$ reflects non-random aggregation by parasitoids because it reflects differences among patches to which parasitoids respond. In contrast, the Poisson variation in p reflects a purely random distribution of parasitoids.

The analysis of this general model is possible if λ is close to one. The derivation in Hassell *et al.* (1990) shows that the host–parasitoid interaction is stable if approximately $CV^2 - 1/p^* > 1$, where p^* is the average number (not density) of parasitoids that visit a patch at equilibrium. This average is again calculated with respect to a randomly chosen host. If, on average, there are several parasitoid visits per patch, then we approximately recover the $CV^2 > 1$ rule. If, on the other hand, the average visits per patch is very low (e.g. $\leqslant 1$) the value of CV^2 required for stability will increase. For example, with an average of one parasitoid visit per patch the stability criterion becomes $CV^2 > 2$. This arises because the term $1/p^*$ is the component of CV^2 that is caused solely by purely random (Poisson) variation in parasitoid abundance among patches. Because this quantity is subtracted from CV^2, Poisson variation does not contribute in any way to stability. Thus, host density independent heterogeneity only facilitates stability if it reflects non-random aggregation of parasitoids.

The next 2 models are used to demonstrate that the $CV^2 > 1$ rule is robust to at least some major changes in the biological assumptions underpining models I–III.

Model IV

The previous models have all been set in an explicitly patchy environment. We now turn to a model that breaks away from this mould and shows that the $CV^2 > 1$ rule can also apply when the heterogeneity arises in quite different ways. Insects in general possess a powerful haemocytic defence mechanism that enables them to encapsulate foreign objects, such as parasitoid eggs and larvae, recognized as ‘non-self’. Heterogeneity now arises if there is variability between individual hosts in their ability to encapsulate parasitoids within them. Godfray & Hassell (1990) consider two cases: (i) all-or-none encapsulation where the probability of a host individual escaping parasitism by encapsulation is constant, irrespective of the number of parasitoid larvae it contains, and (ii) dosage-dependent encapsulation where the probability of a host surviving parasitism decreases with parasitoid load. Other forms of encapsulation (e.g. a threshold number of parasitoid larvae required to trigger the host’s response) are possible, but were not considered.

In all-or-none encapsulation there are essentially two classes of host: those not encountered by parasitoids and those encountered one or more times. Within the framework of equation (1), this reduces to a straightforward refuge model whose properties are well-known (Hassell 1978). Furthermore, if heterogeneity is now introduced by individual hosts varying in their ability to encapsulate parasitoids Godfray & Hassell (1990) show this to have no affect on population dynamics.

With dosage-dependent encapsulation, however, explicit account must now be taken of hosts that are attacked once, twice, three times, etc. Assuming random search and a fixed probability, r, of a parasitoid being encapsulated leads directly back to the Nicholson–Bailey model with the searching parameter a reduced to $a' = a(1-r)$. The stability properties, which are independent of a', are thus unchanged. This is no longer the case, however, if heterogeneity in the ability of hosts to encapsulate is introduced. Let us assume that r now varies randomly among hosts with probability density function $q(r)$. If the number of attacks per host is a Poisson random variable, if one egg is laid per attack and if the probability that any one host successfully encapsulates n eggs is r^n, then we may write $f(\cdot)$ as

$$f(P_t) = \int_0^1 q(r)\, \mathrm{e}^{(-aP_t\{1-r\})}\, \mathrm{d}r. \tag{6}$$

In effect, each host is being viewed as a patch and, because of inter-host variation in r, there is inter-patch variation in the mean level of successful parasitism. Godfray & Hassell (1990) then derive a local stability criterion, whereby as long as λ is not too large, then the necessary and sufficient condition for local stability is approximately

$$\sigma_r^2/(1-\bar{r})^2 > 1, \tag{7}$$

where σ_r^2 is the variance of r, and $(1-r)$ is the mean. Thus, once again the $CV^2 > 1$ rule emerges as an approximate stability condition.

Model V

The preceding models assume that all hosts and parasitoids redistributed themselves each generation among the available patches. While there are many natural examples of this, particularly from univoltine species, there are also many cases of less complete mixing, where some of the hosts and parasitoids tend to remain within the patch from which they originated. This final model caters for this by allowing some hosts and parasitoids to stay in the patches from which they emerged, while the remainder enter a 'pool' to be redistributed anew in the next generation (Hassell & May 1988; Reeve 1988; Hassell *et al.* (1990)). There is thus a continuum from complete host or parasitoid mixing to no host mixing at all. Assuming, for simplicity, that the probability of leaving a patch is density independent and that there is no mortality associated with the movement, the hosts and adult parasitoids in the ith patch, N_i and P_i respectively, are now given by

$$N_i(t+1) = \lambda\left[S_i(1-x_i) + \alpha_i\left\{\sum_{j=1}^{n} S_j x_j\right\}\right], \tag{8a}$$

$$P_i(t+1) = N_{ai}(1-y_i) + \beta_i\left\{\sum_{j=1}^{n} N_{aj} y_j\right\}. \tag{8b}$$

Here α_i and β_i are the fractions of dispersing hosts and parasitoids, respectively, that enter the ith patch, S_i is the number of these hosts surviving from parasitism and N_{ai} the number of hosts parasitized $(N_i[1-\exp(-aP_i)])$. Finally, x_i and y_i are the fraction of host and parasitoid progeny, respectively, leaving the patch for subsequent redistribution in the next generation, and n is the total number of patches.

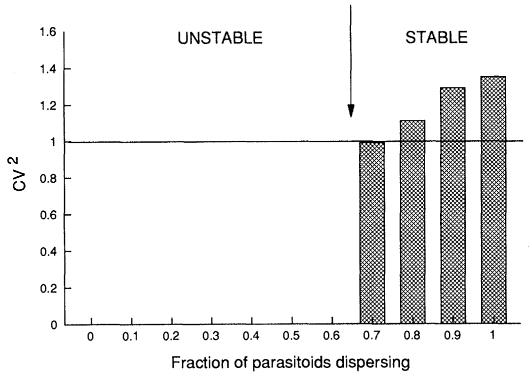

Figure 3. A numerical example from model V. The histogram bars show the values of CV^2 at equilibrium as a function of the fraction of parasitoids that disperse each generation (y in equation (8)). The model is stable, with $CV^2 \geqslant 1$, provided that at least 70 % of the parasitoids disperse each generation. Below this value, the model shows limit cycles and CV^2 fluctuates with mean values less than one. Parameter values are: $\lambda = 2$, $a = 0.01$, k (negative binomial) $= 1$ and $n = 20$. (From Hassell *et al.* 1990.)

Numerical studies indicate that the CV^2 rule is a good indicator of stability for the model. For example, suppose that there is complete host mixing in each generation ($x_i = 1$), and that the fraction of parasitoids mixing varies from zero to one. We assume an even host distribution, and that those parasitoids that do disperse redistribute themselves according to a negative binomial distribution. The model is thus an HDI-model, not dissimilar to model I. Figure 3 shows a numerical example in terms of the CV^2 of the parasitoid distribution at equilibrium as the fraction of parasitoid mixing is changed. Once again the $CV^2 > 1$ rule is a good indicator of stability.

(*b*) *Parasitoid aggregation and the heterogeneity of risk*

Chesson & Murdoch (1986) define a quantity, ρ, the relative risk of parasitism for a host individual, as a means of unifying the analysis of both HDI-models (their 'pure-error' models) and HDD-models (their 'pure-regression' models). Heterogeneity in this risk of parasitism is the commodity that affects the stability of the populations. The '$CV^2 > 1$ rule', in contrast, is defined in terms of heterogeneity in the distribution of searching parasitoids. In models I–V (with assumed type I functional responses) this is generally the same as heterogeneity in the risk of parasitism. As Ives (1990) has recently pointed out, however, the distribution of parasitoids may differ from the distribution of risk if functional responses are sufficiently nonlinear. Thus in cases involving nonlinear functional responses, CV^2 refers specifically to heterogeneity in risk of parasitism. Further numerical work is needed to assess the generality of the $CV^2 > 1$ rule when functional responses are markedly nonlinear over realistic ranges of host densities per patch.

(*c*) *Continuous redistribution in discrete time*

Finite difference equation models such as (1) are probably most appropriate for species with non-overlapping generations. Such species are more typical of seasonal habitats, although Godfray & Hassell (1987, 1989) showed that developmental time-lags can, under some conditions, lead to cycles of approximately one generation period in species that would otherwise reproduce continuously. Even so, many continuously reproducing species do have overlapping generations and are probably best modelled by systems of differential equations.

Although restricted to species with non-overlapping generations, the discrete time model (1) can account for death and movement that occur continuously within a discrete generation. Models I–III, however, are most applicable to host species that are distributed amongst patches at the beginning of a growing season and do not disperse until the following season. The parasitoids in these models, however, may be viewed as either sedentary or highly dispersive (or anything in between). For example, model I is appropriate if parasitoids are sedentary within a season and are gamma distributed amongst patches, or if the time spent in each patch by each continuously dispersing parasitoid is gamma distributed.

In contrast, rapid within-season movement by hosts tends to expose each host to the same suite of parasitoid densities and hence to the same risk of parasitism. Rapid host movement can thus decrease heterogeneity (CV^2) caused by parasitoids. Godfray & Pacala (1990) show that with infinitely rapid within-season movement, and thus no heterogeneity of risk, the system collapses to the unstable Nicholson–Bailey model if parasitoid aggregation is independent of host density. If aggregation is density dependent, however, stability is possible if the host distribution is sufficiently clumped.

(*d*) *Overlapping generations in continuous time*

In this section we turn to continuous-time analogues of (1) and argue that, despite recent claims to the contrary (Murdoch & Oaten 1989), heterogeneity is also generally stabilizing in continuous-time host–parasitoid models.

Perhaps the most simple system of differential equations in a patchy environment analogous to (1) is:

$$\mathrm{d}N_i/\mathrm{d}t = bN_i - aP_i\,N_i - e_n\,N_i + z_i\,e_n \sum_{j=1}^{Q} N_j, \tag{9a}$$

$$\mathrm{d}P_i/\mathrm{d}t = vaP_i\,N_i - \mathrm{d}P_i - e_p\,P_i + q_i\,e_p \sum_{j=1}^{Q} P_j, \quad (1 = 1, 2, \ldots Q), \tag{9b}$$

where P_i and N_i are the local densities of parasitoid and host in patch i, b is the density independent growth rate of the host, a is the mean searching efficiency of the parasitoid, ν is the reproductive efficiency of the parasitoid, d is the death rate of the parasitoid, and e_n and e_p are the rates at which hosts and parasitoids disperse. Finally, z_i and q_i are the fractions of dispersing hosts and parasitoids, respectively, that enter patch i, where q_i may be either constant or a function of local host density.

The model (9) assumes that parasitized hosts that have not yet died have no effect on dynamics. The inclusion of living, but parasitized 'zombies' would make the system more directly analogous to the discrete-time models above (Godfray & Pacala 1991). Zombies affect dynamics because most parasitoids cannot perfectly distinguish infected and uninfected hosts. If parasitoids disperse to regions of high total host density, then parasitoids will occur primarily where uninfected hosts do not. This is one mechanism that reduces C_e (spatial covariance between uninfected hosts and parasitoids) and thus stabilizes the dynamics of discrete-time models (see above).

Host–parasitoid models such as (9) have received much less attention than corresponding models in discrete time. In a recent study, Godfray & Pacala (1991) showed that (9) has neutrally stable dynamics in several cases where there is no heterogeneity of risk at equilibrium – for example, when there is (1) no dispersal $e_n = e_p = 0$, (2) purely homogeneous dispersal ($z_i = z_j$ and $q_i = q_j$ for all i and j), and (3) infinite dispersal ($e_n \rightarrow \infty$, $e_p \rightarrow \infty$). If there is finite and heterogeneous dispersal, however, they showed that heterogeneity of risk caused by parasitoid aggregation is typically stabilizing, just as it is in discrete models.

In a recent study of continuous-time models, Murdoch & Oaten (1989) arrive at the opposite conclusion. Specifically, they contend that density independent aggregation has no effect on stability in continuous time and that the effect of density dependent aggregation depends on the rate at which the variance in local host density changes with the mean. If this variance is equal to AN^x, where N is the mean host density and A and x are constants then density dependent aggregation is stabilizing for $x > 2$ and destabilizing for $x < 2$. They argue that the latter case is more common in nature (Taylor *et al.* 1980). Interestingly, these results are identical to those obtained by Godfray & Pacala (1991) from their discrete-time model referred to above with infinitely rapid host and parasitoid movement within each season.

Murdoch & Oaten (1989) do not explicitly model the dynamics of each of several spatial patches. Rather, their model consists of a pair of differential equations governing the mean parasitoid and host abundances across all patches. They also assume that the covariance of local parasitoid and host abundances is given by one of several simple functions of the mean abundances across all patches. Godfray & Pacala (1990) show that Murdoch & Oaten's model is obtained from explicitly spatial models, such as (9), in the limit of infinitely rapid movement by both species (i.e. $e_n \rightarrow \infty$, $e_p \rightarrow \infty$), or if dispersal is governed by complex and biologically implausible density dependent rules.

Since the host stages attacked by parasitoids are generally relatively sedentary, and some degree of heterogeneity of risk is likely to be the rule in nature systems, models with infinite interpatch movement and no heterogeneity of risk would generally seem to be inappropriate. We conclude that aggregation by parasitoids generally acts to stabilize host–parasitoid interactions whether in continuous or discrete time.

ESTIMATING PARAMETERS FROM FIELD DATA

(*a*) *The method*

The $CV^2 > 1$ rule is in terms of the distribution of searching parasitoids. Such data, however, are rarely available from natural populations (see also Waage 1983; Casas 1988, 1989); most of the information is in the form of relationships between percent parasitism and host density per patch (figure 1). In this section we show how the parameters necessary to calculate the CV^2 can be estimated from such data. Full details of the technique are given in Pacala & Hassell (1990).

We begin by assuming that the local parasitoid density can be described by the regression function (see model II above) with multiplicative residual ϵ:

$$p = cP_t\left(\frac{n}{N_t}\right)^{\mu}\epsilon, \qquad (10)$$

where n is the local host density, c and μ are constants, and ϵ is a unit mean gamma distributed random variable (as for model 1). Thus, host density dependent heterogeneity is described by the expression $(n/N_t)^{\mu}$, with the magnitude of the association between local parasitoid and local host density governed by the value of μ. The magnitude of any host density independent heterogeneity is determined by the gamma distributed random variable ϵ, with the magnitude of HDI increasing with the variance of ϵ. (Note that the methods described below also apply to functional forms of HDD other than (10). To extend the methods outlined below to another function $g(n)$, the steps are merely repeated after substituting the new $g(n)$ for $(n/N_t)^{\mu}$.)

With these assumptions

$$f(N_t, P_t) = \int_0^{\infty}\int_0^{\infty} \phi(n/N_t)\,\gamma(\epsilon)\,\frac{n}{N_t} \times \exp\left(-acP_t(n/N_t)^{\mu}\epsilon\right)\mathrm{d}\epsilon\,\mathrm{d}n, \qquad (11)$$

where $\phi(n/N_t)$ is the distribution of n/N_t and $\gamma(\epsilon)$ is the unit-mean gamma density.

Pacala & Hassell (1991) show that $CV^2 > 1$ from this general model may be approximated as:

$$CV^2 \approx C_I C_D - 1. \qquad (12)$$

Here $C_I = 1+\sigma^2$ and represents the component of heterogeneity that is independent of host density, and $C_D = 1 + V^2\mu^2$ and represents the component that depends on host density. The term σ^2 is the variance of ϵ and V is the coefficient of variation of the local host density calculated with respect to a randomly chosen host.

Notice from (12) that (1) $CV^2 > 1$ if either $C_I > 2$ or $C_D > 2$, (2) that C_I and C_D both affect $CV^2 > 1$ in the same way and (3) that because the slope-determining expression μ in C_D is squared, the effects of direct (positive slope) and inverse (negative slope) dependence on local host density are identical. Thus all of the patterns shown in figure 1 contribute to population regulation in the same way irrespective of whether the density dependence is positive or negative, or if there is no density dependence at all. The degree to which each affects stability just depends on the magnitude of the CV^2.

The method of obtaining CV^2 from field data involves estimating μ, σ^2 and acP_t from equation (11) by using a maximum likelihood procedure, and estimating V^2 for the host distribution directly from the data on local host densities. (Values of σ^2 were constrained to be $\geqslant 0.05$ to prevent numerical overflow during computation. As a result, estimates of σ^2 reported as 0.05 are actually between 0 and 0.05. In these cases, the calculated value of CV^2 is an overestimate, but the bias is less than 5% (Pacala & Hassell 1991).

(*b*) *The data*

The data set that we have analysed involves 65 examples from field studies reporting percent parasitism versus local host density per patch, for each of which we have obtained estimates of σ^2, μ and V^2, and thence calculated C_I, C_D and CV^2 (table 3). Of these, 32 are listed in two recent reviews by Stiling (1987); Walde & Murdoch (1988). We have also added a further 33 examples, mainly from unpublished studies. The full assemblage is listed in table 2. Other studies, listed but not used here, were found to be unsuitable for our analysis, usually because the per patch sample sizes were unreported. Several of these data sets are temporal or spatial replicates of others. We have, therefore, also produced a reduced assemblage of 26 data sets by choosing a single replicate at random and omitting all others (designated by an asterisk in the first column of the table 3). Each of this reduced set thus describes a different pair of species.

(*c*) *Results*

For each example in tables 2 and 3, the estimated value of the CV^2 can be evaluated in relation to the $CV^2 > 1$ rule and the relative importance of density dependent and density independent heterogeneity (HDD versus HDI) to the total heterogeneity determined. It is also possible in each case to predict the mean percent parasitism in relation to host density per patch from the expression:

$$100\left[1-\alpha\left(\alpha+acP_t\left(\frac{n}{N_t}\right)^{\mu}\right)\right]^{\alpha}, \qquad (13)$$

where α, acP_t and μ are the maximum likelihood estimates referred to above. This is shown for the range of examples in figure 1 where each fitted curve represents the percent parasitism predicted by the mean of (11) evaluated at the maximum likelihood estimates. In all cases the correspondence is quite close between the predicted and actual mean levels of parasitism.

Figure 1*a* shows an example of a direct density dependent pattern of parasitism (data set 25 in tables

Table 2. *List of data sets analysed*

no.	host species (family)	parasitoid species (family)	author(s)
1	*Bupalus piniaria* (Geometridae)	*Dusona oxyacanthae* (Ichneumonidae)	N. Broekhuizen (unpublished data)
2	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
3	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
4	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
5	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
6*	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
7	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
8	*B. piniaria*	*D. oxyacanthae*	N. Broekhuizen (unpublished data)
9*	*Lymantria dispar* (Lymantriidae)	*Ooencyrtus kuwanai* (Encyrtidae)	Brown & Cameron (1979)
10*	*Trirhabda virgata* (Chrysomelidae)	Mirmithid nematodes	N. Cappuccino (unpublished data)
11*	*Microrhopala virtata* (Chrysomelidae)	Erythreid mites	N. Cappuccino (unpublished data)
12*	*Eurosta solidasinus* (Tephritidae)	*Eurytoma gigantea* *E. obtusiventris* (Eurytomidae)	N. Cappuccino (unpublished data)
13*	*Rhopalomyia californica* (Cecidomyiidae)	*Torymus baccaridis* (Torymidae)	Ehler (1986)
14*	*R. californica*	*Tetrasticus* sp.	Ehler (1986)
15*	*Silo pallipes* (Goeridae)	*Agriotypus armatus* (Agriotypidae)	Elliott (1983)
16*	*Sceliphron assimile* (Sphecidae)	*Mellittobia chalybii* (Eulophidae)	Freeman & Parnell (1983)
17	*Andricus quercuscalicis* (Cynipidae)	*Mesopolobus fuscipes* (Pteromalidae)	Hails (1988)
18*	*A. quercuscalicis*	*M. fuscipes*	Hails (1988)
19*	*A. quercuscalicis*	*Mesopolobus xanthocerus*	Hails (1988)
20	*A. quercuscalicis*	*M. xanthocerus*	Hails (1988)
21*	*Phytomyza ilicis* (Agromyzidae)	*Chrysocharis gemma* (Eulophidae)	Heads & Lawton (1983)
22*	*Papilio xuthus* (Papilionidae)	*Trichogramma papilionis* (Trichogrammatidae)	Hirose *et al.* (1976)
23	*Delia radicum* (Anthomyiidae)	*Trybliographa rapae* (Eucoilidae)	Jones & Hassell (1988)
24	*D. radicum*	*T. rapae* (Eucoilidae)	Jones & Hassell (1988)
25	*D. radicum*	*T. rapae*	Jones & Hassell (1988)
26	*D. radicum*	*T. rapae*	Jones & Hassell (1988)
27*	*D. radicum*	*T. rapae*	Jones & Hassell (1988)
28	*D. radicum*	*T. rapae*	Jones & Hassell (1988)
29	*D. radicum*	*T. rapae*	T. H. Jones (unpublished data)
30	*D. radicum*	*T. rapae*	T. H. Jones (unpublished data)
31	*D. radicum*	*T. rapae*	T. H. Jones (unpublished data)
32*	*Chirosia histricina* (Anthomyiidae)	Total parasitism	J. H. Lawton (unpublished data)
33*	*Fiorinia externa* (Diaspidae)	*Aspidiotiphagus citrinus* (Eulophidae)	McClure (1977)
34	*F. externa*	*A. citrinus*	McClure (1977)
35	*F. externa*	*A. citrinus*	McClure (1977)
36*	*Epinotia tedella* (Tortricidae)	*Apanteles tedellae* (Braconidae)	Munster-Swendsen (1980)
37*	*E. tedellae*	*Pimplopterus dubius* (Ichneumonidae)	Munster-Swendsen (1980)
38	*Parlatoria oleae*	*Aphytis paramaculicornis* (Aphelinidae)	Murdoch *et al.* (1984)
39*	*P. oleae*	*Coccophagoides utilis* (Aphelinidae)	Murdoch *et al.* (1984)
40*	*P. oleae*	*A. paramaculicornis*	Murdoch *et al.* (1984)
41	*Coleophora laricella* (Coleophoridae)	*Agathis pumila* (Braconidae)	R. B. Ryan (unpublished data)
42*	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
43	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
44	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)

Table 2. (*cont.*)

45	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
46	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
47	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
48	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
49	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
50	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
51	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
52	*C. laricella*	*A. pumila*	R. B. Ryan (unpublished data)
53	*Aonidiella aurantii* (Diaspidae)	*Aphtyis melinus* (Aphelinidae)	Smith & Maelzer (1986)
54*	*A. aurantii*	*A. melinus*	Smith & Maelzer (1986)
55	*A. aurantii*	*A. melinus*	Smith & Maelzer (1986)
56	*A. aurantii*	*A. melinus*	Smith & Maelzer (1986)
57*	*Eupteryx cyclops* *E. urticae* (Cicadellidae)	*Anagrus* sp. (Mymaridae)	Stiling (1980)
58*	*Polistes exclamans* (Vespidae)	*Elasmus polistis* (Elasmidae)	Strassmann (1981)
59	*P. exclamans*	*E. polistis*	Strassmann (1981)
60*	*Icerya purchasi* (Margarodidae)	*Cryptochaetum iceryae* (Cryptochaetidae)	Thorarinsson (1990)
61	*I. purchasi*	*C. iceryae*	Thorarinsson (1990)
62	*I. purchasi*	*C. iceryae*	Thorarinsson (1990)
63	*I. purchasi*	*C. iceryae*	Thorarinsson (1990)
64*	*Trypargilum politum* (Sphecidae)	*Melittobia* sp. (Eulophidae)	Trexler (1985)
65	*Lymantria dispar* (Lymantriidae)	*Ooencyrtus kuwanai* (Encyrtidae)	Weseloh (1972)

* Reduced list with no spatial or temporal replicates.

2 and 3). The calculated CV^2 for this data set is 1.16, from which we can predict (assuming this to be a typical result in successive generations) that such levels of heterogeneity in parasitism would be just sufficient to stabilize the interaction. The relatively large value for C_D (2.06) and small value for C_I (1.05) indicate that virtually all of the stabilizing heterogeneity in this example comes from the host density dependent heterogeneity (HDD). This arises from two biological properties of the interacting populations: (i) the large spatial covariance of the parasitoids with local host density, indicated by the relatively large value of μ (= 1.37), and (ii) the highly aggregated spatial distribution of the host (indicated by the relatively large coefficient of variation of the host population (V^2 = 0.66).

Figure 1*b* shows a further example (data set 9) where parasitism is strongly correlated with host density per patch, but in this case the pattern is inversely density dependent ($\mu = -0.95$). However, in contrast to the previous example, HDD is only slightly larger than HDI ($C_D = 1.29$; $C_I = 1.11$) and they thus combine to produce only a low level of total heterogeneity in parasitism ($CV^2 = 0.37$). The small effect of HDD, despite the relatively large value of μ, stems from the low level of spatial aggregation of the host population ($V^2 = 0.25$). In short, such levels of heterogeneity would be too small to contribute significantly to population regulation. This prediction is reassuring in the light of the spectacular quasi-periodic outbreaks sometimes shown by the gypsy moth (Myers 1988).

Figure 1*c* (data set 33) provides a further example of pronounced spatial density dependence ($\mu = 0.87$) but a low value of CV^2 (= 0.34). Once again, there is too little aggregation in the host distribution ($V^2 = 0.21$) for the HDD to contribute appreciably to heterogeneity.

Figure 1*d* (data set 14) shows a case that is quite different from the previous three. Density independent (HDI) variation in parasitism is now much more important than density dependent variation (HDD) ($C_I = 8.25$; $C_D = 1.01$). Furthermore, because C_I is significantly greater than 2, heterogeneity independent of host density is large enough by itself to stabilize the interacting populations. The prediction of stability thus does not depend at all on the host's spatial distribution, or on the existence of spatial covariance (beginning-of-season) between the parasitoid and host. Hence, although the data in 1*d* appear erratic, they actually contain more evidence of factors that could stabilize dynamics than do the previous examples.

Finally, figure 1*e* (data set 39) shows another example with virtually no stabilizing heterogeneity. There is no appreciable effect from HDI (the estimate of σ^2 is at the constrained lower bound), and there is no sign of the parasitoids responding to local host density ($\mu = 0.07$).

Rather than survey each of the 65 examples in table 2 in this one-by-one way, we now turn to a broader comparison of the range of CV^2 values and the relative contribution to these of HDD and HDI heterogeneity. In the first place, figure 4 shows all the examples plotted in relation to the values of C_I and C_D, with the $CV^2 = 1$ contour also overlaid. In 18 of the 65 cases, $CV^2 > 1$ indicating that heterogeneity at this level ought to be

Table 3. *Maximum likelihood estimates used in calculating C_D, C_I and CV^2*

no.	host species (family)	C_D (= HDD)	C_I (= HDI)	CV^2	μ (95 % limits)	σ^2 (95 % limits)	acP (95 % limits)	V^2 (95 % limits)
1	*Bupalus piniaria* (Geometridae)	1.04	1.26	0.30	0.34 (−0.46 : 1.18)	0.26 (0.05 : 20.0)	0.04 (0.02 : 0.06)	0.32
2	*B. piniaria*	1.04	1.11	0.10	0.53 (−0.13 : 1.19)	0.05 (0.05 : 0.26)	0.75 (0.60 : 1.01)	0.16
3	*B. piniaria*	1.05	1.06	0.12	0.60 (−0.52 : 1.61)	0.06 (0.05 : 20.0)	0.36 (0.20 : 0.60)	0.17
4	*B. piniaria*	1.02	1.60	0.63	0.22 (−0.49 : 1.02)	0.60 (0.05 : 2.81)	0.07 (0.05 : 1.12)	0.45
5	*B. piniaria*	1.08	1.05	0.14	−0.86 (−2.12 : 0.68)	0.05 (0.05 : 20.0)	0.47 (0.23 : 0.86)	0.11
6*	*B. piniaria*	1.99	2.30	3.48	1.87 (0.67 : 3.34)	1.25 (0.17 : 5.44)	0.06 (0.03 : 0.15)	0.29
7	*B. piniaria*	1.04	3.87	3.03	0.25 (−0.79 : 1.37)	2.87 (0.65 : 15.1)	0.02 (0.00 : 0.04)	0.68
8	*B. piniaria*	1.44	3.30	3.73	0.86 (−0.54 : 2.31)	2.30 (0.05 : 20.0)	0.02 (0.01 : 0.04)	0.61
9*	*Lymantria dispar* (Lymantriidae)	1.29	1.11	0.37	−0.95 (−1.0 : 0.87)	0.11 (0.05 : 0.15)	0.49 (0.47 : 0.52)	0.25
10*	*Trirhabda virgata* (Chrysomelidae)	1.00	2.63	1.64	0.77 (0.13 : 1.36)	1.63 (0.93 : 2.97)	0.52 (0.37 : 0.74)	0.01
11*	*Microrhopala virtata* (Chrysomelidae)	1.01	2.60	1.62	0.20 (−0.35 : 0.83)	1.60 (0.68 : 4.43)	0.04 (0.03 : 0.06)	0.26
12*	*Eurosta solidasinus* (Tephritidae)	1.18	1.05	0.24	−0.61 (−1.21 : −0.10)	0.05 (0.05 : 0.50)	0.60 (0.39 : 0.88)	0.48
13*	*Rhopalomyia californica* (Cecidomyiidae)	1.01	4.00	3.02	0.25 (−0.93 : 1.00)	2.99 (1.91 : 4.74)	0.41 (0.28 : 0.61)	0.10
14*	*R. californica*	1.01	8.25	7.33	−0.24 (−1.23 : 0.71)	7.25 (4.70 : 11.2)	0.39 (0.26 : 0.61)	0.20
15*	*Silo pallipes* (Goeridae)	1.01	1.05	0.06	−0.20 (−0.84 : 0.47)	0.05 (0.05 : 0.19)	0.22 (0.15 : 0.30)	0.18
16*	*Sceliphron assimile* (Sphecidae)	1.04	1.23	0.28	0.20 (0.01 : 0.48)	0.23 (0.12 : 0.38)	0.70 (0.48 : 0.94)	1.03
17	*Andricus quercuscalicis* (Cynipidae)	1.00	1.92	0.92	−0.02 (−0.28 : 0.24)	0.92 (0.37 : 1.73)	0.09 (0.07 : 0.11)	0.57
18*	*A. quercuscalicis*	1.06	1.75	0.86	−0.27 (−0.48 : −0.06)	0.75 (0.14 : 1.50)	0.07 (0.06 : 0.09)	0.86
19*	*A. quercuscalicis*	1.10	3.60	2.96	−0.42 (−0.72 : −0.10)	2.60 (1.49 : 4.13)	0.10 (0.08 : 0.13)	0.57
20	*A. quercuscalicis*	1.08	1.66	0.79	−0.30 (−0.51 : −0.10)	0.66 (0.07 : 1.30)	0.07 (0.06 : 0.09)	0.86
21*	*Phytomyza ilics* (Agromyzidae)	1.09	1.23	0.34	0.57 (0.12 : 1.03)	0.23 (0.20 : 0.53)	0.19 (0.15 : 0.24)	0.28

22*	*Papilio xuthus* (Papilionidae)	1.16	1.05	0.21	−0.69 (−1.56:0.09)	0.05 (0.05:2.00)	1.11 (0.64:1.83)	0.33
23	*Delia radicum* (Anthomyiidae)	1.62	1.05	0.70	1.59 (0.59:2.00)	0.05 (0.05:0.11)	0.11 (0.07:0.18)	0.24
24	*D. radicum*	1.47	1.11	0.63	0.91 (0.60:1.23)	0.11 (0.05:0.39)	0.33 (0.26:0.42)	0.57
25	*D. radicum*	2.06	1.05	1.16	1.37 (1.14:1.65)	0.05 (0.05:0.22)	0.09 (0.07:0.12)	0.56
26	*D. radicum*	1.36	1.05	0.43	0.88 (0.65:1.10)	0.05 (0.05:0.08)	0.28 (0.23:0.35)	0.47
27*	*D. radicum*	1.00	1.79	0.79	−0.10 (−0.55:0.39)	0.79 (0.31:1.79)	0.30 (0.21:0.44)	0.36
28	*D. radicum*	1.00	1.05	0.05	−0.09 (−0.54:0.37)	0.05 (0.05:0.13)	0.30 (0.23:0.38)	0.25
29	*D. radicum*	1.01	1.05	0.06	0.17 (−0.26:0.60)	0.05 (0.05:0.13)	0.27 (0.21:0.35)	0.28
30	*D. radicum*	1.27	1.05	0.34	0.51 (0.20:0.78)	0.05 (0.05:0.13)	0.13 (0.08:0.18)	1.05
31	*D. radicum*	1.01	1.05	0.06	0.13 (−0.65:0.84)	0.05 (0.05:0.11)	0.11 (0.07:0.17)	0.30
32*	*Chirosia histricina* (Anthomyiidae)	1.00	1.12	0.12	−0.05 (−0.55:0.48)	0.12 (0.05:0.96)	0.33 (0.22:0.49)	0.36
33*	*Fiorinia externa* (Diaspidae)	1.16	1.16	0.34	0.87 (0.52:1.12)	0.16 (0.08:0.37)	0.33 (0.27:0.40)	0.21
34	*F. externa*	1.02	1.08	0.10	0.40 (0.32:0.49)	0.08 (0.05:0.14)	0.38 (0.35:0.39)	0.15
35	*F. externa*	1.08	1.05	0.13	0.63 (0.45:0.80)	0.05 (0.05:0.06)	0.58 (0.54:0.64)	0.20
36*	*Epinotia tedella* (Tortricidae)	1.08	1.50	0.61	1.03 (0.10:2.00)	0.50 (0.05:0.68)	0.04 (0.03:0.05)	0.07
37*	*E. tedellae*	1.02	1.40	0.42	−0.43 (−1.39:0.39)	0.40 (0.40:0.49)	0.04 (0.03:0.05)	0.07
38	*Parlatoria oleae*	1.07	1.05	0.12	0.36 (0.09:0.64)	0.05 (0.05:0.19)	0.23 (0.19:0.28)	0.51
39*	*P. oleae*	1.00	1.05	0.05	0.07 (−0.14:0.28)	0.05 (0.05:0.15)	0.56 (0.49:0.63)	0.61
40*	*P. oleae*	1.05	1.05	0.10	0.51 (0.19:0.86)	0.05 (0.05:0.16)	0.65 (0.56:0.76)	0.18
41	*Coleophora laricella* (Coleophoridae)	1.06	2.40	1.54	−0.57 (−2.15:0.89)	1.40 (0.26:11.7)	0.10 (0.05:0.22)	0.17
42*	*C. laricella*	1.00	1.13	0.13	−0.02 (−0.73:0.74)	0.13 (0.05:0.55)	0.15 (0.11:0.21)	0.12
43	*C. laricella*	5.00	1.41	6.06	0.50 (−0.10:1.17)	0.41 (0.18:1.37)	0.24 (0.16:0.36)	16.19
44	*C. laricella*	1.00	2.05	0.05	−0.06 (−0.32:0.20)	0.05 (0.05:0.20)	1.78 (1.46:2.17)	0.39

Table 3. (*cont.*)

45	*C. laricella*	2.06	2.51	4.18	−1.97 (−4.24:−0.08)	1.51 (0.34:8.38)	0.16 (0.07:0.33)	0.27
46	*C. laricella*	1.02	1.05	0.07	−0.43 (−0.93:0.08)	0.05 (0.05:0.14)	1.79 (1.51:2.13)	0.11
47	*C. laricella*	1.01	1.05	0.06	0.12 (−0.67:0.90)	0.05 (0.05:1.00)	2.50 (1.40:4.53)	0.40
48	*C. laricella*	1.04	1.05	0.10	0.26 (−0.59:1.05)	0.05 (0.05:0.40)	0.04 (0.02:0.072)	0.66
49	*C. laricella*	1.85	1.33	1.47	1.30 (0.55:2.19)	0.33 (0.05:1.00)	0.13 (0.07:0.22)	0.50
50	*C. laricella*	2.09	1.38	1.88	0.44 (−0.57:1.57)	0.38 (0.15:1.29)	0.37 (0.25:0.56)	5.55
51	*C. laricella*	1.08	1.05	0.13	0.34 (−0.04:0.71)	0.05 (0.05:0.14)	0.79 (0.68:0.92)	0.69
52	*C. laricella*	1.00	1.05	0.05	−0.04 (−0.41:0.33)	0.05 (0.05:0.09)	2.22 (1.87:2.65)	0.15
53	*Aonidiella aurantii* (Diaspidae)	1.02	1.16	0.19	0.16 (−0.02:0.35)	0.16 (0.07:0.32)	0.53 (0.44:0.64)	0.87
54*	*A. aurantii*	1.00	3.00	2.00	−0.03 (−0.70:0.45)	2.00 (1.46:2.70)	0.30 (0.17:0.58)	0.48
55	*A. aurantii*	1.01	1.05	0.06	0.24 (−0.42:0.90)	0.05 (0.05:0.18)	0.57 (0.46:0.71)	0.10
56	*A. aurantii*	1.00	1.06	0.06	−0.04 (−0.77:0.73)	0.06 (0.05:0.37)	0.24 (0.18:0.32)	0.13
57*	*Eupteryx cyclops* *E. urticae* (Cicadellidae)	1.21	1.05	0.27	0.93 (0.39:1.50)	0.05 (0.05:0.19)	0.48 (0.35:0.64)	0.24
58*	*Polistes exclamans* (Vespidae)	1.00	2.72	1.73	0.08 (−0.40:−0.25)	1.72 (0.95:3.58)	0.24 (0.17:0.32)	0.46
59	*P. exclamans*	1.08	8.14	7.77	−0.46 (−0.87:−0.33)	7.14 (2.34:10.0)	0.03 (0.03:0.07)	0.36
60*	*Icerya purchasi* (Margarodidae)	1.00	1.05	0.05	0.01 (−0.22:0.25)	0.05 (0.05:0.18)	1.11 (0.92:1.33)	0.31
61	*I. purchasi*	1.01	1.05	0.06	0.05 (−0.23:0.34)	0.05 (0.05:0.20)	0.74 (0.57:0.94)	0.57
62	*I. purchasi*	1.03	1.30	0.34	−0.25 (−0.75:0.26)	0.30 (0.11:0.81)	1.42 (1.01:2.03)	0.48
63	*I. purchasi*	1.00	1.05	0.05	−0.01 (−0.16:0.14)	0.05 (0.05:0.19)	1.11 (0.92:1.34)	0.79
64*	*Trypargilum politum* (Sphecidae)	1.02	2.05	1.09	0.18 (−0.43:0.89)	1.05 (0.13:6.58)	0.08 (0.04:0.12)	0.56
65	*Lymantria dispar* (Lymantriidae)	1.07	1.09	0.17	−0.53 (−0.63:−0.38)	0.09 (0.07:0.13)	0.42 (0.39:0.43)	0.24

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* Reduced list with no spatial or temporal replicates.

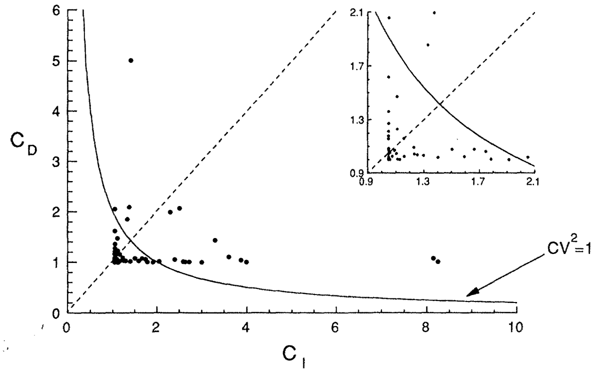

Figure 4. Host density dependent heterogeneity (C_D) plotted against host density independent heterogeneity (C_I). The points correspond to the 65 examples in tables 2 and 3. The solid curve is the contour where $CV^2 = 1$. The dashed line separates points for which $C_I > C_D$ from those for which $C_D > C_I$. The inset magnifies the cluster of points nearest to the origin. See text for further details. (After Pacala *et al.* 1990.)

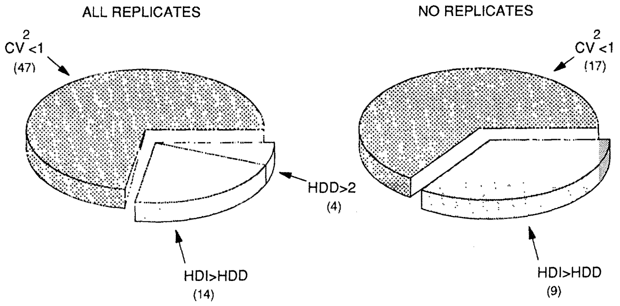

Figure 5. Pie diagrams distinguishing between examples in table 3 where $CV^2 < 1$ from those $CV^2 > 1$. (Left): all 65 data sets; (right) only the 26 data sets marked by asterisks which include no spatial or temporal replicates. Where $CV^2 > 1$, the shaded segment corresponds to host density independent heterogeneity predominating and the unshaded segment to where host density dependent heterogeneity is the more important.

sufficient to stabilize the populations. Interestingly, in 14 of these 18 cases $C_I > C_D$ and, furthermore, in each case $C_I > 2$, indicating that the level of heterogeneity in C_I alone is sufficient to make $CV^2 > 1$. In the remaining 47 examples in figure 4 where $CV^2 < 1$, the majority (see inset) CV^2 show heterogeneity having no appreciable affect on population regulation. In eight cases, however, $CV^2 > 0.6$, at which level heterogeneity could be promoting stability to some degree. Figure 5 further summarizes these results and also shows that the pseudoreplication does not appear to be biasing the different categories since the same qualitative picture emerges from the reduced data set of 26 different species pairs.

Thus, contrary to the popular view, this analysis suggests that density independent spatial patterns of parasitism (e.g. figure 1*c*) may be more important in promoting population regulation than density dependent patterns. In the six cases where HDD is important (i.e. C_D contributes substantially to the $CV^2 > 1$), the density dependence is inverse in only one case (data set 55; $\mu = -1.97$). This ratio is smaller than expected from the overall frequencies of significant direct and inverse density dependent patterns summarised in figure 6.

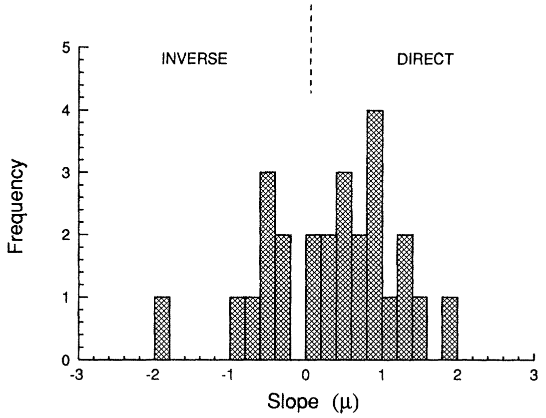

Figure 6. Frequency distribution of estimated values of μ from table 3 which are significantly different from $\mu = 0$ ($p < 0.05$).

CONCLUSIONS

Although, the discrete-time models discussed in this paper show that the '$CV^2 > 1$ rule' may apply across a wide variety of host–parasitoid interactions, several fundamental assumptions about the biology of the interactions have been made which do not apply to all host–parasitoid systems.

(i) The assumption of coupled, synchronized interactions restricts our analysis to parasitoids that are effectively specialists on the one host species. The dynamics of generalist parasitoids and their hosts can be very different (Hassell & May 1986; Latto & Hassell 1988) and will require a separate treatment.

(ii) Having discrete host and parasitoid generations does not permit appreciable overlapping of host and parasitoid generations which would be better represented in continuous time. Whether or not comparable stability criteria exist for the affects of heterogeneity in such continuous interactions has yet to be determined.

(iii) The extent to which the parasitoids encounter hosts at random within patches, as assumed in our models, and the importance of any deviations from this, will depend in part on the size of the patches relative to the foraging area of the parasitoids. This introduces important questions on the scales at which HDD and HDI heterogeneity exert their main effects. For example, any covariance between parasitoid distribution and local host densities per patch is likely to be scale-dependent, since it depends critically on what a foraging parasitoid recognizes as a patch (Waage 1979). Likewise, HDI heterogeneity depends on any differences in the attractiveness of patches independent of the host density that they contain, as well as any Poisson and other 'errors' in the parasitoids' decision making. Both of these are likely to be strongly influenced by the scale of patchiness being examined.

(iv) By neglecting interference between parasitoids or competition among hosts, the models in this paper focus on situations where interactions between host and parasitoid populations are of predominant im-

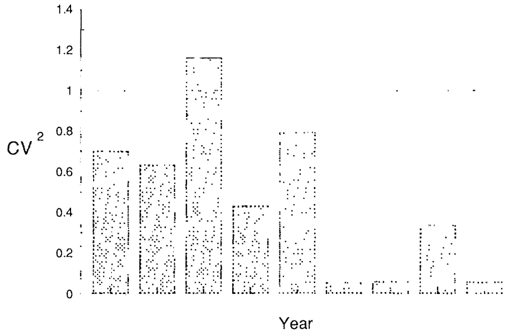

Figure 7. Values of CV^2 from data sets 23 to 31 in table 3. These are annual replicates between 1980 and 1989. (From T. H. Jones & M. P. Hassell, unpublished data.)

portance for the overall dynamics. Any additional density-dependent and density-independent factors that markedly influence fecundity or survival of the hosts and parasitoids will also affect dynamics and change the overall stability conditions. In neglecting these, our emphasis is primarily on understanding the extent to which one factor, the distribution of parasitism amongst hosts, can contribute to population regulation.

(v) Many of the examples in tables 2 and 3 come from single-generation studies with no temporal replication. We can thus only predict effects on dynamics assuming that the estimated values of CV^2 are typical for each interaction. In fact, those studies with some temporal replication show considerable variation in CV^2 from generation to generation, as shown by the example in figure 7. Procedures are now needed for evaluating how fluctuating CV^2 values affect population dynamics.

An often contentious issue in ecology has concerned the relevance of general models in understanding the dynamics of natural systems in the field. This study shows how relatively simple models of host–parasitoid systems can profitably be applied to field data on levels of parasitism in a patchy environment. Such heterogeneity has often been regarded as a complicating factor in population dynamics, and one that rapidly leads to analytical intractability. Clearly, this need not necessarily be so. The $CV^2 > 1$ rule explains the consequences of heterogeneity for population dynamics in terms of a simple description of the heterogeneity itself. The rule gives a rough prediction of the effects of heterogeneity and also identifies the kinds of heterogeneity that contribute to population regulation.

APPENDIX

In this appendix we derive simplified expressions for the stability conditions (2*a*) and (2*b*). Let

$$f(N_t, P_t) = E[n\,\mathrm{e}^{-P_t g(n)\epsilon}], \qquad \text{(A 1)}$$

where E ( ) stands for expectation (average across all patches), n is the relative abundance of hosts in a patch (local abundance divided by mean abundance), $g(n)$ describes the tendency of local parasitoid abundance to change with n, and ϵ is a unit-mean random variable that governs spatial variance in local parasitoid abundance which is unrelated to local host density. Also, the term $\mathrm{e}^{-P_t g(n)\epsilon}$ is the probability of surviving in a patch if the parasitoids in the patch search randomly (see the explanation of model I in the text). In a pure HDD model, ϵ is a constant and equal to one, and in a pure HDI model, $g(n)$ is constant. Hassell *et al.* (1990) review the many previous studies that employ special cases of (A 1).

Further, let $\bar{g} = E(g(n))$ and $\bar{g}' = E(ng(n))$, and observe that:

$$-\partial f(N^*, P^*)/\partial P_t = E(ng(n)\,\epsilon\,\mathrm{e}^{-P^* g(n)\epsilon}). \qquad \text{(A 2)}$$

The covariance between local parasitoid and unparasitized host density at the end of a growing season is:

$$\begin{aligned}\begin{bmatrix}\text{end-of-season}\\ \text{covariance}\end{bmatrix} &= E\left[\begin{pmatrix}\text{parasitoids}\\ \text{in patch } i\end{pmatrix}\begin{pmatrix}\text{surviving}\\ \text{hosts in patch } i\end{pmatrix}\right]\\ &\quad - E\begin{bmatrix}\text{parasitoids}\\ \text{in patch } i\end{bmatrix} E\begin{bmatrix}\text{surviving}\\ \text{hosts in patch } i\end{bmatrix},\\ &= E[(P^* g(n_i)\,\epsilon)(N^* n_i\,\mathrm{e}^{-P^* g(n)\epsilon})]\\ &\quad - E[P^* g(n_i)\,\epsilon]E[N^* n_i\,\mathrm{e}^{-P^* g(n)\epsilon}],\\ &= P^* N^* \{E[n_i\,g(n_i)\,\epsilon\,\mathrm{e}^{-P^* g(n)\epsilon}]\\ &\quad - E[g(n_i)\,\epsilon]\,E[n_i\,\mathrm{e}^{-P^* g(n)\epsilon}]\}.\end{aligned}$$

Of the three expectations in the above equation, the first is given by (A 2), the second is $\bar{g}$ and the third is given by (A 1). Thus

$$[\text{end-of-season covariance}] = P^* N^* [\partial f(N^*, P^*)/\partial P_t - \bar{g} f(N^*, P^*)].$$

At equilibrium, the mean parasitoid abundance per patch is simply $P^*\bar{g}$, the mean end-of-season abundance of unparasitized hosts is $f(N^*, P^*)\,N^*$, and $f(N^*, P^*) = 1/\lambda$. Thus, C_e, the end-of-season covariance divided by the product of mean parasitoid and unparasitized host abundance $(N^* P^* \bar{g}/\lambda)$ is:

$$C_e = [-\lambda/\bar{g}\cdot\partial f(N^*, P^*)/\partial P_t] - 1. \qquad \text{(A 3)}$$

Similarly, the equilibrium covariance at the beginning of the season is:

$$\begin{aligned}[\text{beginning-of-season covariance}] &= E(N^* n P^* g(n)\,\epsilon) - N^* P^* \bar{g},\\ &= N^* P^* [E(ng(n)\,\epsilon) - \bar{g}],\end{aligned}$$

and so the scaled covariance C_b is:

$$C_b = \bar{g}'/\bar{g} - 1. \qquad \text{(A 4)}$$

By using (A 3) and (A 4), we may write condition (2*a*) as:

$$C_e + 1/C_b + 1 < \lambda - 1/\lambda P^* \bar{g}' = Z(\lambda). \qquad \text{(A 5)}$$

Now, consider the equilibrium equation from (1*a*):

$$1/\lambda = E[n\,\mathrm{e}^{-P^* g(n)\epsilon}]. \qquad \text{(A 6)}$$

It is straightforward to show from (A 6) that P^* increases monotonically from zero to infinity as λ increases from one to infinity. As $\lambda \to 1$, (A 6) approaches:

$$1/\lambda = E(n(1 - P^* g(n)\,\epsilon)),$$

which reduces to $1/\lambda = 1 - P^* \bar{g}'$.

Thus, as $\lambda \rightarrow 1$, condition (A 5) approaches:

$$C_e + 1 < 1/\lambda(C_b + 1),$$

and as $\lambda \rightarrow \infty$, (A 5) approaches:

$$C_e < -1.$$

Turning now to condition (2*b*), we first observe from the equilibrium equation from (1*b*) that:

$$P^* = N^*(1 - 1/\lambda).$$

Thus (2*b*) may be written as:

$$P^*(-\partial f(N^*, P^*)/\partial P_t) > N^* \cdot \partial f(N^*, P^*)/\partial N_t.$$

If $P_t = e^X$ and $N_t = e^Y$, then

$$\partial f(N^*, P^*)/\partial X = P^* \, \partial f(N^*, P^*)/\partial P_t,$$

and

$$\partial f(N^*, P^*)/\partial Y = N^* \, \partial f(N^*, P^*)/\partial N_t,$$

and so condition (2*b*) may be written:

$$-\partial f(N^*, P^*)/\partial X > \partial f(N^*, P^*)/\partial Y.$$

Because $\partial f/\partial X < 0$ (host survival decreases as parasitoid density increases) this condition is always satisfied unless $\partial f/\partial Y > 0$ (an increase in host abundance increases host survival). Although $\partial f/\partial Y$ may indeed be positive in some cases involving parasitoid satiation, in the majority of biologically plausible models there is either no density dependence affecting the host ($\partial f/\partial Y = 0$) or ($\partial f/\partial Y < 0$). As a result, condition (2*a*) is usually necessary and sufficient for stability.

In summary, therefore, condition (2*a*) is equivalent to:

$$C_e + 1 < (C_b + 1)\, Z(\lambda),$$

and condition (2*b*) is equivalent to:

$$-\partial f(N^*, P^*)/\partial X > \partial f(N^*, P^*)/\partial Y.$$

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Discussion

G. A. Tingley (*Imperial College of Science, London, U.K.*). We have seen that patch size is of importance in the observed spatial pattern. What is the likely sensitivity of this type of analysis to the selection of inappropriate patch size in field observations?

M. P. Hassell. As our estimates of CV^2 are weighted for host density per patch, part of the problem of spatial scale is avoided: heterogeneity is measured at the level of the individual hosts. However, scale remains important in a number of ways. First, our assumption that the exploitation of hosts within a patch is random is satisfactory for relatively small patch sizes, but could introduce significant error as patch size gets large. Second, is the very interesting problem of identifying the scales at which host density dependent and host density independent heterogeneity have their maximal effect. These need not necessarily occur at the same scale.