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Inferring Host-Parasitoid Stability from Patterns of Parasitism among Patches

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A central result of host-parasitoid theory is that host-parasitoid dynamics in simple models can be stabilized by aggregation of risk among hosts (Chesson and Murdoch 1986). Aggregation of risk describes the degree of variability in the distribution of attack probabilities over all hosts in the population; the more variable this distribution is, the more aggregated risk is said to be. For example, if all hosts have an equal probability of being parasitized, risk is equally spread among hosts, but if some hosts have a greater chance of being parasitized, risk of parasitism is aggregated in a subset of the population. When the distribution of risk is sufficiently aggregated, this aggregation stabilizes host-parasitoid dynamics through pseudointerference among foraging parasitoids (Free et al. 1977; Walde and Murdoch 1988). Pseudointerference decreases the number of hosts killed per parasitoid as parasitoid densities rise above equilibrium, thereby squelching the onset of unstable oscillations that ultimately result in the extinction of the parasitoid.

Aggregation of risk is often envisioned as arising from differences in risk among patches of hosts. An extensive body of theoretical work has shown that variability in risk across host patches can be stabilizing when risk is concentrated in patches with many hosts (host density-dependent aggregation; Hassell and May 1973), in patches with few hosts (inverse density-dependent aggregation; Hassell 1984), or in a particular subset of patches regardless of host distribution (density-independent aggregation; Chesson and Murdoch 1986). Variability in risk among patches is not the only pathway to a variable distribution of risk among hosts (Bernstein 1987; Taylor 1993). Differential susceptibility to attack among hosts, due to variability among hosts' defense capabilities or hosts' abilities to avoid detection, can also give rise to stabilizing aggregation of risk (Bailey et al. 1962), as can partial temporal asynchrony between parasitoids' periods of foraging and hosts' windows of susceptibility (Munster-Swendsen and Nachman 1978).

In order to explore the prevalence of aggregation of risk as a stabilizing phenomenon in natural host-parasitoid systems, ecologists have attempted to devise a method to infer from field data both the distribution of risk among hosts and whether the aggregation of risk is sufficient to generate stability (Reeve and Murdoch 1985; Hassell and May 1988). The most recent attempt to use the distribution of parasitism among patches to assess variability in host risk is the $CV^2 > 1$ rule (Pacala et al. 1990; Hassell et al. 1991), which posits that the aggregation of risk is roughly sufficient for stability if the square of the coefficient of variation of the density of searching parasitoids per host exceeds unity. While the $CV^2 > 1$ rule has been shown to hold in theory for an impressive array of discrete-time models (Hassell et al. 1991), its practical utility hinges on the ability to infer the distribution of risk among hosts from field data.

In practice, it is difficult to observe the distribution of risk among single hosts because typically it is only possible to determine whether a host is parasitized or not. When hosts are patchily distributed, a common strategy (employed with the $CV^2 > 1$ rule) for inferring distribution of risk is the use of the proportion of hosts parasitized in a patch as a measure of the risk experienced by all hosts in that patch, assuming that all members of the patch share an identical risk (Reeve and Murdoch 1985; Pacala and Hassell 1991). A statistical algorithm is then used to decompose the variability in parasitism among patches into variability explained by host density (density-dependent aggregation) and unexplained variability (density-independent aggregation). These two descriptions of aggregation are used in conjunction with the distribution of hosts among patches to calculate the distribution of risk among hosts (Pacala and Hassell 1991). Thus, estimating the distribution of risk among

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hosts in this way assumes that the variability in the distribution of parasitism among patches reflects the variability in the distribution of risk among hosts.

A difficulty arises, however, when parasitoids attack multiple hosts within patches. In this case, variability in parasitism among patches will exceed the variability in risk among hosts without altering the stability of the interaction. Although this was recognized by Hassell et al. (1991, p. 576), it is typically ignored when analyzing data. In this note, we examine in more detail how parasitoid foraging patterns alter the correspondence between the distribution of parasitism among patches and the underlying distribution of risk among hosts. In doing so, we highlight the need to understand the mechanism generating variability in parasitism among patches before mapping this variability to variability in risk among hosts.

Foraging parasitoids generate variability in parasitism among patches by two processes, which we will refer to as among-parasitoid and within-parasitoid aggregation of attacks. Among-parasitoid aggregation describes the aggregation of parasitoid visits among patches, which produces variability in parasitism among patches in an obvious way: when parasitoids concentrate their foraging bouts in a subset of patches, these patches will show higher incidences of parasitism. Within-parasitoid aggregation describes the delivery of multiple attacks by an individual parasitoid during a single visit to a patch. This aggregation also inflates variability in parasitism among patches because many hosts are parasitized when a parasitoid visits a patch. The distinction between these two sources of variability is critical because, even though both among- and within-parasitoid aggregation affect the variability in parasitism among patches, only among-parasitoid aggregation increases the aggregation of risk among hosts. Therefore, only among-parasitoid aggregation can stabilize population dynamics (Hassell et al. 1991). Conversely, within-parasitoid aggregation increases the variability in parasitism among patches without influencing stability. As a consequence, calculating the distribution of risk among hosts using the distribution of parasitism among patches while ignoring the possibility of multiple attacks within patches can lead to incorrect estimates of the variability in risk among hosts and erroneous conclusions about the role of aggregation of risk in stabilizing the system.

We illustrate this pitfall here using a simple model. We employ a Nicholson-Bailey multipatch model with a negative binomial distribution of parasitoid visits among patches and a random distribution of attacks per host per parasitoid visit (cf. May 1978). By modulating the clumping parameter of the negative binomial distribution and the mean number of attacks delivered per host per patch visit, we can control the degree of among- and within-parasitoid aggregation of attacks, respectively. A standard stability analysis shows that the model's stability depends on the distribution of parasitoid visits among patches (among-parasitoid aggregation) but not on the number of hosts attacked per visit (within-parasitoid aggregation). We then generate hypothetical data sets from model host-parasitoid systems to show how varying the magnitude of among- and within-parasitoid aggregation of attacks can cause systems with dissimilar stability properties to exhibit qualitatively similar patterns of parasitism among patches. Next, we explain in detail how within-parasitoid aggregation of attacks increases the variability in parasitism among patches without altering the stability of the host-parasitoid dynamics. Finally, we use a more general version of our model to demonstrate that any observed pattern of variability among patches can be explained by either among- or within-parasitoid aggregation of attacks. We use our illustration as a caution against inferring the distribution of risk among hosts (and consequently the stability of host-parasitoid systems) from the distribution of parasitism among patches.

The Model

We begin with the standard Nicholson-Bailey host-parasitoid model (Hassell 1978),

$$\begin{aligned} N_{t+1} &= \lambda N_t F(N_t, P_t), \\ P_{t+1} &= cN_t[1 - F(N_t, P_t)], \end{aligned} \tag{1}$$

where N_t and P_t are the average per patch host and parasitoid densities, respectively, at time t; λ is the host's population multiplication rate; $F(N_t, P_t)$ is the fraction of hosts escaping parasitism; and c is the average number of parasitoid offspring produced per parasitized host, which we will assume equals 1. The equilibrium of this model is given by $1 = \lambda F(N^*, P^*)$ and $P^* = N^*(1 - \lambda^{-1})$ (Hassell et al. 1991), and the stability criteria are (Hassell 1978)

$$N^* \frac{\partial F(N^*, P^*)}{\partial N_t} < -P^* \frac{\partial F(N^*, P^*)}{\partial P_t} < \frac{\lambda - 1}{\lambda^2}. \tag{2}$$

We assume that the per host risk of attack is a linear function of local parasitoid density, that is, a Type I functional response, and so the fraction of hosts escaping parasitism is independent of host density, that is, $F(N_t, P_t) = F(P_t)$. Thus, $\partial F(P_t)/\partial N_t = 0$, and the left-hand inequality of (2) is satisfied as long as the equilibrium parasitoid density is positive and the escaping host fraction decreases as parasitoid density grows. Therefore, the stability of the system is governed by the right-hand inequality of (2).

We model the distribution of parasitoid visits among

patches, V, as a negative binomial random variable with mean wP_t, where w is the mean number of patch visits made by an individual parasitoid, and clumping parameter k_V (May 1978), which controls the degree of among-parasitoid aggregation. Parasitoid visits become more aggregated as k_V becomes smaller; as k_V goes to infinity, parasitoid visits become randomly (Poisson) distributed. We assume that parasitoid visits are distributed independently of hosts, and so V is also the distribution of foraging parasitoids per host, the distribution that the $\mathrm{CV}^2 > 1$ rule seeks to ascertain. We also assume that during a single patch visit, the number of attacks delivered by a parasitoid to each host, A, is a Poisson random variable with mean α. Equivalently, we can say that the total number of attacks delivered during a visit to a patch with n hosts is a Poisson random variable with mean αn, and that these attacks are then distributed randomly among the hosts in the patch. Because α determines the number of hosts a parasitoid attacks in each patch visit, it also governs the magnitude of within-parasitoid aggregation.

To derive $F(P_t)$, the fraction of hosts escaping parasitism, we calculate the distribution of attacks across all hosts, Y. Compounding the generating functions for the distributions of parasitoid visits among patches, $G_V(z)$, and attacks per host per parasitoid visit, $G_A(z)$, gives the generating function for Y, $G_Y(z)$ (Feller 1968; Chesson and Murdoch 1986),

$$G_Y(z) = G_V(G_A(z)) = G_V(e^{\alpha(z-1)})$$

$$= \left\{1 + \frac{wP_t}{k_V}[1 - e^{\alpha(z-1)}]\right\}^{-k_V}. \qquad (3)$$

The fraction of hosts escaping parasitism is given by $G_Y(0) = [1 + (wP_t/k_V)(1 - e^{-\alpha})]^{-k_V}$.

Calculating P^* and $\partial F/\partial P_t$ and plugging these into the right-hand inequality of (2) gives

$$-P^* \frac{\partial F(N^*, P^*)}{\partial P_t} = k_V\left[\lambda^{-1} - \lambda^{-(k_V+1)/k_V}\right] < \frac{\lambda - 1}{\lambda^2}.$$

A little algebra shows that inequality (4) is identical to the well-known stability criterion of May's (1978) negative-binomial model, which is stable when $k_V < 1$. (May's model can also be recovered by substituting the transformations $\tilde{P}_t = wP_t[1 - e^{-\alpha}]$ and $\tilde{c} = wc[1 - e^{-\alpha}]$ into [1].) Thus, stability depends only on the distribution of parasitoid visits among patches through k_V and is independent of both the mean number of visits per parasitoid, w, and the mean number of attacks per visit, α.

To illustrate how both among- and within-parasitoid aggregation of attacks affect variability in parasitism rates among patches, we designed three hypothetical host-parasitoid systems. In system 1, parasitoid visits are nearly randomly distributed among patches ($k_{V_1} = 100$), and thus the equilibrium is unstable. The mean number of attacks per host per patch visit, α_1, equals 0.04. In system 2, attacks per visit are increased tenfold ($\alpha_2 = 0.4$), driving an increase in within-parasitoid aggregation of attacks. However, the degree of among-parasitoid aggregation is the same as in system 1 ($k_{V_2} = 100$), and consequently the dynamics remain unstable. System 3 possesses a stable equilibrium due to an aggregated distribution of parasitoid visits ($k_{V_3} = 0.9$), but the mean number of attacks per patch visit is the same as in system 1 ($\alpha_3 = 0.04$). In all systems, the host's population growth rate, λ, is set to 2, so that at equilibrium one-half of all hosts escape parasitism (i.e., $F[P^*] = 0.5$). Equilibrium densities are held constant across systems by modulating the mean number of patch visits per parasitoid, w.

We simulated hypothetical data sets of percentage of hosts parasitized versus patch size for all three systems, with the number of hosts per patch, n, ranging from 1 to 100 (fig. 1). For each patch, we first simulated the number

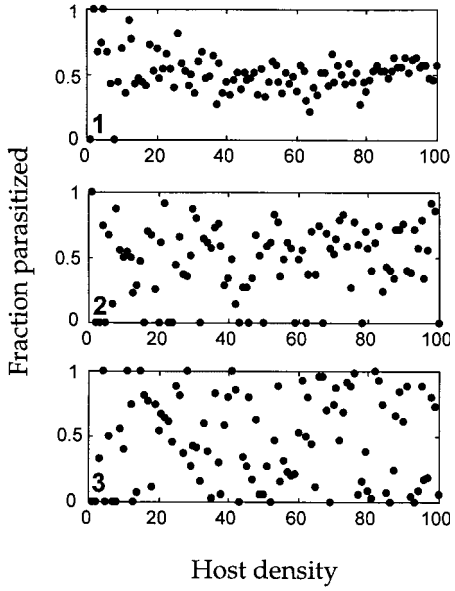

Figure 1: Hypothetical data from three model systems. Each data set consists of 100 patches, with host densities ranging from 1 to 100 hosts per patch. The equilibriums for systems 1 and 2 are equally unstable, while system 3's equilibrium is stable. The increased variability in system 2 relative to system 1 is driven by an increased number of attacks per parasitoid visit ($\alpha_2 = 0.4$ vs. $\alpha_1 = \alpha_3 = 0.04$), while the increase in variability in 3 relative to 1 is driven by density-independent aggregation of parasitoids ($k_{V_3} = 0.9$ vs. $k_{V_1} = k_{V_2} = 100$). The mean number of visits per parasitoid are $w_1 = 1.77$, $w_2 = 0.21$, and $w_3 = 2.66$. All systems have equilibrium densities $N^* = 20$ and $P^* = 10$.

of parasitoid visits, v, by a pseudorandom draw from the distribution of V. The total number of attacks administered by visiting parasitoids was then simulated by an independent realization of a Poisson random variable with mean αnv. Finally, each attack was randomly assigned to one of the hosts in the patch.

Because we have assumed a Type I functional response, the expected percentage of parasitism for all patches in all systems is equal to $1 - F[P^*]$, or 50%. However, the variability in our simulated data about the expected percentage of parasitism is greater for systems 2 and 3 (fig. 1). If we had used Pacala and Hassell's (1991) strategy to analyze these data for aggregation of risk, the variability in systems 2 and 3 would have been interpreted as density-independent aggregation of risk contributing to the stability of the host-parasitoid interaction. However, parasitoid visits are only aggregated among patches in system 3; in system 2 (as well as in system 1), parasitoid visits are nearly randomly distributed among patches. Because the stability of these systems depends only on among-parasitoid aggregation, the stability properties of systems 1 and 2 are identical, despite the inflated variability in parasitism rates observed in system 2. Only system 3 is stable.

Why Does Increasing Within-Parasitoid Aggregation Inflate Variability in Parasitism among Patches?

The variability in parasitism among patches is composed of both variability in individual hosts' risk of parasitism and covariances in risk among hosts sharing a patch. We can illustrate this relationship by writing the fraction of hosts parasitized in a patch as an average of indicator variables. For a patch with n hosts, we define the indicator variables I_j ($j = 1, \ldots, n$), which take the value 1 if host j is parasitized, and 0 if host j escapes parasitism. The fraction of hosts parasitized per patch is the random variable $\bar{I}_\cdot = (1/n)\Sigma_{j=1}^{n} I_j$. The variance of parasitism among patches with n hosts is thus

$$\begin{aligned} \mathrm{V}[\bar{I}_\cdot] &= n^{-2}\left(\sum_{j=1}^{n} \mathrm{V}[I_j] + 2\sum_{j=2}^{n}\sum_{k=1}^{j-1} \mathrm{Cov}[I_j, I_k]\right) \\ &= n^{-2}\left(\sum_{j=1}^{n} E[I_j](1 - E[I_j]) + 2\sum_{j=2}^{n}\sum_{k=1}^{j-1} \mathrm{Cov}[I_j, I_k]\right). \end{aligned} \quad (5)$$

When mean parasitism, $E[I_j]$, remains constant, the variance in parasitism among patches is driven solely by the covariances in risk of attack among hosts sharing the same patch. As the average number of attacks delivered by a parasitoid during a patch visit increases, the chance that any one host in a patch is parasitized becomes more tightly coupled with the chance that any other host in the same patch is also parasitized. Thus, whenever a parasitoid has an opportunity to parasitize multiple hosts during a single patch visit, within-parasitoid aggregation of attacks increases the covariances in risk among hosts sharing a patch. Equation (5) demonstrates that these covariances among hosts in the same patch lead to increased variance in parasitism among patches. This result is independent of the distribution of parasitoid visits among patches and therefore holds for any model of foraging parasitoids.

Why Doesn't Variability Generated by Within-Parasitoid Aggregation of Attacks Impart Stability?

The difference between variability generated by among- and within-parasitoid aggregation can be understood in terms of pseudointerference among parasitoids (Free et al. 1977). Pseudointerference stabilizes population dynamics by concentrating parasitoid foraging among a subset of the host population, thus accelerating competition among parasitoids for unparasitized hosts as parasitoid densities increase (Walde and Murdoch 1988; Taylor 1993). This competition forces attacks to be wasted on previously parasitized hosts, decreasing the parasitoid's foraging efficiency as parasitoids become more numerous. This inefficiency suppresses parasitoid recruitment into the next generation and allows a portion of the host population to escape parasitism. In contrast, when parasitoids are distributed randomly among patches, all hosts are equally accessible, and so competition among parasitoids for new hosts is less fierce. Thus, fewer attacks are wasted on previously parasitized hosts, and foraging efficiency does not decline as rapidly as parasitoid densities rise.

The delivery of many attacks during a patch visit also compromises foraging efficiency when a parasitoid attacks a single host more than once. However, for a given patch size, the number of attacks a parasitoid wastes on hosts that it has already attacked during the same visit is independent of parasitoid density. Because stabilizing mechanisms must act in a density-dependent fashion, foraging inefficiency caused by within-parasitoid aggregation of attacks cannot affect the stability of the host-parasitoid interaction.

The difference between these two types of aggregation can be conceptualized with a thought experiment. Consider two host-parasitoid systems, the first with only among-parasitoid aggregation of attacks, and the second with only within-parasitoid aggregation of attacks. Both systems consist of 100 patches, and each patch contains 100 hosts. Initially, the first parasitoid population is composed of 100 parasitoids inhabiting the same patch, and each parasitoid delivers one attack (at random) to the hosts

in that patch (i.e., attacks per parasitoid are a fixed quantity and not a random variable). In the second population, where parasitoids are distributed randomly among patches, the initial parasitoid population consists of one adult parasitoid that delivers 100 attacks randomly among the hosts in its patch. Thus, each system has one patch in which 100 attacks occur, resulting in the same pattern of parasitism across patches for both systems.

Now, additional parasitoids are added to each population. To the first population, we add 100 parasitoids. Because these parasitoids are perfectly aggregated among patches, all new parasitoids forage in the patch already inhabited by the initial parasitoid cohort. The expected increase in the number of parasitized hosts is the difference between the zero term of a Poisson distribution with a mean of two attacks per host and the zero term of a Poisson distribution with a mean of one attack per host multiplied by 100, or $100\,(e^{-1} - e^{-2}) \approx 23$. Conversely, when we add 100 attacks, or one parasitoid, to the second population, the new parasitoid has only a 1% chance of inhabiting the patch that is already occupied. Thus, there is a 1% chance that the parasitoid will attack 23 unparasitized hosts and a 99% chance that 63 new hosts will be parasitized ($100[1 - e^{-1}] \approx 63$). On average, the new parasitoid will attack about 63 new hosts. The difference in additional foraging success between these two populations reflects how pseudointerference reduces the parasitoids' success rate when parasitoid visits are aggregated among patches.

How Much Variability Can Be Generated by Within-Parasitoid Aggregation of Attacks?

Recall that implementation of the $CV^2 > 1$ rule uses the distribution of parasitism among patches to infer the variability in risk among hosts, and hence stability (Pacala and Hassell 1991). This link is made by estimating the CV^2 of the mean number of attacks suffered per host and then equating this measure with the CV^2 of risk among hosts. According to the assumptions of the CV^2 calculation, attacks are distributed randomly within a patch. For a fixed-patch size, therefore, the CV^2 of mean number of attacks per host is equivalent to the CV^2 of total attacks per patch. Thus, to find a measure analogous to CV^2 in our model, we can use generating functions to compute the CV^2 of the total number of attacks incurred in a patch with n hosts, U_n. When we compound the generating function of the distribution of visits among patches with the generating function of the distribution of total attacks in a patch with n hosts (a Poisson random variable with mean αn), we find that the CV^2 of U_n is

$$\mathrm{CV}^2_{U_n} = \frac{1}{\alpha n E[V]} + \frac{1}{E[V]} + \frac{1}{k_V}, \tag{6}$$

where $E[V]$ is the mean number of visits per patch. When the number of hosts in a patch, n, becomes moderately large, the term $1/\alpha n E[V]$ becomes negligibly small, leaving two terms that describe how within- and among-parasitoid aggregation combine to produce variability in parasitism among patches. The term $1/E[V]$ quantifies within-parasitoid aggregation of attacks because, for a given level of parasitism, the number of attacks per visit, α, must be inversely related to the mean number of visits per patch. The term $1/k_V$ quantifies among-parasitoid aggregation of attacks because, as $1/k_V$ grows large, k_V becomes small and hence visits become increasingly variable among patches. Note that when within-parasitoid aggregation approaches 0 (e.g., $1/E[V]$ becomes negligibly small), the stability criterion, $k_V < 1$, holds if and only if $\mathrm{CV}^2_{U_n} > 1$. Yet as equation (6) illustrates, when we observe a value of CV^2 from field data, it is impossible to know how much of this variability is attributable to stabilizing among-parasitoid aggregation and how much is due to within-parasitoid aggregation. Equation (11) in Hassell et al. (1991) gives a similar conclusion from a different derivation.

To this point, we have considered the total number of attacks delivered per patch visit to be a Poisson random variable. We now relax this assumption and allow parasitoids to deliver a negative binomial number of attacks per patch visit, with mean αn and clumping parameter k_A. Attacks are still distributed randomly among hosts in the patch, thus the number of attacks suffered per host per patch visit is a negative binomial with mean α and clumping parameter k_A. This model is a generalization of our first model, in which $k_A \to \infty$. Therefore, the stability criterion remains unchanged—the model is stable only when $k_V < 1$. However, another exercise with generating functions shows that the CV^2 of total attacks per patch is now

$$\mathrm{CV}^2_{U_n} = \frac{1}{\alpha n E[V]} + \frac{1}{E[V]} + \frac{1}{k_A E[V]} + \frac{1}{k_V}, \tag{7}$$

with the first term on the right again becoming negligible when host densities are moderately large. Thus, variability in the total number of attacks delivered per parasitoid visit contributes to variability in parasitism among patches through the term $1/k_A E[V]$ without affecting the stability of the system. This further degrades the correspondence between the observed CV^2 and stability. Indeed, by eliminating among-parasitoid aggregation (allowing $k_V \to \infty$), we can still explain observed levels of variability in parasitism among patches completely by within-parasitoid ag-

gregation. We illustrate this in figure 2*A*, which shows how variability in attacks per parasitoid patch visit produces large values of CV^2, even when stabilizing among-parasitoid aggregation is absent. Indeed, even without among-parasitoid aggregation, modest within-parasitoid aggregation will produce levels of variability in parasitism among patches sufficient to satisfy the $CV^2 > 1$ rule (fig. 2*B*).

As a final illustration, we consider a data set in Pacala and Hassell (1991) with one of the largest values of CV^2—the data set attributed to Ehler's (1986) study of the midge *Rhopalomyia californica* Felt and its endoparasite *Tetrastichus* sp. The data (fig. 3*A*, reproduced from figure 1*D* of Pacala and Hassell [1991]) exhibit extreme variability in parasitism among patches, with a faint hint of inversely density-dependent aggregation. Pacala and Hassell (1991) calculated a CV^2 of 7.3 for the data, with nearly all of the variability unexplained by host density-dependent aggregation. Working from these data, we constructed a model with similar mean parasitism and variability in total attacks per patch but without any among-parasitoid aggregation. For simplicity, we ignored host density-dependent aggregation. Visits were distributed randomly among patches, with each patch experiencing a mean of $E[V] = 4.24$ visits. Total attacks per parasitoid visit were modeled by a negative binomial–random variable with high variance ($k_A = 0.033$) and a mean of $\alpha = 0.25$ attacks per host per parasitoid patch visit. Host densities were chosen to match those shown in Pacala and Hassell (1991), and our simulation algorithm was the same as that used to generate figure 1. Our simulated data set (fig. 3*B*) exhibits the same qualitative patterns shown in Pacala and Hassell (1991), namely tremendous variability in parasitism among patches and a moderately low mean incidence of parasitism. However, the exaggerated variability in figure 3*B* is attributable solely to within-parasitoid aggre-

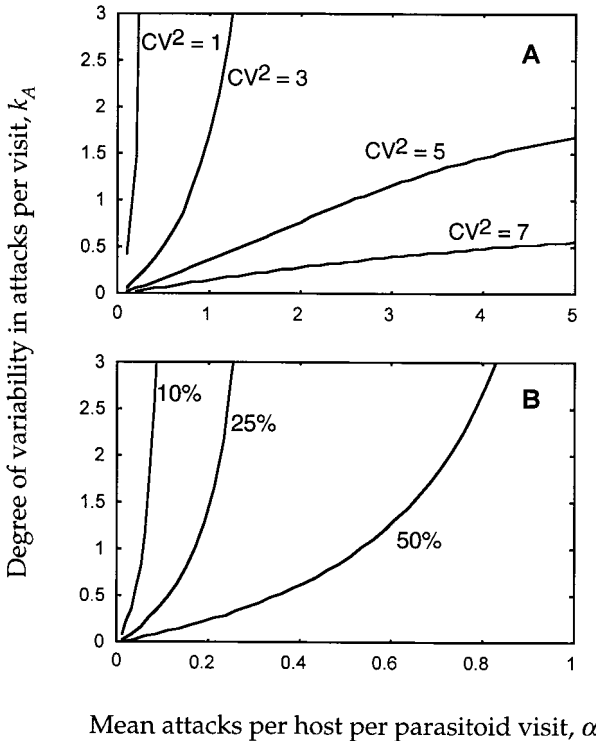

Figure 2: Levels of variability in the total number of attacks per patch that can be generated in the absence of among-parasitoid aggregation. Each contour was generated by simultaneously solving for the variability in total attacks among patches, $CV^2_{U_n} = 1/E[V](1 + 1/k_A)$ (equation [7], with n moderately large and $k_V \to \infty$), and the mean parasitism level, $1 - F^* = \exp\{E[V]([1 + \alpha/k_A]^{-k_A} - 1)\}$, where F^* is the fraction of hosts escaping parasitism. *A*, Contours for a fixed level of 25% parasitism. *B*, Contours for $CV^2_{U_n} = 1$ with 10%, 25%, and 50% parasitism.

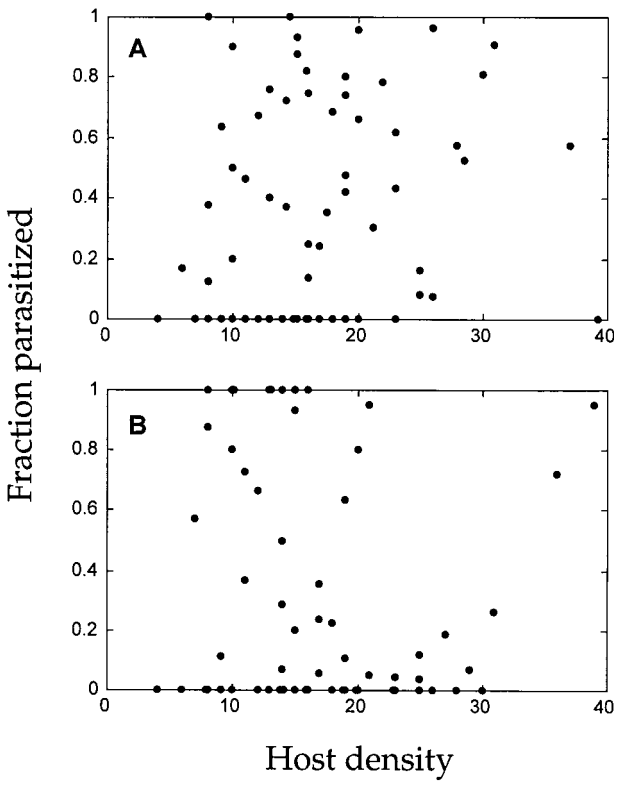

Figure 3: *A*, Parasitism of *Rhopalomyia californica* Felt by *Tetrastichus* sp. (Ehler 1986), as shown in Pacala and Hassell (1991). *B*, Simulated data from a model with no among-parasitoid aggregation. Visits per patch are randomly (Poisson) distributed with an average of 4.24 visits per patch. Total attacks per parasitoid patch visit are distributed as a negative binomial random variable with mean $0.25n$ (where n is the number of hosts in the patch) and clumping parameter $k_A = 0.033$. Parameter values were selected to give an average of 25% parasitism and $CV^2_{U_n} = 7.3$. The model producing these data is not stable.

gation of attacks; among-parasitoid aggregation is absent, and thus the system is unstable.

Our presentation of figures 2 and 3 and equations (6) and (7) is not meant to imply that one should seek to decompose an observed CV^2 into components due to among- and within-parasitoid aggregation, and then use the former to infer stability. Instead, we are simply demonstrating that any level of variability in parasitism among patches can be explained equally well by either stabilizing among-parasitoid aggregation or nonstabilizing within-parasitoid aggregation. Thus, observing high levels of variability in parasitism among patches is not grounds by itself for inferring that pseudointerference among parasitoids is operating to stabilize the system.

Discussion

The $CV^2 > 1$ rule is the latest in a series of attempts to use the distribution of parasitism among patches to assess the stabilizing potential of aggregation of risk among hosts in natural systems (Walde and Murdoch 1988). Appealing as such an analytical technique may be, its utility is hamstrung by the multitude of assumptions needed to link the distribution of parasitism among patches with stabilizing pseudointerference among parasitoids (Taylor 1993; Ives 1995). In this article, we have addressed one of these assumptions, namely that variability in the distribution of parasitism among patches reflects variability in the distribution of risk among hosts. This assumption only holds when parasitoids attack no more than one host during each visit to a patch. When parasitoids can attack multiple hosts in a patch visit, within-parasitoid aggregation of attacks inflates the variability in parasitism among patches beyond the variability in risk among hosts. Thus, without some knowledge of a parasitoid's foraging behavior within a patch, inferring the distribution of risk among hosts from the distribution of parasitism among patches is impossible.

Linking a static pattern of percentage of parasitism with the stabilizing temporal density dependence of pseudointerference requires numerous assumptions, none of which can be verified through a single snapshot of percentage of parasitism. Because the dynamical consequences of violating these assumptions are poorly understood, inferring stability from patterns of parasitism is a risky venture. Even though our analysis could be construed as providing a modified application of the $CV^2 > 1$ rule, we discourage this use. Instead, the stabilizing role of pseudointerference can best be detected by studying the density dependence of host-parasitoid interactions directly (Taylor 1993; Ives 1995). Although the abundance of published distributions of percentage of parasitism in the literature invites comparative examination and review (e.g., Lessells 1985; Stiling 1987; Walde and Murdoch 1988; Pacala and Hassell 1991), the conclusions one can draw about host-parasitoid dynamics from these data are tenuous.

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