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AGGREGATION OF RISK: RELATIONSHIPS AMONG HOST-PARASITOID MODELS

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The tendency for parasitoids to aggregate in patches where host density is high ("aggregation to host density") has been suggested as a powerful stabilizing force in host-parasitoid systems (Hassell and May 1973, 1974; Beddington et al. 1978; May and Hassell 1981; Heads and Lawton 1983; Waage 1983; Hogarth and Diamond 1984). Moreover, unlike some other potentially stabilizing mechanisms, aggregation to host density can be consistent with a low host equilibrium (Beddington et al. 1978), and thus a plausible explanation of successful biological control. Murdoch et al. (1984, 1985), however, cast doubt on whether this mechanism actually explains real cases of successful biological control.

Early models of host-parasitoid dynamics involving aggregation to host density (Hassell and May 1973, 1974) were complex and difficult to analyze. Such aggregation, however, results in a clumped distribution of parasitoid eggs among hosts. Thus, the distribution of eggs among hosts may be similar to a negative binomial distribution. Recognizing this, May (1978) proposed the following simple model to summarize the effects of aggregation:

$$H_{t+1} = FH_t(1 + aP_t/k)^{-k}, \tag{1a}$$

$$P_{t+1} = H_t - F^{-1}H_{t+1}. \tag{1b}$$

In these equations H_t and P_t are the host and parasitoid densities in generation t, F is the net rate of increase of the host in the absence of the parasitoids, a is the attack rate per parasitoid ("area of discovery"), and k is a parameter indicating the amount of aggregation of the parasitoids, that is, the clumping parameter of the negative binomial probability distribution. Equation (1a) is the unique feature of this model (eq. 1b holds for a variety of models); therefore, equation (1a) will be referred to as "May's equation."

Here we present a general class of models that includes May's phenomenological model, more-complex models incorporating explicit aggregation to host density, and models in which parasitoids aggregate in a manner not related to the dispersion of hosts in space. We examine the relationships among these different

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sorts of models in order to reveal implicit biological assumptions and to highlight the effects of these assumptions. In particular, we find implicit assumptions in May's model, which suggest that it is most naturally a model of parasitoid aggregation independent of host densities. However, we do find a narrow range of circumstances under which it represents aggregation to host density.

Our analysis highlights the dependence of aggregation on the average density of hosts in the system, and demonstrates that aggregation independent of host density is a potentially more general stabilizing force than aggregation to host density.

THE MODELING FRAMEWORK

We base our framework on a general model for populations in a patchy and locally stochastic environment developed previously (Chesson 1981). The model assumes a system of many patches, supporting local populations, with migration between patches. Important variables are the local population densities (e.g., P_j for the number of parasitoids on patch j) and the spatial averages of these densities (e.g., $\bar{P}$ for the spatial average of parasitoid density). In general, $\bar{P}$ will be a random variable, but with many patches and a degree of independence between the stochastic processes occurring on each patch, we can use the approximation $\bar{P} = EP$ (Chesson 1981), where E refers to the theoretical mean or expected value and is calculated here according to the probability distribution of P_j. Approximating $\bar{P}$ by the theoretical mean implies that $\bar{P}$ has a small variance, but it is important to keep in mind that stochastic processes may still be having strong effects on its behavior, as has been shown for the general class of models of this sort (Chesson 1981).

Murdoch and Oaten (1975) pointed out that models of host-parasitoid aggregation implicitly assume a complete redistribution of local population numbers every generation. Thus, every individual in the population must migrate. This convenient assumption will be made here also; moreover, as is usual in discrete-time models, we assume that migration and population growth processes are separated in time. Migration periods are of length $h < 1$; $P_j(t + h)$ refers to the population of parasitoids on patch j immediately after a migration period; and $P_j(t)$ refers to the population before a migration period. Some latitude in the interpretation of $P_j(t + h)$ is permissible. If parasitoids migrate several times during one time unit, then $P_j(t + h)$ is obtained by adding up the fractions of time that different mature (egg-laying) parasitoids spend in patch j during the time interval $(t, t + 1)$. Thus, a parasitoid that spends half her life in patch j and one-third in patch j' contributes ½ to $P_j(t + h)$ and ⅓ to $P_{j'}(t + h)$.

In order to compare our model with previous ones, we assume, as did earlier authors, that population dynamics within a patch follow the Nicholson-Bailey equations. Hence,

$$H_j(t + 1) = FH_j(t + h) \exp[-aP_j(t + h)] \tag{2a}$$

$$P_j(t + 1) = H_j(t + h) - F^{-1}H_j(t + 1). \tag{2b}$$

When population sizes are to be treated as integer variables, expressions (2) can be regarded as conditional means. Averaging over patches in expressions (2), we obtain

$$\bar{H}(t + 1) = FE\{H_j(t + h) \exp[-aP_j(t + h)]\} \tag{3a}$$

$$\bar{P}(t + 1) = \bar{H}(t) - F^{-1}\bar{H}(t + 1), \tag{3b}$$

where the theoretical mean value in equation (3a) is calculated using the joint probability distribution of $H_j(t + h)$ and $P_j(t + h)$. Note that equation (3b) depends on no further details and hence is the same for all of the more particular models below. Thus, in all that follows, we are concerned with equation (3a).

To model aggregation behavior in a natural way, we consider the regression of local parasitoid density on local host density. In general, this regression is a nonlinear function $f[H_j(t + h)]$, and by definition it is given by the equation

$$f[H_j(t + h)] = E[P_j(t + h) \mid H_j(t + h)] , \tag{4}$$

that is, $f[H_j(t + h)]$ is the conditional mean number of parasitoids on a patch with $H_j(t + h)$ hosts. As a regression function, in principle it can be measured in the field by standard methods. It should be kept in mind, however, that $f[H_j(t + h)]$ will depend not only on $H_j(t + h)$, but also on the spatial average densities $\bar{P}(t)$ and $\bar{H}(t)$.

There will be "error" or variation about the regression, and this variation can be just as important, as we shall see, as the regression itself. Because we expect the variance to increase with the mean and nonnegative variables are being modeled, we use a multiplicative model for the variation about the regression. Thus,

$$P_j(t + h) = f[H_j(t + h)]U_j(t + h), \tag{5}$$

where $U_j(t + h)$ is a random variable with mean 1 giving the multiplicative deviation from the regression equation for patch j and time $t + h$. This sort of multiplicative model is most appropriate when different individual parasitoids do not interact with one another. In addition, it requires local parasitoid densities to be continuous quantities, as they will be if $P_j(t + h)$ represents the fractional amounts that each parasitoid spends in patch j. If, however, the $P_j(t + h)$ are integer quantities, a suitable model is obtained by assuming that $P_j(t + h)$ is conditionally Poisson-distributed with mean $f[H_j(t + h)]U_j(t + h)$, given $H_j(t + h)$ and $U_j(t + h)$.

From the continuous model (5), it follows that

$$\bar{H}(t + 1) = FE\Big(H_j(t + h)\psi\{af[H_j(t + h)]\}\Big), \tag{6}$$

where ψ is the Laplace transform of $U_j(t + h)$:

$$\psi(u) = E \exp[-uU_j(t + h)], \tag{7}$$

and the expected value in expression (6) involves only the probability distribution of $H_j(t + h)$. The corresponding equation for integer parasitoid densities is very similar. For it, a is replaced by

$$\alpha = 1 - e^{-a} \tag{8}$$

to give

$$\bar{H}(t + 1) = FE\Big(H_j(t + h)\psi\{\alpha f[H_j(t + h)]\}\Big). \tag{9}$$

The integer model (9) gives lower rates of parasitism compared with the discrete model because $\alpha < a$. Although this difference will be of no significance for small a, it can be quite important if a is large.

We shall be interested mainly in two extreme types of models within this framework. The *pure-error*, or PE, models assume that $f[H_j(t + h)]$ is constant as a function of $H_j(t + h)$ and hence that patch-to-patch variation in parasitoid densities is explained entirely by the variation or "error" about the regression function. In such PE models, the parasitoids still aggregate, but not in response to host density. The parasitoids will have a very patchy distribution if the error variables, the $U_j(t + h)$, have high variances, but patches of high parasitoid density will not necessarily be patches of high host density.

Note that for the case in which $P_j(t + h)$ is assumed to be a continuous variable, the distribution of $P_j(t + h)/\bar{P}(t)$ (relative parasitoid density) is independent of average parasitoid density, $\bar{P}(t)$. This condition has the natural interpretation that parasitoids aggregate not in response to each other, but in response to spatially varying environmental factors. The discrete form of the PE model, involving the conditional Poisson assumption, has this interpretation also, even though it is clear that the distribution of relative parasitoid density must then depend on $\bar{P}(t)$. Later we discuss models, in which the distribution of relative host density does not vary with the average density of the host, and corresponding interpretations for the mechanisms of host aggregation.

At the end of the spectrum opposite the PE models, *pure-regression*, or PR, models assume that $P_j(t + h) = f[H_j(t + h)]$, so that parasitoid densities are explained without error by the regression equation. If f is an increasing function, patches of high host density will also be patches of high parasitoid density, and vice versa. Thus, the parasitoid distribution is explained completely by aggregation to host density.

Another kind of model that we encounter assumes that parasitoids respond in a deterministic manner to the hosts (i.e., $P_j(t + h) = f[H_j(t + h)]$) but that the attack rate, a, applicable to a given patch, is a random variable $a_j(t + h)$ that varies from patch to patch, and possibly also in time. Thus, hosts may be more vulnerable in some patches than in others, as discussed by Bailey et al. (1962). If we let a be the average value of $a_j(t + h)$ and define $U_j(t + h) = a_j(t + h)/a$, we get exactly the same equations as before for average host and parasitoid densities, but with the variation in degree of vulnerability taking the place of the "error" in parasitoid aggregation.

To give a unified analysis of these different sorts of models, we define a particular quantity called the "relative risk of parasitism" for a host individual. Consider a model containing possibly all of the features discussed above: vulnerability varies from patch to patch, parasitoids aggregate to host density, and parasitoids make "errors" when aggregating. The general expression for the

probability of parasitism for an individual host in patch j is $1 - \exp[-a_j(t + h)P_j(t + h)]$ such that $a_j(t + h)P_j(t + h)$ determines the absolute risk of parasitism for a host in patch j. We define ρ, the relative risk of parasitism, as $a_j(t + h)P_j(t + h)/a\bar{P}(t)$, which compares the risk of parasitism of the host in this model with that applying in the Nicholson-Bailey model. Note that whenever it is possible to identify hosts with the same risk, ρ can be estimated as $-\ln(\text{fraction not parasitized})/a\bar{P}(t)$. When constructing distributions of ρ from such estimated values, it should be remembered that sampling error will contaminate the distribution of natural variation in ρ.

We now use this general framework to explore existing models.

DERIVATION OF EXISTING MODELS

To derive May's equation we assume a PE model, which will usually mean

$$f[H_j(t + h)] = \bar{P}(t) \tag{10}$$

unless some parasitoids are missing, for example, in transit between patches. As a PE model, equation (10) implies that the parasitoids are not responding to local host density at all; thus any aggregation they exhibit is independent of the host distribution. Substituting equation (10) in (6) we obtain the dynamical equation

$$\bar{H}(t + 1) = F\bar{H}(t)\psi[a\bar{P}(t)] \ . \tag{11}$$

If the $U_j(t + h)$ have a gamma or Pearson Type III distribution (Johnson and Kotz 1970) with coefficient of variation $\sqrt{1/k}$, then $\psi(s) = (1 + s/k)^{-k}$ and

$$\bar{H}(t + 1) = F\bar{H}(t)[1 + a\bar{P}(t)/k]^{-k}, \tag{12}$$

which is May's equation.

Like our derivation, May's (1978) also assumes a gamma distribution of parasitoids among patches, with coefficient of variation $\sqrt{1/k}$. Unlike our derivation, May's makes no explicit statement about the relationships between host and parasitoid densities. However, in Appendix A we show that this gamma distribution of parasitoids among patches and the end result (May's equation) imply that local host and parasitoid densities are in fact uncorrelated. Thus, our derivation is essentially a formalization of May's, in which the assumptions are made explicit.

Note that the derivation above also serves to show that May's equation results when parasitoid densities do not vary from patch to patch but that the attack rate varies from patch to patch according to a gamma distribution, for example, as a result of variation from patch to patch in the degree of host vulnerability. The latter derivation was first given by Bailey et al. (1962) and has recently been discussed by Murdoch et al. (1984).

An important feature of these derivations is that ρ, which equals the error variable, $U_j(t + h)$, here, has a gamma distribution with mean 1 and variance $1/k$. We find this property to be key to all derivations of May's equation.

We have seen how May's equation arises naturally as a PE model. It was originally proposed, however, as a summary of the sort of aggregation behavior modeled by Hassell and May (1973, 1974). We now show that the Hassell and May

models fall naturally within our framework as PR models, at the end of the spectrum opposite PE models.

Because PR models assume no aggregation error, $U_j(t + h) \equiv 1$, which means that $\psi(u) = e^{-u}$. Substituting in expression (6) we obtain

$$\bar{H}(t + 1) = FE\Big(H_j(t + h)\exp\{-af[H_j(t + h)]\}\Big), \tag{13}$$

which is a form slightly more general than equation (12) of Hassell and May (1974). To make this a little clearer we can employ their notation and define $p(m) = P[H_j(t + h) = m]$, the probability that a patch has m prey, so that equation (13) becomes

$$\bar{H}(t + 1) = F\sum_{m=0}^{\infty} mp(m)\exp[-af(m)]. \tag{14}$$

Hassell and May (1974) chose the following particular formula for the regression function, f:

$$f(m) = n\bar{P}(t)T(m)/[T_0 + n\sum_{\ell=0}^{\infty} p(\ell)T(\ell)]. \tag{15}$$

In this formula T_0 is the total transit time between patches, $T(m)$ is the amount of time spent in a patch with m prey, and n is the number of patches. Since this model involves the amount of time $T(m)$ that a parasitoid spends in a patch of density m, we shall refer to it as the *patch-time* model, or PT model.

Since the PT model is a PR model, there is no error about the regression of parasitoids on hosts; parasitoid effort in any patch is determined completely by three things: the number of hosts in the patch, the host distribution among patches, and the mean parasitoid density.

In the PT model the host distribution greatly affects model behavior. For instance, if hosts do not vary from patch to patch, then the model reduces to the unstable Nicholson-Bailey equations, regardless of the form of $T(m)$. If, however, host density has a negative binomial distribution with small k, then a stable equilibrium is possible for a variety of forms of $T(m)$ (Hassell and May 1974). In contrast, for PE models, the distribution of the hosts has no effect on the dynamics of $\bar{P}$ and $\bar{H}$. Thus, the features of nature that these two models emphasize are very different.

DERIVING MAY'S EQUATION FROM MODELS OF AGGREGATION TO HOST DENSITY

As discussed above, the literature assumes that May's equation comes from aggregation of parasitoids to host density. We have shown, however, that the derivation of the equation (May 1978), which is generally regarded as supporting this claim, in fact implicitly assumes that host and parasitoid spatial distributions are uncorrelated. In this section we find a derivation of the equation that does indeed depend on aggregation to host density. We start with a PR model and ask how it can be made to conform to May's equation. In this way we are able to identify constraints on the nature of the aggregation to host density that will lead to May's equation.

The constraints are related to the fact that the relative risk of parasitism, ρ, must have a distribution among hosts that is independent of average host density $\bar{H}$. If it is postulated that parasitoids respond to relative host density, $H_j/\bar{H}$, and are not affected by the average host density, $\bar{H}$, which is an absolute density measure, then it is not difficult to identify aggregation functions and host distributions that satisfy the constraints. This does not necessarily mean the constraints are likely to be satisfied in nature, but only that an interesting class of models can be identified where the constraints are satisfied. For example, one of the constraints implies that the shape of the aggregation function is related to the shape of the host distribution in a particular way. In the section "Generalizations of May's Equation," we consider whether violation of this particular constraint is likely to lead to an equation differing in important respects from May's equation.

Proceeding with the derivation, we assume that parasitoids do not affect the movements and egg-laying activities of individual conspecifics. Then we can write

$$f(H_j) = \bar{P}(t)g(H_j), \tag{16}$$

where $g(H_j)$ measures the relative density of parasitoids in patches with host density H_j (which we now use as a shorthand for $H_j(t + h)$), and depends on $\bar{H}$ but not on $\bar{P}$. This particular form of the regression function applies to the PT model, among others. We assume here that $g(H_j)$ is an increasing function of H_j (as it is in the PT version of the PR model), but the results below hold with minor modification for decreasing functions also.

Substituting equation (16) in equation (13) for the PR model, we obtain

$$\bar{H}(t + 1)/F\bar{H}(t) = E\left\{\frac{H_j}{\bar{H}(t)} \exp[-a\bar{P}(t)g(H_j)]\right\}, \tag{17}$$

and from May's equation we have

$$\bar{H}(t + 1)/F\bar{H}(t) = [1 + a\bar{P}(t)/k]^{-k}. \tag{18}$$

Since $a\bar{P}(t)$ is a continuous variable, expression (17) can be regarded as the Laplace transform (with argument $a\bar{P}(t)$) of the random variable $g(H_j)$. This Laplace transform is not taken with respect to the usual probability distribution of $g(H_j)$, but with respect to the distribution obtained by weighting the usual distribution by $H_j/\bar{H}$. This weighted distribution is appropriate if one samples $g(H_j)$ by host rather than by patch. Note that here $\rho = g(H_j)$; note also that the weighted distribution of $g(H_j)$ can be interpreted as the distribution of ρ for a randomly chosen host.

Now expression (18) is the Laplace transform of a gamma random variable with mean 1 and coefficient of variation $1/k$. This gamma random variable has probability density

$$[k^k/\Gamma(k)](y^{k-1}e^{-ky}), \qquad y > 0 \ . \tag{19}$$

From the uniqueness of Laplace transforms, it follows that expressions (17) and (18) are equal if and only if the weighted distribution of $g(H_j)$ (or, equivalently, the distribution of ρ) has the density (19). This can only be so if H_j has a continuous distribution (not the discrete distribution in Hassell and May 1974). Defining $h(g)$

as the inverse function of $g(h)$, we find that the unweighted probability density of $g(H_j)$ must be

$$[k^k/\Gamma(k)]\ [\bar{H}/h(g)]\ (g^{k-1}e^{-kg}), \qquad g > 0\,, \tag{20}$$

and the probability density of H_j must therefore be

$$(\bar{H}/h)\ [k^k/\Gamma(k)]\ [g(h)]^{k-1}\exp[-kg(h)]g'(h)\,. \tag{21}$$

Conversely, starting with expression (21) and substituting in equation (17), May's equation is derived.

Although we appear to have solved the problem of obtaining May's equation from the PR model, one crucial element is lacking. We have no assurance that expression (21) actually is a probability density; that is, there is no reason to believe that it will integrate to 1. If expression (20) is a probability density, it follows that expression (21) is also; thus the problem of obtaining May's equation from the PR model becomes that of finding functions $g(h)$ such that expression (20) is a probability density.

It turns out that a variety of forms of $g(h)$ will work. For example, a useful class of functions $g(h)$ is obtained by seeking $h(g)$ in the form

$$h(g) = ch_0(g)\,, \tag{22}$$

where c is a function of $\bar{H}$, but $h_0(g)$ is a function of g alone. This implies that $g(h) = g_0(h/c)$ where g_0 is the inverse of h_0, and equation (22) specifies the dependence of $g(H_j)$ on H_j up to a scale factor c depending on $\bar{H}$. We now solve for c by substituting in expression (20), integrating from 0 to ∞, and equating to 1. Defining

$$\kappa = [k^k/\Gamma(k)] \int_0^\infty [g^{k-1}e^{-kg}/h_0(g)]dg\,, \tag{23}$$

the solution is $c = \bar{H}\kappa$; thus,

$$g(H_j) = g_0(H_j/\bar{H}\kappa)\,. \tag{24}$$

The sole requirement for the existence of this solution is that κ should be finite. It follows then that the function (24) and the density (21) serve to derive May's equation from the PR model.

Example.—In analogy with Hassell and May (1973), let $g_0(h) = h^\theta$, then $\kappa = k^{1/\theta}\Gamma(k - 1/\theta)/\Gamma(k)$ and

$$g(H_j) = [(H_j/\bar{H})^\theta/k]\ [\Gamma(k)/\Gamma(k - 1/\theta)]^\theta\,. \tag{25}$$

As in all models based on equation (22), the density of $H_j/\bar{H}$ is independent of $\bar{H}$, and in this example the density of $Y_j = H_j/\bar{H}$ is

$$[\theta k^k/\Gamma(k)\kappa^{\theta k}]y^{\theta k-2}\exp\,[-y^\theta k/\kappa^\theta], \qquad y > 0\,. \tag{26}$$

The condition $\kappa < \infty$ is the condition $k > 1/\theta$. Thus if $k > 1/\theta$, the aggregation function (25) and the host distribution (26) lead to May's equation.

For all of the solutions in this section, including the particular example just presented, the relative parasitoid density, $g(H_j)$, is a function of relative host density, $H_j/\bar{H}$, alone, and the distribution of $H_j/\bar{H}$ among patches is independent

of $\bar{H}$. A great variety of shapes for g_0, relating relative parasitoid density to relative host density, is possible since the only requirement is that expression (23) be finite. However, g_0 and the host distribution cannot be chosen independently; indeed, if g_0 and k are given, then the host distribution is precisely determined. If k is not predetermined, then there is some flexibility in the relationship between g_0 and the host distribution: each g_0 determines a one-parameter family of host distributions. The single parameter is k, and for different values of k the host distribution will have different shapes. The manner in which the shape changes with k is determined in a precise way by g_0. Therefore, it follows that if g_0 and the probability density of $H_j/\bar{H}$ are chosen in some random manner from the collection of all possible host distributions and aggregation functions, it is very unlikely that May's equation will be derived. The significance of this finding is explored below ("Generalizations of May's Equation").

AGGREGATION INVOLVING ABSOLUTE HOST DENSITY

We have seen how May's equation can be derived if parasitoids respond only to the local relative density, $H_j/\bar{H}$, of hosts. Most detailed models of aggregation, however, require consideration of the average host density, $\bar{H}$ (e.g., Hassell and May 1974; Murdoch and Oaten 1975; Comins and Hassell 1979). Thus, it is important to see if aggregation involving $\bar{H}$ can lead to May's equation. We do not try to do this in complete generality but instead restrict attention to a single broad class of models, the PT models of Hassell and May (1974). A desirable feature of these models is that the aggregation function can be interpreted in terms of a plausible model for the behavior of the parasitoids.

The PT models involve the important quantities $T(H_j)$, the time spent in a patch with H_j hosts, and $\tau = T_0/n$, the mean transit time between patches. We find that May's equation is unavailable from PT models with $\tau > 0$. It is available for one form of the PT model in which $\tau = 0$, which is the only form of the model in which $g(H_j)$ can be expressed as a function of $H_j/\bar{H}$ alone, and does not appear to be available from any forms of the PT model that differ appreciably from this particular form.

To overcome this problem we suggest modifications to May's equation that let aggregation depend on absolute host densities. These modifications make the attack rate a, or the clumping parameter k, functions of $\bar{H}$.

To see what happens in the PT model, we start with expression (15) and divide by $\bar{P}$ to get

$$g(H_j) = c'T(H_j), \tag{27}$$

where

$$c' = [\tau + ET(H_j)]^{-1}, \tag{28}$$

with $ET(H_j) = \sum_{\ell=0}^{\infty} p(\ell)T(\ell)$ being the mean time in a patch. The parameter τ is assumed to be a constant, independent of host density, and $ET(H_j)$ is a function of $\bar{H}$.

With this form for $g(H_j)$ we seek a solution c', as a function of $\bar{H}$, to the equation

$$\int_0^\infty [k^k/\Gamma(k)]\,[\bar{H}/T^{-1}(g/c')]g^{k-1}e^{-kg}dg = 1\,, \tag{29}$$

which again is merely the condition that expression (20) is a probability density.

Under the assumptions that T is strictly increasing, $T(0) = 0$, and $T(\infty) = \infty$, we show in Appendix B that there is a unique solution c', as a function of $\bar{H}$, provided that the left-hand side of (29) is finite for some positive value of c'. Although its occurrence is unlikely in nature, the requirement that $T(0) = 0$ cannot be relaxed; otherwise, it is impossible for the weighted distribution of $g(H_j)$ to be gamma. The second condition, $T(\infty) = \infty$, is also necessary for this gamma distribution.

Having obtained a solution for c', we ask, Can it be put in the form of expression (28)? If we insist that τ, the mean transit time, is truly a constant (as assumed in Hassell and May 1974), the answer is no, because c' is a decreasing function of $\bar{H}$ and tends to infinity as $\bar{H}$ tends to zero (Appendix B). This means that $\tau + ET(H_j)$ must tend to zero as $\bar{H}$ tends to zero, and this can only be so if τ tends to zero as $\bar{H}$ tends to zero. Although parasitoids would be well advised to hurry between patches when hosts are scarce, it seems unlikely that τ could be made arbitrarily small. If one argues that τ is not really a mean transit time, but time out from oviposition activities to do other things, and the true transit time between patches is essentially 0, then τ might conceivably tend to zero as $\bar{H}$ tends to zero, as oviposition becomes a more urgent activity. This modifies the intent of the PT model, and we must conclude that in its original form the PT model does not give rise to May's equation. An example is instructive.

For the special case $T(H_j) = H_j^\theta$, the problem of finding c' is equivalent to the problem of finding c discussed in the preceding section. This case can be solved explicitly from the example there, in which $g_0(h) = h^\theta$. Although Hassell and May (1974) suggested more-complicated functions for $T(H_j)$, this form of T is capable of approximating some of their forms, provided host densities are generally low.

From expression (25) in the earlier example we see that

$$c' = [1/k]\,[\Gamma(k)/\bar{H}\,\Gamma(k - 1/\theta)]^\theta\,. \tag{30}$$

A simple integration using (20) shows that

$$Eg(H_j) = 1 - 1/k\theta\,. \tag{31}$$

Combining results (30) and (31), we see that

$$\tau = \theta^{-1}[\bar{H}\,\Gamma(k - 1/\theta)/\Gamma(k)]^\theta\,, \tag{32}$$

demonstrating a high dependence of τ on host density.

A transit time so heavily dependent on host density contradicts the spirit of the PT model, as discussed above. What are the alternatives? One alternative is that k depends on host density, which means from (32) that k varies according to the equation

$$\Gamma(k)/\Gamma(k - 1/\theta) = \bar{H} \times \text{constant}. \tag{33}$$

Since the gamma function is log convex, the left-hand side of (33) increases in k, approaches zero as $k \to 1/\theta$, and approaches infinity as $k \to \infty$. Thus if τ is held fixed, k will be an increasing function of host density.

Another alternative is that the parameter a in May's equation is a function of host density. This parameter can be interpreted as an attack rate; even though we assume that its value in any patch is independent of local and average host densities, it is quite natural to expect that the "effective" value of a for the entire system will vary with the average host density. For instance, at low, as compared with high, host densities, parasitoids should spend less time in patches and more time in transit between patches, leading to lower numbers of attacks per host. A model with these properties originates from a slight modification of the present example. We choose the same host distribution, which is given by expression (26), and the same function $T(H_j) = H_j^\theta$, but we insist that τ be positive and constant.

With the host distribution (26), we have

$$ET(H_j) = (\bar{H})^\theta\,(k - 1/\theta)\,[\Gamma(k - 1/\theta)/\Gamma(k)]^\theta, \tag{34}$$

and $g(H_j)$ is then given by the standard form $g(H_j) = T(H_j)/[\tau + ET(H_j)]$, for the PT model. From formula (17) for the PT model we obtain

$$\bar{H}(t+1) = F\bar{H}(t)\{1 + a\bar{P}(\bar{H})^\theta/[\tau d(k,\theta) + (k - 1/\theta)(\bar{H})^\theta]\}^{-k}, \tag{35}$$

where $d(k,\theta) = [\Gamma(k)/\Gamma(k - 1/\theta)]^\theta$. Thus we obtain from a PT model a result that amounts to substituting the following function of host density for a in May's equation:

$$ka(\bar{H})^\theta/[\tau d(k,\theta) + (k - 1/\theta)(\bar{H})^\theta]. \tag{36}$$

Interestingly, this result is only slightly more complicated than May's equation. Even so, it is distinguished from May's equation by the important property that the fraction of hosts surviving strongly depends on host density. Moreover, the fraction of hosts surviving decreases as host density increases, reflecting the smaller time loss incurred in transit between patches at higher host density. This is similar to the generation of Type III functional responses for models of predation in a patchy environment (Murdoch and Oaten 1975). As we have emphasized, it is likely that the fraction of hosts surviving will often depend on $\bar{H}$. This fact is frequently overlooked in analyses of host-parasitoid aggregation models based on the concept of "pseudointerference" as defined in Free et al. (1977).

The above arguments have all assumed that $\tau > 0$. If τ is set equal to 0, which requires arguing that transit time is truly negligible at all host densities, then May's equation can be obtained in the case where $T(H_j)$ is proportional to H_j^θ, for then $T(H_j)/ET(H_j)$ can be a function of $H_j/\bar{H}$ alone. Putting $\tau = 0$ in the last example above achieves this but note that a in May's equation is replaced by $a/(1 - 1/k\theta)$. It is not difficult to see that this is the only case in which $T(H_j)/ET(H_j)$ depends solely on $H_j/\bar{H}$. From the above results (in "Deriving May's Equation from Models of Aggregation to Host Density"), it follows that other cases require distributions of relative density ($H_j/\bar{H}$) that vary with $\bar{H}$ if there is to be any hope of deriving May's equation. Moreover, Appendix C shows that the class of situations in which May's equation is derivable from PT models with $\tau = 0$ is

small indeed. All models in this class have a property that makes them very close to the model with $T(H_j) = H_j^\theta$.

GENERALIZATIONS OF MAY'S EQUATION

Although it is not likely that May's equation will arise as a result of aggregation to host density, is it possible that an equation with similar properties could arise? May's equation is a special case of equation (11), $\bar{H}(t + 1) = F\bar{H}(t)\psi[a\bar{P}(t)]$, which was given earlier for the general PE model. Because an equation of this same form was originally derived by Bailey et al. (1962) under the assumption that the attack rate varies among host individuals, we call it the BNW equation. We ask whether May's equation reflects some general useful properties of its generalization, the BNW equation, and examine the various ways in which the BNW equation can be derived.

In equation (11), ψ is the Laplace transform of a positive random variable U. As noted earlier in the PE model, the error variable, U, can be equated with ρ, the relative risk of parasitism. In the new models below that yield (11), ψ can still be interpreted as the Laplace transform of ρ from a randomly chosen host.

Previously, U was assumed to have mean 1; because this assumption is not critical to the properties of the equation, we shall relax this assumption and simply allow U to have any constant mean value. Thus, it is no longer just an error variable.

It is shown in Appendix D that the BNW equation, like May's equation, gives unique, positive, locally stable host and parasitoid equilibrium densities if the distribution of U is sufficiently skewed. Skewness is measured here by the spread of the distribution of $\log U$.

To obtain the BNW equation from a PR model (aggregation to host density), we follow the same method used for May's equation. We see that the density of H_j must be of the form

$$(\bar{H}/h)p[g(h)]g'(h)\,, \tag{37}$$

where p is the probability density of U. Since p is arbitrary here, this formulation removes the linkage between the shape of the distribution of H_j and the shape of the aggregation function g, at least for a fixed value of $\bar{H}$. That (37) must integrate to 1 means that constraints are still present. These constraints manifest themselves through restrictions on the manner in which g can depend on $\bar{H}$ and through restrictions on how the shape of the density of H_j changes with $\bar{H}$.

The restriction on the manner in which g depends on $\bar{H}$ is best illustrated by the PT model where $g(H_j) = c'T(H_j)$ with $c' = [\tau + ET(H_j)]^{-1}$. As we found for May's equation, it is essentially impossible to obtain a solution for c' unless we assume that $\tau \to 0$ as $\bar{H} \to 0$ (Appendix B).

If we reintroduce the special assumption that parasitoids respond solely to the relative density of the hosts, then it is indeed possible to derive the BNW equation without significant further restrictions on the manner in which parasitoids react to the hosts. However, it must be assumed also that relative host density $(H_j/\bar{H})$ is distributed among patches in a manner that does not change with average host density $(\bar{H})$.

To derive the BNW equation with these special assumptions, let $g(H_j) = \tilde{g}(H_j/\bar{H})$, where $\tilde{g}(H_j/\bar{H})$ depends just on the single variable $H_j/\bar{H}$, and let ϕ be the probability density of $H_j/\bar{H}$. It follows that

$$\begin{aligned}\bar{H}(t+1)/F\bar{H}(t) &= E(H_j/\bar{H})\exp[-a\bar{P}g(H_j)] \\ &= E(H_j/\bar{H})\exp[-a\bar{P}\tilde{g}(H_j/\bar{H})] \\ &= \int_0^\infty y\,\exp[-a\bar{P}\tilde{g}(y)]\phi(y)dy \\ &= \int_0^\infty \tilde{h}'(u)\tilde{h}(u)\phi[\tilde{h}(u)]\exp(-a\bar{P}u)du\,, \qquad (38)\end{aligned}$$

where $\tilde{h}$ is the inverse of the function $\tilde{g}$. Thus, letting $p(u)$ be the density function,

$$p(u) = \tilde{h}'(u)\tilde{h}(u)\phi[\tilde{h}(u)]\,, \qquad (39)$$

with ψ its Laplace transform, we obtain equation (11). This shows that equation (11) results from any PR model, in which relative parasitoid density, $g(H_j)$, is merely an increasing function of relative host density, $H_j/\bar{H}$, with the probability density ϕ of this latter quantity not depending on $\bar{H}$. To arrive at equation (11), it is not necessary that $\tilde{g}$ be increasing; for example, $\tilde{g}$ could be decreasing. Indeed, $\tilde{g}$ need not even be monotonic, but then formula (39) must be replaced by something more complicated.

Formula (39) shows how p and hence ψ are determined by $\tilde{g}$ and ϕ. Moreover, equation (39) would still apply if $\tilde{g}$ and/or ϕ depended on $\bar{H}$. Therefore, it is impossible to have p (and hence ψ) independent of $\bar{H}$ if $\tilde{g}$, but not ϕ, depends on $\bar{H}$, or if ϕ, but not $\tilde{g}$, depends on $\bar{H}$. It is possible to have p independent of $\bar{H}$ if both $\tilde{g}$ and ϕ depend on $\bar{H}$, but we know of no biologically interesting examples.

Because greater aggregation is believed to lead to more stability in May's equation, it is important to find out if greater aggregation leads to more stability in the BNW equation as derived here. We shall not pursue this in general but just give a simple example.

Let $\tilde{g}(H_j/\bar{H}) = c(H_j/\bar{H})^\theta$, where c is a constant. Large values of θ mean a higher concentration of effort in patches with high host density. In the presence of mild regularity conditions, Appendix D shows that stability always results from a sufficiently large θ. Thus, greater aggregation, in the sense of large θ, does indeed lead to more stability. The stabilizing effect of such aggregation does not follow, however, from the tendency of parasitoids to aggregate to patches with more hosts, per se, but from the tendency to aggregate in some subset of the patches. In fact, stability would also result if parasitoids aggregated instead to patches with few prey. This is perhaps not plausible behavior, and we illustrate the point by considering instead how egg depletion, prey-handling time, or some other factor might cause relative risk to be greater in patches with few hosts. Such stabilizing effects of inverse density dependence have also been found by Hassell (1984).

Until now we have interpreted the function $g(H_j)$ as the relative density of parasitoids in patch j, as in equation (16). In this situation, the vulnerability of a host, a, is the same for all hosts, but the number of parasitoids varies. An alternative approach is to assume that vulnerability varies among hosts, but that

all patches receive the same number of parasitoids, $\bar{P}$, as in Bailey et al. (1962). Now, $g(H_j)$ can be interpreted as a weighting function modifying the vulnerability of hosts in patch j, according to the local host density.

When all patches have the same number of parasitoids, individual hosts in patches containing many hosts are probably at less risk than those containing few hosts because parasitoids in dense patches will spend more time handling hosts or will deplete their egg supply more frequently. Thus, $g(H_j)$ will be decreasing. In the example discussed, we would then have $g(H_j/\bar{H}) = c(H_j/\bar{H})^{\theta}$, with θ negative. Appendix D shows that stability always results from a sufficiently large value for $|\theta|$. As risk becomes more inversely density-dependent in space, stability becomes likely.

DISCUSSION

May's negative binomial model (eq. 1a) has been used in a variety of situations in which parasitoids are assumed to aggregate in areas of high host density (Beddington et al. 1978; Hassell 1980, 1982; May and Hassell 1981; Hogarth and Diamond 1984). The model is very simple, provides an explanation for stable coexistence of hosts and parasitoids, and has seemed to apply to a variety of situations. It has thus been very appealing. Our analysis shows, however, that the range of situations to which it should be applied is quite narrow. Although most recent references to the model assume that it arises from the aggregation of parasitoids in patches of high host density, we have shown here that it arises most naturally when the aggregation of parasitoids is not related to local host densities; indeed, we showed that the latter is an implicit assumption of May's derivation.

The key to the derivation of May's model is the distribution of a quantity, ρ, which is the relative risk of parasitism. This quantity can vary from host individual to host individual for two reasons. First, different host individuals are exposed to different densities (or qualities) of parasitoids. Second, some host individuals may be more difficult to find or parasitize than others because of their microhabitat or because of phenotypic differences; thus, different hosts may be exposed to different values of the parasitoid attack rate (the area of discovery, a).

To arrive at May's equation, the distribution of ρ among hosts must follow the gamma probability distribution. In the more general model of Bailey et al. (1962), ρ may have any distribution. Especially significant is that both models require that the parameters of the distribution of ρ must *not* change with the average densities of the host and parasitoid populations. Also, in both models, stability requires the distribution of relative risk to be highly skewed, with a small fraction of the hosts accounting for most of the risk of parasitism.

Field studies typically have not inquired into the distribution of ρ, but rather have examined the spatial distribution of risk as a function of local host density. A wide range of patterns has been found. In some studies risk increased with local host density, in others it decreased, and, perhaps most commonly, in others there was no relationship (e.g., Heads and Lawton 1983; Waage 1983; Murdoch et al. 1984; Reeve and Murdoch 1985; for reviews, see Morrison and Strong 1980; Hassell 1982).

The distribution of ρ, or of a variable closely related to it, has been studied by

Hassell (1980), Murdoch et al. (1984), and Reeve and Murdoch (1985). Hassell showed that while risk in the winter moth was highly skewed in some years, the skewness decreased with average moth density. The other two studies showed, by contrast, that the distribution of the risk of parasitism in two species of scale insects under biological control is not strongly skewed, but has a surprisingly moderate variance.

Although the distributions of risk in the scale-insect studies seem to have been random with respect to local density, the winter-moth data show that risk increases with local host density, suggesting that the parasitoids aggregate to host density. Evidence for such aggregation has been found for predators in the field (Kareiva 1984) and for parasitoids in the laboratory (e.g., Cook and Hubbard 1977). When considering the models under discussion, however, it is critical to know whether the allocation of search time among patches with different host densities changes as a function of the average host density. Surprisingly, this problem seems not to have been studied experimentally.

It seems unlikely that parasitoids would always respond in exactly the same way to a given relative density, or set of relative densities, regardless of the absolute host densities. Models of optimal foraging or oviposition generally reveal the importance of absolute host densities, as can be seen by inspecting the equations in such studies (e.g., Charnov 1976; Cook and Hubbard 1977; Oaten 1977; Comins and Hassell 1979; Green 1980). If a parasitoid is not exhibiting "optimal" behavior, but is merely "trying to do well enough," then absolute host densities are possibly even more important, for then a parasitoid would be expected to remain a long time in a dense patch of hosts regardless of its sparseness relative to other patches (e.g., these properties occur in models 1 and 2 of Hassell and May 1974).

May (1978) suggested his equation as a simple summary of the results of aggregation to host density. Because of the likely involvement of absolute host density, we have argued that May's equation is not an adequate summary of the effects of such aggregation. We found, however, that we could modify May's equation to incorporate the effects of absolute host density. We looked at two cases. In one case, the value of the aggregation parameter k was allowed to vary with average host density, and in the second case, the attack-rate parameter a was made a function of average host density. (This second case does not imply that attack rates within a patch vary with absolute host density; they do not. Because varying amounts of time are wasted in transit between patches, the effective attack rate for the entire system, as represented by a, does vary with absolute host density.)

A general discussion of the dynamical properties of these and other alternatives to May's equation that represent aggregation involving absolute host densities is deferred to a subsequent article. However, some conclusions are clear from the present study. (1) Our analysis of the equation of Bailey et al. (1962) shows that aggregation by parasitoids, independent of host density, is a general stabilizing force in Nicholson-Bailey-type models. (2) When parasitoid aggregation to local host density leads to stability, it does so because aggregation in any subset of patches leads to variation in the relative rate of parasitism among hosts, not

because patches with many parasitoids contain many hosts. (3) Aggregation to patches containing relatively few hosts has the same effect on stability as aggregation to patches with many hosts. That is, the intuitive argument that stability follows from spatial density dependence per se is incorrect; stability follows equally from inverse density dependence in space. Egg depletion and handling time are likely mechanisms yielding such inverse density dependence. Both forms of spatial density dependence can lead to "pseudointerference" (Free et al. 1977), and stability can be understood in these terms.

SUMMARY

We explore the relationships among host-parasitoid models involving aggregation, with particular emphasis on May's negative binomial model. Models in which parasitoid density in a patch is strictly a function of host density in the patch, with no "error" about this function, are *pure-regression* models. Those in which there is random variation in the number of parasitoids per patch, with no relationship between local parasitoid density and local host density, are *pure-error* models. The key factor in these models is not the distribution of parasitoids per se, but the distribution of the relative risk of parasitism, ρ, which in the present formulation can result from variation in the number of parasitoids in a patch, P_j, or from variation in host vulnerability, a.

We show that May's model, and the Bailey et al. (1962) model of which it is a special case, arises naturally from pure-error models. By contrast, it is very difficult to obtain May's model from biologically plausible pure-regression models. There are severe constraints to obtaining May's model under pure regression: (1) the parasitoids must aggregate in response to relative host density only, and must be unresponsive to the absolute number of hosts in a patch; (2) they must be unresponsive to parasitoid density; (3) the distribution of hosts must be independent of average host density; (4) once the parasitoid aggregation function is established, only one corresponding host distribution yields May's model; (5) ρ must be gamma-distributed with constant parameters. It is not possible to obtain May's model if parasitoid aggregation to local host density involves a transit time between patches. Only the first three of these constraints apply to obtaining the model of Bailey et al.

When parasitoids aggregate to local host density and the above constraints are not met, modifications of May's model may arise in which the constants a and k are replaced by functions of average host density. There is then no necessary relationship between aggregation and stability. By contrast, random aggregation in space, independent of local host density, leads to May's model (or, more generally, to that of Bailey et al.) and serves as a powerful stabilizing force in the Nicholson-Bailey context. However, stability is obtained at the cost of lowered parasitoid efficiency.

Where parasitoid aggregation in relation to local host density occurs under the constraints listed above, we find that aggregation to patches with few prey is as effective as aggregation to patches with many prey in yielding both May's model and local stability. Under the identified constraints, any process (e.g., egg deple-

tion or handling time) that strongly concentrates relative risk in sparser patches, thus producing inverse spatial density dependence, will promote stability as effectively as does spatial density dependence.

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APPENDIX A

Equation (3a) and May's equation imply that

$$E(H_j/\bar{H})e^{-aP_j} = (1 + a\bar{P}/k)^{-k}. \tag{A1}$$

Defining $\phi(P_j) = E[H_j/\bar{H}|P_j]$, equation (A1) implies that

$$E\phi(P_j)e^{-aP_j} = (1 + a\bar{P}/k)^{-k}. \tag{A2}$$

Assuming that this relation holds for an interval of a values for a fixed joint distribution of H_j and P_j, the left-hand side of equation (A2) is the Laplace transform of the distribution of P_j weighted by $\phi(P_j)$. An explicit assumption in May's (1978) derivation is that P_j has a gamma distribution with mean $\bar{P}$ and coefficient of variation $1/k$. The ordinary Laplace transform of this gamma distribution is the right-hand side of equation (A2). Thus, the uniqueness of Laplace transforms implies that $\phi(P_j) = 1$, which means that $E[H_j|P_j] = \bar{H}$; that is, the regression of host density on parasitoid density has zero slope. This is stronger than the statement that H_j and P_j are uncorrelated but not as strong as statistical independence, since P_j could influence the variance of H_j.

APPENDIX B

To see that there are solutions to equation (29), let G be a gamma random variable with mean 1 and variance $1/k$. Then equation (29) can be written

$$E[T^{-1}(G/c')]^{-1} = \bar{H}^{-1}. \tag{B1}$$

Assuming that $T(0) = 0$, $T(\infty) = \infty$, and T is strictly increasing, then $[T^{-1}(G/c')]^{-1}$ is strictly increasing in c', converging to ∞ as $c' \to \infty$ and to zero as $c' \to 0$. Assuming that $E[T^{-1}(G/c')]^{-1} < \infty$ for all c', the dominated and monotonic-convergence theorems imply that $E[T^{-1}(G/c')]^{-1}$ is continuous and is strictly increasing in c', converging to zero and ∞, respectively, as $c' \to 0$ or ∞. Thus, under these conditions there must be a unique solution c' to equation (B1) for every $\bar{H}$. Note also that c' is necessarily a decreasing function of $\bar{H}$, converging to ∞ as $\bar{H} \to 0$.

To see that $E[T^{-1}(G/c')]^{-1}$ is finite for all c' if it is finite for any c', substitute $y = g/c'$ in the left-hand side of equation (29) to obtain

$$E[T^{-1}(G/c')]^{-1} = c'^{k}\int_0^\infty [k^k y^{k-1} e^{-c'ky}/\Gamma(k)T^{-1}(y)]dy. \tag{B2}$$

Since $T^{-1}(y)$ is increasing, the integral will be finite for all positive c' if and only if $y^{k-1}/T^{-1}(y)$ is integrable near 0. Thus if equation (B2) is finite for any positive c', it is finite for all positive c'.

The case in which we seek the BNW equation from PT models is dealt with almost identically. The variable G is then assumed to have Laplace transform ψ, and density

function p, but it is no longer true that the left-hand side of equation (B1) is finite for all c' if it is finite for any c'. However, imposing the mild condition $\log p(y)/\log y \to \nu$ as $y \to 0$, where ν is some finite number, preserves the relevant properties of the gamma distribution and the critical result that $c' \to \infty$ as $\bar{H} \to 0$. This particular result means that c' cannot take the form $[\tau + ET(H_j)]^{-1}$, as discussed in the text. The case $\nu = \infty$ might occasionally arise in practice, but then quite unrealistic T are necessary before $E[T^{-1}(G/c')]^{-1} = \infty$, such that the results here can be considered quite general.

APPENDIX C

The PT models with $\tau = 0$ have $g(H_j) = T(H_j)/ET(H_j)$, which implies that $Eg(H_j) = 1$. We seek to identify the breadth of situations in which the BNW equation (which includes May's equation as a special case) is available from such PT models. Writing $h(g)$, the inverse of $g(h)$, as $1/w(g)$, we obtain $\bar{H}p(g)w(g)$ for the density of $g(H_j)$ such that

$$Eg(H_j) = \bar{H} \int_0^\infty gp(g)w(g)dg . \tag{C1}$$

Since the density of $g(H_j)$ must integrate to 1, equation (C1) can be written

$$Eg(H_j) = \int_0^\infty gp(g)w(g)dg / \int_0^\infty p(g)w(g)dg . \tag{C2}$$

In general, $g(H_j)$ and $w(g)$ will depend on $\bar{H}$; however, by hypothesis, $Eg(H_j)$ does not. Using D to represent the derivative with respect to $\bar{H}$, we obtain

$$\begin{aligned} 0 &= DEg(H_j) \\ &= \int_0^\infty gp(g)Dw(g)dg / \int_0^\infty p(g)w(g)dg \\ &\quad - [\int_0^\infty p(g)Dw(g)dg][\int_0^\infty gp(g)w(g)dg] / [\int_0^\infty p(g)w(g)dg]^2 \qquad \text{(C3)} \\ &= E[GD \log w(G)] - (EG)E[D \log w(G)] \qquad \text{(C4)} \\ &= C[G, D \log w(G)] , \qquad \text{(C5)} \end{aligned}$$

where C means covariance. Now $D \log w(G) = -D \log h(G)$, so that equation (C5) means that on the average $D \log h(G)$ must neither increase nor decrease with G, to produce this zero covariance. For $g(H_j) = H_j^\theta / c(\bar{H})$, where $c(\bar{H})$ is some function of $\bar{H}$, we get $h(G) = [Gc(\bar{H})]^{1/\theta}$ and $\log h(G) = \theta^{-1} \log G + \theta^{-1} \log c(\bar{H})$. Because $\log h(G)$ separates into a sum of a function of G and a function of $\bar{H}$, $D \log h(G)$ is independent of G, and equation (C5) is therefore satisfied. However, if $g(H_j) = T(H_j)/c(\bar{H})$, for $T(H_j) \neq H_j^\theta$, then $\log h(G)$ does not separate into a sum of a function of G and a function of $\bar{H}$; it follows that $D \log h(G)$ depends on G, making it very unlikely that the covariance (C5) will be zero for all values of $\bar{H}$. For example, if $T(H_j) = \log(1 + bH_j)$, for positive b, the covariance has the same sign as $-Dc(\bar{H})$.

These results indicate that the class of PT models with $\tau = 0$ that leads to the BNW equation is small indeed.

APPENDIX D

The equation for general pure-error models is

$$\bar{H}(t+1) = F\bar{H}(t)\psi[a\bar{P}(t)] , \tag{D1}$$

where ψ is the Laplace transform of the error variable U. We assume here that U is a positive random variable with any mean. For the case in which U is a gamma random variable with mean 1 and variance $1/k$, equation (D1) gives May's equation. May's equation becomes stable for $k < 1$, which corresponds to a skewed distribution of U with a mode at

zero and a long tail. We show here that in a certain sense sufficient skewness of the distribution of U in general PE models also leads to a locally stable equilibrium.

Equation (D1) implies that the equilibrium parasitoid density is the solution P^* to the equation

$$\psi(aP^*) = F^{-1}. \tag{D2}$$

Since $F^{-1} < 1$, the properties of Laplace transforms ensure that equation (D2) has a unique solution. From equation (3b) the equilibrium host density is $H^* = P^*/(1-F^{-1})$.

From a general analysis of host-parasitoid models (Hassell 1978, Appendix III), it follows that the equilibrium will be locally stable if

$$-aH^*\psi'(aP^*) < F^{-1}. \tag{D3}$$

Defining $\delta = aP^*$, and noting that $\psi'(\delta) = -EUe^{-\delta U}$, inequality (D3) becomes

$$E\delta Ue^{-\delta U} < F^{-1}(1-F^{-1}). \tag{D4}$$

One interpretation of increasing skewness of the distribution of U is that the distribution of $\log U$ becomes more spread out. This can be defined precisely for all positive L as

$$\sup_{-\infty<a<\infty} P(a < \log U < a+L) \downarrow 0; \tag{D5}$$

that is, the probability that $\log U$ is found in an interval of fixed length decreases uniformly to zero. This property is satisfied when U has the gamma distribution (which leads to May's equation) and $k \downarrow 0$.

No matter how δ changes with the distribution of U, δU must satisfy (D5) in place of U, whenever U satisfies (D5). It follows that $\delta Ue^{-\delta U}$ converges in distribution to zero; being bounded, its expected value must also converge to zero. Thus, inequality (D4) is eventually satisfied when skewness is increased in this way, and equation (D1) becomes locally stable.

If a definition of increasing skewness other than (D5) is used, simple counterexamples show that (D4) need not be satisfied. For example, replacing $\log U$ by U in (D5) does not provide a definition of increasing skewness that necessarily leads to the local stability of (D1).

Now we show that the aggregation model with $g(H_j) = c(H_j/\bar{H})^\theta$ satisfies the skewness condition for large enough θ. For this case, $\bar{h}(u) = bu^{1/\theta}$, for some constant b. It follows that

$$p(u) = \theta^{-1}b^2u^{2/\theta-1}\phi(bu^{1/\theta}) \tag{D6}$$

and the density of $\log U$ is therefore equal to

$$b^2\theta^{-1}e^{2v/\theta}\phi(be^{v/\theta}). \tag{D7}$$

Introducing the mild condition on the density ϕ that

$$\sup_v v^2\phi(v) < \infty, \tag{D8}$$

which is satisfied by almost all probability densities with any practical utility, we see that (D7) $\to 0$ uniformly in x as $\theta \to \infty$. Consequently, (D5) is satisfied, for θ sufficiently large, resulting in a locally stable equilibrium for the model of aggregation in response to relative host density with $g(H_j) = c(H_j/\bar{H})^\theta$. (That is, in the model, the parasitoids are attracted to a patch because of its density of hosts relative to the average density of hosts in all patches.)

The above demonstration assumes that θ is positive. If θ is negative, causing greater risk for hosts in patches of low host density, then θ^{-1} must be replaced by $|\theta^{-1}|$ above, and it is clear that the skewness condition for stability is achieved as $|\theta| \to \infty$.

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