HOST-PATHOGEN CYCLES IN SELF-REGULATED FOREST INSECT SYSTEMS: RESOLVING CONFLICTING PREDICTIONS

Anderson and May (1981) show that substituting realistic parameter values into a host-pathogen model with free-living infective stages (their model G) suggests that cyclic fluctuations observed in empirical studies of insect host-pathogen systems (Baltensweiler 1964; Varley et al. 1973) may be generated by the host-pathogen interactions alone. Recently, two studies have adapted model G to include the additional biological realism of host self-regulation (Bowers et al. 1993; Dwyer 1994). The results of these two studies are in conflict. Bowers et al. suggest that self-regulation reduces the likelihood of cycles, whereas Dwyer suggests that the likelihood is greatly increased. Here we resolve the confusion caused by the conflicting predictions.

First, we recall model G of Anderson and May (1981):

$$\frac{dH}{dt} = rH - \alpha Y, \tag{1}$$

$$\frac{dY}{dt} = \nu W(H - Y) - (\alpha + b + \gamma)Y, \tag{2}$$

and

$$\frac{dW}{dt} = \lambda Y - (\mu + \nu H)W. \tag{3}$$

Equations (1)–(3) model the dynamics of a host of total density H, within which the density of infected individuals is Y, and of a pathogen with a population of free-living infective stages of density W. The parameters r and b are the intrinsic rate of net increase and death rate, respectively; ν is the transmission coefficient of the disease; α is the rate of disease-induced mortality; λ is the rate at which free-living stages are produced by the host; and μ is the decay rate of these stages. It is common practice to set the recovery rate of hosts $\gamma = 0$, since, for insects, infection is generally fatal. For certain parameter values, this model produces cyclic behavior.

Bowers et al. (1993) additionally include a density-dependent term, which models the effect of resource limitation on the host birth rate, in a manner linearly proportional to host density. Thus, their model is as follows:

$$\frac{dH}{dt} = rH\left(1 - \frac{H}{K}\right) - \alpha Y, \tag{4}$$

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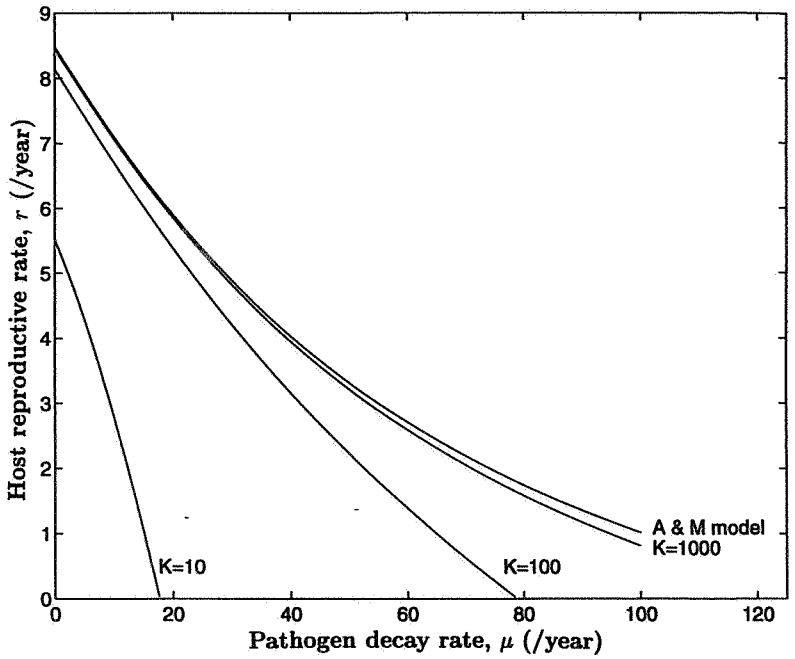

FIG. 1.—Regions in r-μ parameter space for which cycles are expected (*below the lines*), for the model of Bowers et al. (1993). Other parameters are $b = 4\ \text{yr}^{-1}$ and, from Dwyer (1994), $\alpha = 24\ \text{yr}^{-1}$, $\lambda = 2.4 \times 10^9\ \text{yr}^{-1}$, and $\dot{\nu} = 1.4 \times 10^{-7}\ \text{m}^2\ \text{yr}^{-1}$. Increasing K increases the region where cycles are expected, and as $K \to \infty$, the region tends toward that for Anderson and May's model G. It is clear that self-regulation reduces the likelihood of cycles.

$$\frac{dY}{dt} = \nu W(H - Y) - (\alpha + b + \gamma)Y, \tag{5}$$

and

$$\frac{dW}{dt} = \lambda Y - (\mu + \nu H)W. \tag{6}$$

Here, K is the carrying capacity. Figure 1 shows how the inclusion of self-regulation reduces the region in r-μ parameter space where cyclic dynamics are expected. As K increases, this cyclic region increases and approaches the region predicted by the Anderson and May model in the limit as $K \to \infty$ (see Bowers et al. 1993 for a similar result in λ-α parameter space). Thus, as $K \to \infty$ (i.e., when self-regulation is absent), the model of Anderson and May, equations (1)–(3), is recovered exactly.

The adaptation of the Anderson and May model applied by Dwyer (1994) includes density-dependent host reproduction and is as follows:

$$\frac{dX}{dt} = r\left(1 - \frac{X + Y}{K}\right)X - \nu WX, \tag{7}$$

$$\frac{dY}{dt} = \nu WX - \left(\alpha + r\frac{X+Y}{K}\right)Y, \tag{8}$$

and

$$\frac{dW}{dt} = \lambda Y - \mu W. \tag{9}$$

Here, X is the density of susceptible individuals, so that, by combining equations (7) and (8) since $H = X + Y$, we find that the equation for total host density is

$$\frac{dH}{dt} = rH\left(1 - \frac{H}{K}\right) - (\alpha + r)Y. \tag{10}$$

The system can be represented by equations (8)–(10) (with $X = H - Y$). In addition to the inclusion of self-regulation, Dwyer assumes that the natural, density-independent death rate $b = 0$; hence, r refers to birth only. However, density-dependent mortality affects both the susceptible and the infected classes. Also, births stem from the susceptible class only (eq. [7]), and consumption of the pathogen by the host is regarded as negligible in equation (9). These differences, however, are only significant because Dwyer makes a direct comparison with Anderson and May's model (1)–(3) and concludes that self-regulation greatly increases the likelihood of population cycles. However, this comparison is flawed since in the limit as $K \to \infty$, we do not recover equations (1)–(3) from equations (8)–(10). The limit for Dwyer's model is as follows:

$$\frac{dH}{dt} = r(H - Y) - \alpha Y, \tag{11}$$

$$\frac{dY}{dt} = \nu W(H - Y) - \alpha Y, \tag{12}$$

and

$$\frac{dW}{dt} = \lambda Y - \mu W. \tag{13}$$

This model is significantly different from the Anderson and May original, and it is with this that the comparison should be made.

An algebraic analysis of the Jacobian of the true limit model, equations (11)–(13), reveals that the coexistence equilibrium has one real negative eigenvalue and a complex pair with positive real part, for all parameter combinations. This indicates that cycles are expected in the whole of r-μ parameter space, a result supported by numerical studies and that, when compared with the results of

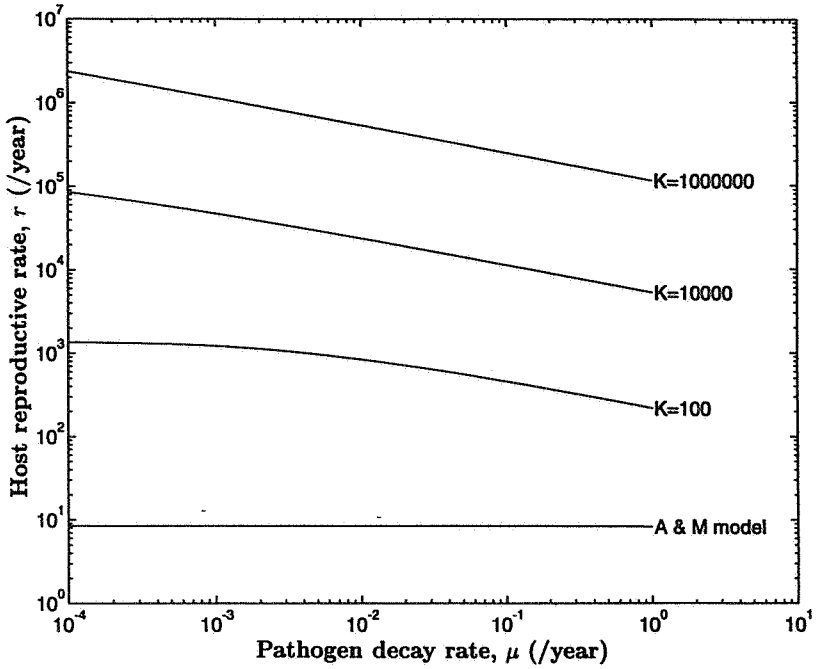

FIG. 2.—Regions in r-μ parameter space for which cycles are expected (*below the lines*), for the model of Dwyer (1994). Parameters are as in figure 1 with the exception of $b = 0$. The region where cycles are expected increases enormously as K increases and diverges from the region predicted by the Anderson and May model, to which Dwyer makes a flawed comparison. The limit model for Dwyer's equations predicts cycles in the whole of r-μ parameter space, and this limit is approached as $K \to \infty$. Again self-regulation reduces the likelihood of cycles.

Dwyer's model, suggests that cycles are again less likely when host self-regulation is included (fig. 2).

Thus, if Dwyer's (1994) model is compared with its true limit ($K \to \infty$), it agrees with the suggestion of Bowers et al. (1993) that the inclusion of host self-regulation decreases the likelihood of coupled host-pathogen cycles.

The underlying question raised by the above work is perhaps whether adding biological realism to Anderson and May's (1981) basic model G strengthens their conclusion that host-pathogen interactions can drive cycles of invertebrate abundance in nature. As we have seen, the addition of biological realism in the form of a particular type of host self-regulation decreases the likelihood of cycles and hence does not strengthen Anderson and May's claim. What, then, can be said about the effects of adding other forms of biological detail? Dwyer's (1994) model, including the limiting case without self-regulation, differs in other aspects from the model of Bowers et al. (1993) and that of Anderson and May (1981), on which the Bowers et al. model is based. In particular, if we assume that the death rate due to the disease is much greater than the natural death rate of the host and that consumption of the pathogen by the host is negligible as a source of pathogen

mortality, then the significant difference between Anderson and May's (1981) model (eq. [1]–[3]) and that of Dwyer (1994) is that infected hosts do not reproduce in Dwyer's formulation. This may be more realistic, since in forest insect systems the infection typically occurs in the larval stages and causes death before maturation. This occurrence may increase the tendency to cycle, since when susceptible host densities are depleted, births into the susceptible class from the relatively large numbers of infecteds, to dampen the decline in the density of susceptibles, will not occur. Additionally, the region of parameter space where cycles are predicted is much greater for Dwyer's model. Hence, this time extra biological realism does strengthen Anderson and May's claim. A similar conclusion was reached by Anderson and May (1981). Thus, the addition of individual factors to the Anderson and May model to enhance biological realism can produce tendencies either increasing or decreasing the likelihood of cycles. However, as far as the conflict resolved in this note is concerned, the answer is unequivocal: self-regulation of the type employed by Bowers et al. (1993) and Dwyer (1994) decreases this likelihood and does not strengthen Anderson and May's conclusion.

Finally, it is important to investigate the possible correspondence between the cycles predicted by the models and those observed in the field. Note here that difficulties in correlating various model predictions and data suggest that, in part, better empirical data may be required to fully understand the mechanisms at work. However, to return to models and their applications to available data, Bowers et al. (1993) provide evidence that parameters estimated for such systems may fail to generate cycles at all. Moreover, even where cycles are generated, while the cycle period often corresponds well with field data, disease prevalence tends to peak at a much higher value than those generally observed in the field, and host density in the models peaks at values lower than the host's carrying capacity, whereas field data suggest that carrying capacities are frequently exceeded. This situation would be equally true for Dwyer's (1994) model, since the self-regulation there, too, does not allow the carrying capacity to be exceeded. On the other hand, models with more complex forms of self-regulation, perhaps with a time delay, would allow cycles to peak at values above the carrying capacity and may therefore come closer to matching all aspects of the cycles observed in nature. Thus, while host-pathogen interactions may play an important role in determining the details of population cycles in forest insects, the cycles observed seem certain to reflect the interaction between this and other factors (Bowers et al. 1993).

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ANDREW WHITE*
ROGER G. BOWERS
DEPARTMENT OF APPLIED MATHEMATICS AND THEORETICAL PHYSICS
LIVERPOOL UNIVERSITY
P.O. BOX 147
LIVERPOOL L69 3BX
UNITED KINGDOM

MICHAEL BEGON
DEPARTMENT OF ENVIRONMENTAL AND EVOLUTIONARY BIOLOGY
LIVERPOOL UNIVERSITY
P.O. BOX 147
LIVERPOOL L69 3BX
UNITED KINGDOM

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*E-mail: aw11@liverpool.ac.uk.

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