Ecology, 75(2), 1994, pp. 271–287

POPULATION REGULATION IN THEORY AND PRACTICE

THE ROBERT H. MACARTHUR AWARD LECTURE
Presented August 1991
San Antonio, Texas, USA

by

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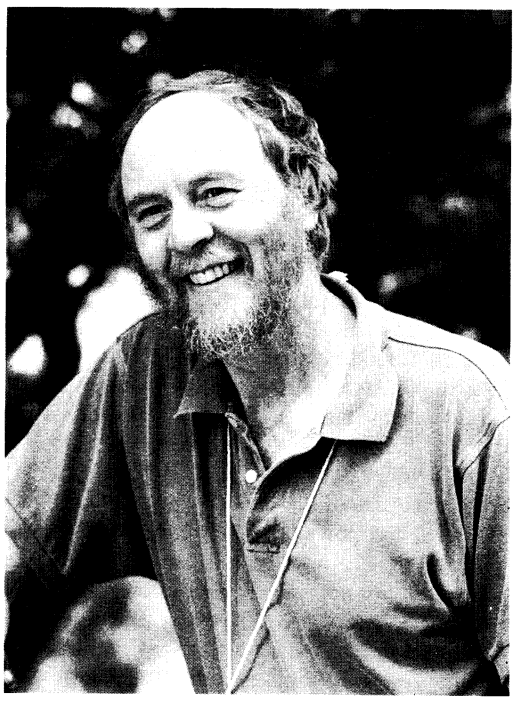

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Abstract. Population regulation is a fundamental process related to most phenomena in ecology, including evolutionary ecology. I review the conceptual basis for defining regulation as bounded fluctuations in abundance, in contrast to the unbounded fluctuations of random-walk populations. Regulation arises as a result of potentially stabilizing density-dependent processes, even when brought about by "non-equilibrium" mechanisms. Although the phenomenon is unambiguous in theory, detecting regulation by finding evidence for density dependence in a series of population estimates faces unsolved statistical problems. So, while there is growing evidence for widespread regulation, severe detection problems remain. I illustrate these with data from bird populations. Whether regulation is typically achieved by local stabilizing mechanisms or via metapopulation dynamics remains to be determined.

I summarize recent studies on a particularly well-regulated system—red scale (*Aonidiella aurantii*) and its controlling parasitoid, *Aphytis melinus*. We tested and failed to find evidence for eight hypotheses that might account for the system's stability, including spatial heterogeneity in attack rates, a refuge, and metapopulation dynamics. We also failed to find evidence for density-dependent parasitism, but such density dependence might be still be present. Recent laboratory and modeling studies have uncovered a number of other potentially stabilizing mechanisms centering on the response of individual *Aphytis* to their size-structured host.

This plethora of size- and stage-dependent interactions leads naturally to a consideration

of the factors controlling *Aphytis*' size-dependent behavioral decisions, and consequently to the elaboration of size-structured models. The latter provide a vehicle for bringing together investigations of selection of life histories, and population dynamics. This is illustrated by a model of *Aphytis* and red scale dynamics that can explain a dramatic case of competitive displacement.

The red scale/*Aphytis* system exemplifies a particularly challenging problem in population regulation, namely to account for the co-occurrence of stability and severe suppression of the prey population. A potentially generic solution is to assume stabilizing density dependence in the parasitoid or predator population; however, this has the consequence of increasing the host or prey population equilibrium. My colleagues and I have shown that observed prey densities in a plankton system are too low for such a mechanism to be operating. Further work is needed to test this and other hypotheses.

Key words: biological control; evolution; insects; life histories; models; parasitoids; population; predators; regulation; stability.

INTRODUCTION

Population regulation and the mechanisms that bring it about have been the subjects of debate and quantitative study in ecology for much of this century. Arguments over its relevance persist, however, and we have few examples in which the regulatory processes have been demonstrated unambiguously. Shifts in emphasis in ecology, for example to niche theory and other aspects of community structure and dynamics, have been to some extent attempts to finesse this difficult problem. Indeed, the study of regulation has gone out of fashion, especially among U.S. ecologists (Krebs 1992), so a comment on its continuing centrality is appropriate.

Population regulation underlies most other ecological problems of interest, such as the dynamics of diseases, competition, and the structure and dynamics of communities. It is also integral to much that is of interest to evolutionary biologists since the major natural selective forces shaping life histories and behavior, for example, also affect population dynamics. Less obviously, perhaps, variants in the outcome of regulation, e.g., stability and population cycles, bear directly on the assumptions and range of application of evolutionary models (Murdoch et al. 1992*b*).

A focus on regulation is apparently at odds with the recent emphasis on non-equilibrium dynamics (e.g., Botkin 1990). Indeed, Krebs (1991) argues that the intimately related notion of density dependence is a bankrupt approach. I therefore begin by discussing why, in my view, regulation is essential to long-term species persistence and why population regulation is still the central dynamical question in ecology. In the process I will try to sharpen the concept of regulation. I then summarize efforts to find the regulatory mechanisms in a particular set of populations: those of California red scale under control by the parasitoid *Aphytis,* a system in which the issue of stability is particularly acute. Finally, that example will motivate some comments on the relationship between population dynamics and evolutionary ecology.

A comment on "non-equilibrium" dynamics is perhaps in order. In the context of population regulation the term seems typically to be used for systems in which local populations appear to go extinct with some frequency. But it also is used sometimes to describe systems in which environmental change, disturbance, and large-amplitude fluctuations occur, a less appropriate usage in my view (Murdoch 1991). The general implication, pursued below, is that the population is not responding to an equilibrium density.

POPULATION PERSISTENCE AND REGULATION

In this section I first examine the theory of regulation, emphasizing the significance of both local and regional dynamics. I then look at a few efforts to determine whether regulation is widespread in real populations.

Conceptual framework

The notion of equilibrium is central to regulation, and we therefore need to inquire into the idea that populations can persist via the operation of so-called "non-equilibrium" dynamics, in which metapopulations are key. I begin by looking at models that describe explicitly changes in abundance of single local populations.

Unregulated local isolate: (1) Non-equilibrium random-walk dynamics (Model 1).—Regulation seems best defined by defining non-regulation, which is random-walk dynamics. Consider therefore the dynamics of an unregulated randomly walking single population unconnected to other populations (i.e., an "isolate"). Its rate of change is defined by

$$\ln N_{t+1} = \ln N_t + r_t, \qquad (1)$$

where the exponential per head rate of change in each generation, r_t, varies at random with mean 0 (i.e., there is no systematic drift) and variance σ^2, and is unrelated to current or previous density. Thus there is no feedback between population density and the per head rate of change.

The key properties of such a population are as follows:

1) Because there is no feedback, the population has no equilibrium density.

2) The fluctuations in density are unbounded; that is, they increase through time without limit (e.g., Fig.

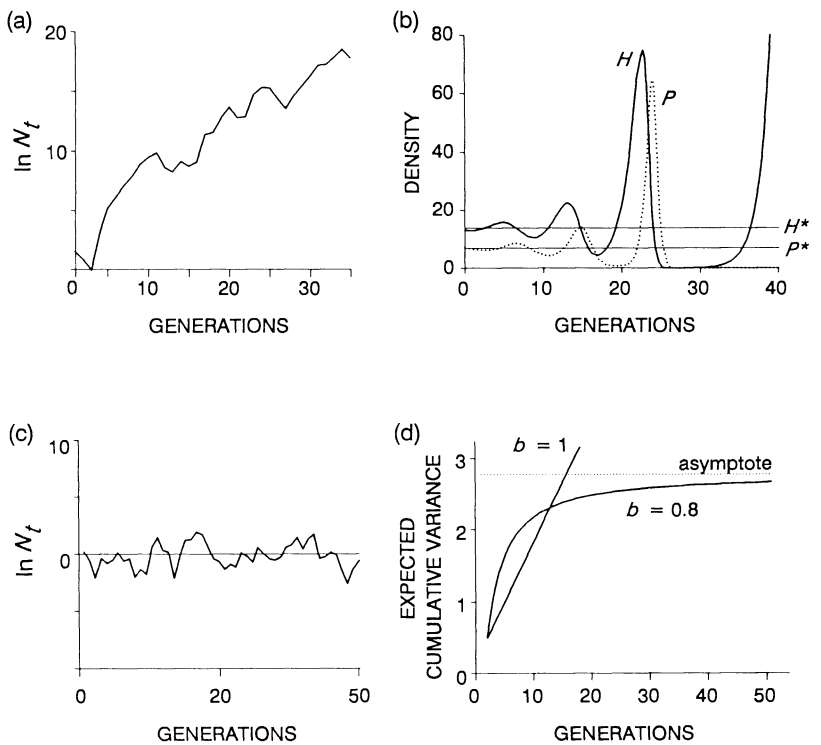

FIG. 1. Illustration of various types of unregulated and regulated population dynamics in local populations ("isolates") unconnected to other populations of the same type, and a practical criterion for regulation. (a) Simulation of an unregulated population that is randomly walking, has no equilibrium, and shows unbounded fluctuations (Model 1). (b) Unregulated dynamics of the host population in a Nicholson-Bailey host–parasitoid system in which there is an unstable equilibrium (Model 2). (c) Simulation of a regulated population whose dynamics are determined by Model 3 ($b = 0.8$). (d) The expected behavior of the cumulative variance of unregulated (unbounded) populations ($b = 1$) and regulated (bounded) populations ($b = 0.8$).

1a). This property of unboundedness can be used to define non-regulation, and hence we can identify regulation with boundedness. Regulated populations thus include not only those with a stable equilibrium but also those with a stable attractor, i.e., cyclic or chaotic populations.

This model has important implications for a "non-equilibrium" theory that relies on persistence via the ensemble dynamics of a collection of non-equilibrial subpopulations: the number of organisms in a metapopulation consisting of a collection of subpopulations, each of which obeys Model 1, in heterogeneous space and linked by random movement, also randomly walks (Chesson 1981, Klinkhammer et al. 1983). Such a collection of subpopulations thus has no equilibrium density and is unbounded and unregulated. This type of non-equilibrium dynamics therefore apparently cannot account for the general persistence of species.

Unregulated local isolate: (2) Equilibrium but unbounded dynamics (Model 2).—Next consider a second class of unregulated single isolated populations, illustrated by the Nicholson–Bailey model for the interaction between insect hosts and parasitoids. In this model, $P(t)$ parasitoids search at random in generation t at instantaneous per head rate a for $H(t)$ hosts so that the fraction surviving is $\exp[-aP(t)]$. If each parasitized host gives rise to a single female parasitoid in the next generation, and in the absence of parasitism the hosts increase at a per head rate, F, we have

$$H(t + 1) = FH(t)e^{-aP(t)} \tag{2a}$$

$$P(t + 1) = H(t)[1 - e^{-aP(t)}]. \tag{2b}$$

Property (1), above, has now changed. In this case there is feedback between the per head rate of change of each population and its previous density. For example, the per head rate of change of hosts in generation $t + 1$ depends on parasitoid density in generation t, which itself depends on host density in generation $t - 1$. This feedback creates an equilibrium. However, there are no stabilizing mechanisms operating and not only is the equilibrium unstable, but fluctuations in density increase through time without limit (Fig. 1b), i.e., they are unbounded. That is, isolated local populations are unregulated.

Parenthetically, this model can resolve confusion in discussions of density dependence (Reeve 1988, Murdoch and Walde 1989). Some theoreticians define Model 2 as having density dependence in the per head rates of population change (e.g., H_{t+1}/H_t), because of

the just-described feedback between them and previous density; I will follow this convention hereafter. (The feedback is peculiar in that H_{t+1}/H_t responds to P_t, and hence H_{t-1}, rather than to H_t.) However, to others including many field ecologists, density dependence necessarily implies that the population's vital rates are functions of its density; the latter type of density dependence can be stabilizing. It is therefore useful to distinguish density dependence in per head rates of change that serves merely to create an equilibrium and is not potentially stabilizing, as seen in the Nicholson–Bailey model, from potentially stabilizing density-dependent mechanisms. The latter are absent from Models 1 and 2.

In contrast with Model 1, however, a metapopulation in which each subpopulation has Model 2 dynamics can be regulated if the various subpopulations exist in a spatially heterogeneous environment and if they are linked by random movement. Indeed, such a metapopulation can be stable (Reeve 1988). The new potentially stabilizing density-dependent mechanism that regulates such a metapopulation appears to be immigration to each subpopulation. The density dependence appears to arise in the following way. Since the number of immigrants coming from other subpopulations that are fluctuating out of phase with the recipient population is independent of the number in the local population, the number arriving per resident decreases with the number of residents (e.g., Murdoch et al. 1992*a*).

The key difference between Models 1 and 2 is the presence in Model 2 of feedback that creates an equilibrium. Thus, while persistence of a metapopulation of such subpopulations is often thought of as non-equilibrium dynamics, in fact the existence of a local equilibrium in each subpopulation appears to be essential for regulation.

Regulated local isolate (Model 3).—Finally, consider the simplest model of a regulated single population. We modify Model 1 in the standard way by rewriting the equation to describe the difference between log density at time t and the mean log density, μ, and by introducing potentially stabilizing density dependence in the local population via the parameter b and the requirement $b < 1$:

$$\ln N_{t+1} - \mu = b(\ln N_t - \mu) + r_t, \text{ where } b < 1. \quad (3)$$

There is still random variation in the per head rate of increase, but the fluctuations are now bounded around a constant equilibrium (Fig. 1c).

Strong's ideas on density vagueness (Strong 1986) illustrate an approach that at first sight may not fit within this general framework. However, Strong assumes there are "ceilings" and "floors" to density, which certainly is consistent with our definition of a regulated population as one showing bounded fluctuations. Density vagueness implies a weak relationship between per head rates of change and density that is masked by a large random component. The idea has not been set in a formal mathematical framework, but it does not seem inconsistent with the existence of an equilibrium density in an isolate, even if the population only fleetingly passes through the equilibrium and only weakly responds to the difference between density and equilibrium level. If, however, over most of the density range there truly is no relationship between per head rates of change and density, it may be more appropriate to couch the problem in terms of stochastic analogs: the stationary distribution of densities of extant populations, and stochastic boundedness (Chesson 1978).

Patch models.—Somewhat different considerations apply in patch models, which provide an alternative framework in which a population on a patch can go extinct and patches can be recolonized. Although these are considered metapopulation models, they are analogs of single-population models in which patches are equivalent to individuals (Hastings and Wolin 1989, Hastings 1991). Typically, local dynamics are ignored and only the presence or absence of the species in "habitat patches" is considered. Hastings (1991) describes a model with essentially these features, but which moves in the direction of including local dynamics by describing the distribution of patches of a given age and, equivalently, density. In Hastings' model a population in a patch is born, ages and grows, reproduces, and dies probabilistically. In these patch models there is no local equilibrium condition of a patch, just as an individual cannot be said to be at equilibrium in a population model. Gyllenberg and Hanski (1992) develop a generalized framework for metapopulation models in which the dynamics of local populations can be described and then show how such a model can be reduced to a true patch model lacking local dynamics.

Although patch models do not contain equilibria at the level of individual patches, they are nevertheless equilibrium models, and the rules for regulation of a single population transfer. For persistence of the population of patches there needs to be an equilibrium number (or fraction) of occupied patches and a potentially stabilizing process. For example, Hastings and Wolin (1989) present a model whose equilibrium is stabilized by a logistic-like density-dependent "birth rate" of patches.

The main point I wish to draw from this brief survey is that the "non-equilibrium" approach has not changed the fundamental question that must be asked about species persistence: that remains, How are populations regulated? Rather it has expanded the kind of answer that is possible. The classical view has been that regulation is a result of locally generated potentially stabilizing mechanisms, i.e., mechanisms that occur within the local subpopulation. The alternative possibility is regulation in the absence of such mechanisms but through the presence of globally generated, potentially stabilizing mechanisms involving spatial heterogeneity and movement. Thus, paradoxically, species persistence through so-called "non-equilibrium" dynamics

requires the existence of an equilibrium or equilibria and causes regulation through potential stabilizing mechanisms, albeit of a unique type. In particular, a collection of subpopulations, each of which in isolation lacks stabilizing mechanisms and is unregulated, can persist when immigration from other subpopulations acts as a local stabilizing process.

One caveat to this conclusion is as follows. While randomly walking populations must go extinct, the passage time to extinction under some circumstances can be extremely long (Nisbet and Gurney 1982: Chapters 6 and 7). For example, Middleton (1993) and I. Hanski et al. (*unpublished manuscript*) show that randomly walking populations with upward drift and a density "ceiling" can persist for effectively infinitely long periods even though they must eventually go extinct. The appropriate question in this context may then be whether such a model can account for the general persistence of species in this way, a question I raise in another context below. That is, we need to determine the distribution of passage times to extinction in a model with realistic values for the rates of increase and decrease that would be observed in populations lacking density dependence (D. A. J. Middleton et al., *unpublished manuscript*).

Detecting regulation: tests for density dependence

The above theory suggests persistent species must consist of regulated single populations or regulated collections of subpopulations. Regulation should therefore be ubiquitous in natural populations, provided we look for it at the appropriate spatial scale (Murdoch et al. 1985: Fig. 1).

The standard approaches to testing for regulation look for the presence of stabilizing density dependence in a time series of estimated population densities. Usually it is assumed that the observed series is produced by underlying dynamics that are well-enough approximated by Model 3 above, though Pollard et al. (1987) modify the test to take account of series that show drift because the initial population value is far from the equilibrium value. The tests ask whether $b < 1$.

This approach and its difficulties have been thoroughly discussed, for example, by Bulmer (1975), Pollard et al. (1987), Turchin and Taylor (1992), and Woiwood and Hanski (1992). Two main problems are that the tests frequently fail to detect density dependence even in data generated by a density-dependent model (e.g., Pollard et al. 1987), and that they are prone to detect density dependence where it does not occur. The second problem is exacerbated in real data since sampling error increases the chance of finding spurious density dependence. (Where ε_t is sampling error, the covariance between initial density and rate of change is $\text{cov}[N_t + \varepsilon_t, (N_{t+1} + \varepsilon_{t+1}) - (N_t + \varepsilon_t)] = \text{cov}(\varepsilon_t, -\varepsilon_t) = -\text{var}(\varepsilon_t) < 0$.) In addition, a model such as Model 3 is clearly a gross approximation to real dynamics, especially where there are complex time lags and/or more or less continuous dynamics with overlapping generations (Turchin and Taylor 1992, Woiwood and Hanski 1992).

A major implication from these points is that little can be inferred from a statistical analysis of the time series of a single population. Thus, if we are interested in exploring regulation in a particular population we need to investigate the mechanisms directly. The second part of this paper is devoted to an example.

We are perhaps less likely to be misled by testing for density dependence in groups of populations. For example, Woiwood and Hanski (1992) analyzed several thousand time series of moth and aphid species collected in traps at various sites in Britain. Using three of the standard methods, they found density dependence in 57–80% of the moth series and in 84–88% of the aphid series when analyses were restricted to series of 20 yr or more. While we cannot be certain that these results are not contaminated by sampling error or bias in the estimators, they are encouraging. Again, Turchin (1990) and Turchin and Taylor (1992) found density dependence in the great majority of populations analyzed by methods designed to detect delayed as well as direct density dependence, and cyclic and chaotic as well as stable dynamics. The two studies found density dependence in all but 1 of 19 forest insect populations and in all but 3 of 22 vertebrate populations. Their methods, however, also bring along new problems in statistical interpretation (Turchin and Taylor 1992).

In spite of these apparent successes, serious problems remain. They are well illustrated by bird populations, which should be well represented among those showing regulation. We know from the wealth of studies on birds that density-dependent mechanisms, such as territoriality, are common. Furthermore, birds counts are among the least variable recorded, suggesting tight regulation. Yet it has been difficult to find evidence for density dependence in their time series.

Greenwood and Baillie (1991), for example, found virtually no statistically significant cases when they analyzed counts of 39 species of British passerines, each over at least 20 yr. Yet we would expect to find evidence for density dependence in these data. Even if metapopulation dynamics were important, counts were made in plots distributed throughout the country (Marchant et al. 1990) and should therefore have captured regional effects, and by any standards the populations have been remarkably constant.

I have analyzed data from a larger but still quite homogeneous set of these data: 92 time series, taken over 20–27 yr, from 59 species of birds living in nonaquatic environments. Some species were counted in woodland, others in farmland, and others in both. The average maximum/minimum ratio of abundance was only 3.3 and the ratio was <3 in more than half the series. I looked for density dependence using the test developed by Pollard et al. (1987). The test calculates

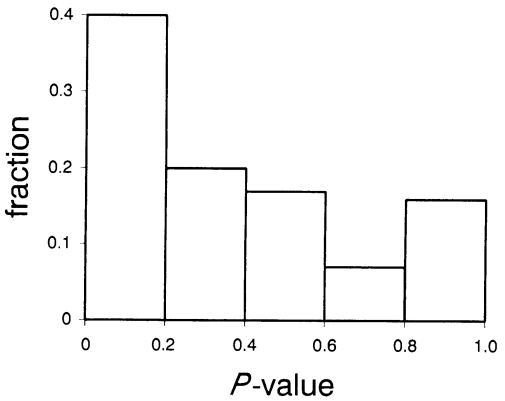

FIG. 2. The frequency distribution of P values calculated using the Pollard test for density dependence in 92 series of population counts of breeding British birds.

the correlation coefficient between the change (d_t) in log density between each successive pair of years, and the log of the density in the first of each pair. Keeping the first observation in the series fixed, a random ordering of the d_t is then chosen to generate a new series of data and the correlation coefficient is recalculated. I repeated this procedure 10 000 times for each observed series. The significance level of the original correlation coefficient is then the fraction of the 10 000 orderings that yielded a more negative value of the correlation coefficient than that calculated from the observed data.

Only 17 (18%) of the series show evidence for density dependence at the 5% probability level, compared with roughly five cases expected by chance. An alternative test (Bulmer 1975) gave only 15 cases. Furthermore, although the frequency distribution of the observed P values is by no means flat, as would be expected if populations were fluctuating at random, a very substantial proportion of the time series is far from showing statistically significant evidence for density dependence (Fig. 2). Thus, it appears as though density dependence is uncommon in the series.

It has been recognized for some time that trends in population density may mask density dependence, and Greenwood and Baillie (1991) suggested this might be the case in these populations. Trends certainly contribute. Seventy-four percent of the series show significant trends at the 5% level. (In many cases the trends are detectable statistically, even though they are extremely weak, because there is very little fluctuation around the trend.) Statistically significant evidence for density dependence is found in a higher fraction of the series without trends (33%, 8 out of 24) than in those with trends (13%). However, 33% is still a rather small fraction of series showing evidence for density dependence.

Marchant et al. (1990) provide plausible evidence that many of the trends have been caused by temporal changes in the environment, and in agricultural practices in particular. It is tempting to remove the trends and test for density dependence in the residuals (Greenwood and Baillie 1991), but there are several potential problems. The Pollard test already allows for a trend. The properties (such as autocorrelation) of the residuals would not be well established. Finally, the danger with such post hoc analysis is that one can milk the data until one finds a result to one's liking.

A second possibility is that the very strictness of regulation makes it difficult to detect: the narrow fluctuations do not allow much variation in the x axis when rate of change is correlated against density, and most of the variation might well be sampling error. However, when we analyze the series lacking a trend with time, there is no correlation between the strength of density dependence (measured by the P value since the series are all about equally long) and the amount of variation in (log) density.

The remarkable invariance of these bird populations makes it difficult to believe they are not in fact well regulated. If they are, we clearly have still not developed adequate means of detection.

In summary, although recent analyses, especially of insects, have increased the evidence for regulation, it has by no means been established that regulation is ubiquitous, as we have every reason to expect it to be. The bird data discussed above suggest that the problem may be mainly one of detection.

Detection of regulation: test for boundedness

The theoretical ideas I outlined above suggest a more robust and generic approach to detecting regulation that might also avoid the statistical problems in testing for $b < 1$. In contrast with randomly walking populations, regulated populations like those described by Model 3 show bounded fluctuations. Based on this contrast, Murdoch and Walde (1989) proposed as a criterion for boundedness, and hence regulation, that the cumulative variance of logarithmic population densities should approach an upper bound. Where $n_t = \ln N_t$ and n is the estimated mean of the log densities over T sampling periods, cumulative variance is $s^2 = \Sigma(n_t - n)^2/(T - 1)$. One potential advantage of the approach is that it is model free and should detect regulation whether the population is stable or unstable but cyclic or chaotic.

Unfortunately, this approach contains severe statistical problems. I summarize it briefly in the hope that this may stimulate the appearance of a better generic approach.

The idea can be illustrated by comparing the expected cumulative variance of Models 1 and 3 (Fig. 1d). In random-walk populations, i.e., when $b = 1$, variance of log density is expected to accumulate linearly with time and there is no tendency for the expectation to asymptote. In regulated populations, illustrated by Model 3 with $b < 1$, variance tends to increase with time but to approach an asymptote. So

the idea is to look for a tendency for the cumulative variance to flatten out. A test procedure is first to test for an increase in cumulative variance with time: a zero or negative slope against time is evidence for an asymptote. Second, for series whose cumulative variance increases with time, Thornby (1972) provides a test for whether the curve is asymptoting (i.e., is concave down).

This approach seemed initially promising. For example, over 50% of the British birds series discussed above gave statistically significant evidence for regulation; 35% because the cumulative variance showed no increase with time, and the remainder because cumulative variance increased but approached an asymptote. Unfortunately, these results could easily be spurious: 47% of 10 000 simulations of a randomly walking population, each over 50 time steps, also showed no increase in cumulative variance, and a further 17% showed increasing but asymptotic cumulative variance. Indeed the level of "detection" of density dependence in these random-walk runs (64%) was actually higher than in the real data, presumably because of trends in the real data. Thus the tests have very little power to distinguish regulated from random-walk populations.

The problem seems to be one mentioned in Murdoch and Walde (1989): although the expected cumulative variance of a random walk increases linearly with time (Fig. 1d), the variance of the cumulative variance increases with t^2 (A. Stewart-Oaten, *personal communication*). In particular, a large proportion of runs accumulate variance slowly over long periods, and these appear to be asymptoting: unlike the example in Fig. 1a, many runs exhibit long periods in which abundance fluctuates about a close-to-constant mean. Many of these populations will of course gain variance rapidly if they are run for longer periods, but other populations will then appear to asymptote as their rate of accumulating variance slows. An additional problem is that the successive values of the cumulative variance, plotted against time, are not statistically independent.

Pimm and Redfern (1988) used cumulative variability (actually cumulative standard deviation of log density, henceforth SDLOGS) of population density, not to detect regulation but to suggest that populations do not show bounded fluctuations. They analyzed an earlier version of the British bird data and concluded that most species were trending over the period because the SDLOGS increased with time.

There seem to be two problems here. First, the cumulative variance of a regulated population is in fact expected to increase with time, albeit at a decreasing rate (Fig. 1d). McArdle (1989) made a similar point in relation to SDLOGS. Thus, continually increasing variance is not in and of itself evidence for non-regulation. Second, Pimm and Redfern (1988) plotted SDLOGS against the logarithm of time, which will of course tend to produce a continuing rapid increase in a curve that is asymptoting against time on an arithmetic scale. My analysis above shows that the British bird data show strong evidence for an asymptote when the data are analyzed against linear time. (The same is true for very long records of an index of insect abundances, which was originally plotted against the logarithm of time by Hanski [1990] and hence showed no asymptotic behavior.) Unfortunately, as noted, evidence for an asymptote does not provide statistically reliable evidence for regulation.

To summarize to this point, I have tried to show that finding the stabilizing mechanisms that lead to regulation remains the central question in population dynamics, even when recent ideas about non-equilibrium dynamics are embraced. Recent studies have suggested that regulation may be widespread in real populations. Whether such regulation results from stabilizing mechanisms operating in isolated local populations, or from the action of metapopulation dynamics as postulated by the "non-equilibrium" approach, remains a question to be answered by empirical investigation.

Stability and prey suppression: a search for mechanisms

My colleagues and I have been working for some years on the biological control of California red scale, *Aonidiella aurantii*, an insect pest of citrus, by its parasitoid wasp *Aphytis melinus*. It is a particularly good example in which to study regulation, first because it is an exemplary case of successful, apparently stable biological control that has persisted for decades, and second because it highlights an especially sharp dilemma: the co-occurrence of stability and severe suppression of prey density.

Populations of the red scale/*Aphytis* system are remarkably constant over time. For example, scale sampled by DeBach for 10 yr (20–30 generations of the scale and 50–80 of the parasitoid) in the 1960s in two orchards in southern California fluctuated within narrow bounds, around an apparently constant mean density with no evidence of local extinction (DeBach et al. 1971; P. DeBach, *unpublished data*). More recently we have sampled a scale population that fluctuates over a narrower range than almost all other populations whose temporal variability has been measured (W. W. Murdoch et al., *unpublished manuscript*; see also Reeve and Murdoch 1986).

This apparent marked stability is associated with suppression of red scale by *Aphytis* to probably less than 1% of the densities it achieved before it was brought under control. The co-occurrence of stability and severe prey suppression poses a dilemma: unless they make special assumptions, models in which the prey is suppressed far below its own resource limits predict instability in the form of very large-amplitude fluctuations (e.g., Rosenzweig 1971, Nisbet et al. 1989). No such fluctuations have been seen in the red scale/*Aphy-*

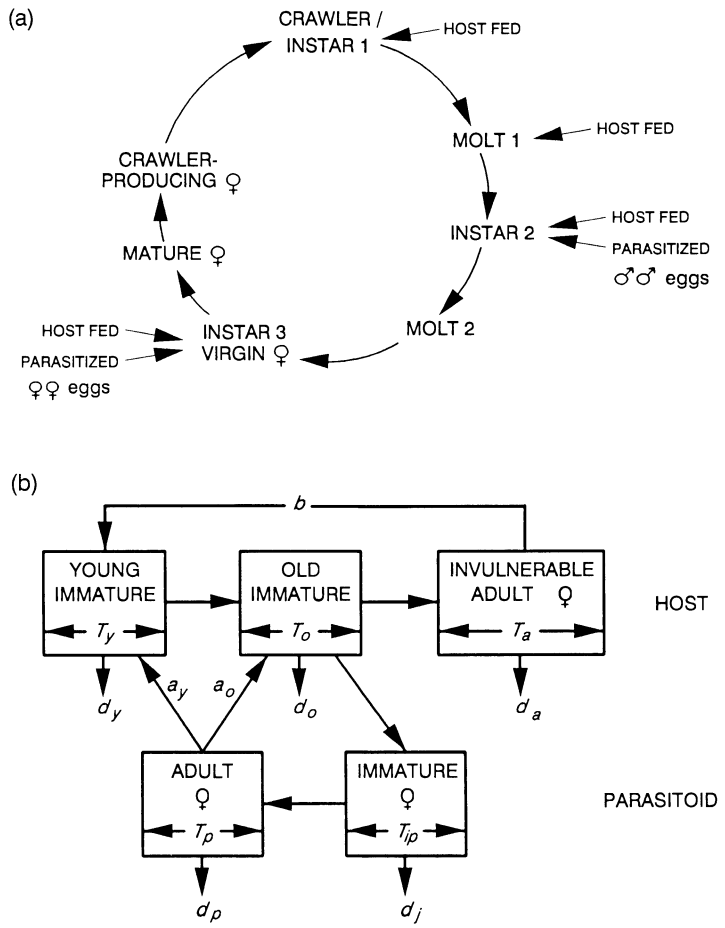

FIG. 3. (a) Diagram of the life history of red scale and pattern of size-dependent attacks by *Aphytis*. All immature stages except molt 2 are vulnerable. (b) Diagram of a model of the *Aphytis*/red scale interaction (Murdoch et al. 1992*b*) that distinguishes between young (or small) immatures, which are attacked but produce no female *Aphytis*, and old (large) immatures, which receive only female *Aphytis* eggs. Adult scale produce crawlers at per head rate b, the immature stages require T_y and T_o d to develop and are attacked at rates a_y and a_o, respectively. The d_i are background death rates. The ratio a_yT_y/a_oT_o measures the relative attack rate on the two stages.

tis system. The central question is therefore how the two phenomena, stability and strong pest suppression, coexist.

A brief synopsis of the life histories is as follows (Fig. 3a). Reproducing adult female red scale produce crawlers that move at most ≈1 m, then settle and insert their mouth parts. Only adult males ever move again. The settlers secrete a protective cover over themselves and the females pass through three instar and two molt stages; males "pupate" after molt 2 and adult males are winged. About two-thirds of the prereproductive life of females (≈400 degree-days) is spent reaching the onset of maturity; i.e., at that point the virgin instar 3 female is ready to be inseminated. For the remainder of the prereproductive life the female is a mature adult that is developing eggs and crawlers, but is not yet producing crawlers. The female can then produce crawlers for 400 degree-days or more. Most stages except the adult are attacked by *Aphytis*.

Aphytis paralyzes the scale and lays one or a few eggs on the scale body under the cover. Smaller immature scale stages tend to receive a male egg or be fed on by the *Aphytis* female, while larger stages (especially instar 3) receive one or a few, mainly female, eggs (Fig. 3a). *Aphytis* develops 2–3 times as fast as scale. The adult female wasp emerges with relatively few (up to ≈12) eggs, but obtains protein from host-feeding that is used to mature more eggs throughout her life.

Initial population study

Our studies of the red scale/*Aphytis* interaction fall into three phases. A 2-yr sampling study in a lemon grove inland from the coast near Santa Barbara, California, tested and rejected four hypotheses for stability in host/parasitoid interactions (Reeve and Murdoch 1985, 1986). These include the notion that the parasitoid aggregates its attacks in space, either (1) in response to local pest density or (2) independent of it.

TABLE 1. Potential stabilizing mechanisms tested and rejected.

Mechanism	Result*	Source†
Parasitoid aggregates to local host density	Mechanism absent[2,3]	a
Aggregation independent of host density	Mechanism absent[2,3]	a
Temporally density-dependent parasitism (also delayed)	Mechanism absent[1,4,5]	b and c
Parasitoid sex-ratio density dependent	Mechanism absent[4]	b
Host-feeding density dependent	Mechanism absent[5]	c
Predation density dependent	Mechanism absent[1,5]	c
Spatial refuge from parasitism	Mechanism present, not stabilizing[6]	c
Metapopulation dynamics	Mechanism absent[6]	c

* Notes. 1. Although no temporal density dependence has been found, it may be present but hard to detect. 2. Analysis of spatial patterns of parasitism in random field samples. 3. Analysis of parasitism of scale outplanted in the field at different densities. 4. Analysis of field samples over time. 5. Analysis of scale outplanted in the field at a single density on different dates. 6. Results of an experiment in which the refuge population was removed and subpopulations (individual trees) were isolated.

† Sources. a. Reeve and Murdoch (1985). b. Reeve and Murdoch (1986). c. W. W. Murdoch et al. (*unpublished manuscript*).

The first result was confirmed using scale "outplanted" in the grove on fruit. Murdoch and Stewart-Oaten (1989) show in any case that whether aggregation is stabilizing in systems with overlapping generations is an open question (see also Godfray and Pacala 1992 and Murdoch et al. 1992*a*). The first study also failed to find either direct or delayed temporal density dependence in (3) parasitism rate or (4) the parasitoid's sex ratio. These results, and those of the second stage, discussed next, are summarized in Table 1.

Experimental test of refuge and metapopulation hypotheses

The second phase was an experimental investigation, in a nearby grapefruit grove, of two other hypotheses to account for stability. First, we speculated that the interaction in the exterior part of the tree was intrinsically unstable but was stabilized by a flow of crawlers from a refuge in the interior of the tree. The tree grows intermittently in one or two flushes per year, and we designate as "exterior" the four most recent flushes; the interior consists of the bark on the internal branches and trunk. We had observed very high densities of scale, and very low parasitism, in the interior region (Reeve and Murdoch 1986). Much theory suggests that a relatively constant refuge that "leaks" recruits to the rest of the population can be stabilizing.

The second hypothesis was that stability might be owing to the operation of metapopulation dynamics, as discussed above (e.g., Murdoch and Oaten 1975, Chesson 1983, Reeve 1988, Hastings and Wolin 1989).

We carried out two studies in this second phase, each lasting ≈ 2 yr. The first showed that the conditions for the refuge mechanism seemed to exist. It confirmed that the interior population was very dense (≈ 100-fold higher than in the exterior); in fact 90% of the crawler-producing females on a tree were in the refuge (Murdoch et al. 1989). The interior is clearly a relative refuge from parasitism by *Aphytis*, which was only $\approx 1/15$ of the level in the exterior. The refuge population was both absolutely very constant, and much more constant than that in the exterior (Fig. 4) (Murdoch et al. 1994). Furthermore, we detected movement of crawlers (using sticky traps wrapped around branches) between the refuge and the exterior.

The second study in this phase tested two hypotheses simultaneously. First, we tested the refuge hypothesis directly, by asking whether fluctuations in the abundance of scale in the exterior of trees whose refuge scale population had been removed was greater than that in control trees. Second, the experiment simultaneously tested the metapopulation hypothesis, i.e., that there are no local stabilizing mechanisms but rather subpopulations in a heterogeneous environment are stabilized by immigration fed by out-of-phase fluctuations. Since crawlers probably remain mainly on the tree on which they are born, the individual tree seems a natural unit for a subpopulation. Half of the trees in the experiment were therefore left as controls and half were isolated from the grove by covering them with fine net cages. Metapopulation models predict that the isolated subpopulations should be less stable, which we took to imply greater temporal variability (Reeve 1988). The experiment thus had a two-way factorial

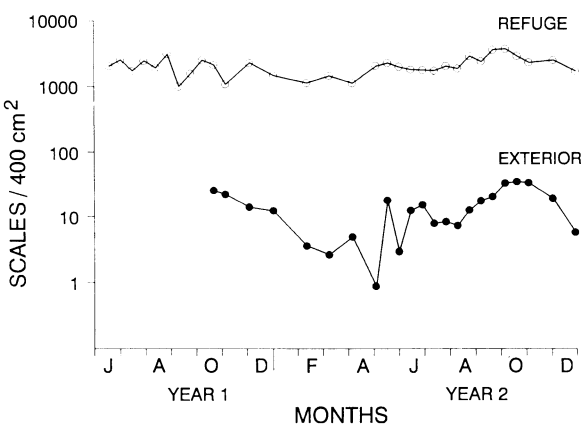

FIG. 4. Densities (log scale) of red scale in the exterior of grapefruit trees, where parasitism by *Aphytis* is relatively high, and in the interior (refuge) where parasitism is low. The latter population is much less temporally variable.

TABLE 2. Factors and mechanisms known to occur in the red scale/*Aphytis* system and their effects on stability in models of the interaction. Unless otherwise noted, mechanisms have been seen in the laboratory and field. Observed stabilizing processes may not be strong enough to account for stability when parameters are assigned realistic values.

Mechanism	Stabilizing in models?	Source
A) Scale size structure and size-dependent attacks		
1. Reproductive adult invulnerable	Yes	Murdoch et al. 1987
2. Prereproductive adult invulnerable	Yes	W. W. Murdoch, *unpublished data*
3. Size-selective host-feeding and sex allocation	Yes and destabilizing	Murdoch et al. 1992*b*
4. More *Aphytis* eggs in larger scale	?	Not yet investigated
5. Scale stage duration varies	Yes	Briggs et al. 1993 and *unpublished manuscript*
B) Flexible responses by *Aphytis*		
1. Host-feeding vs. parasitism on single size class	Neutral	Kidd and Jervis 1989 and Briggs et al., *unpublished manuscript*
2. Host-feeding vs. parasitism on >1 size class	?	Not yet investigated
3. More eggs on larger scale	?	Not yet investigated
C) "Cannibalism" by *Aphytis* (attacks on already parasitized scale)		
1. Host-feeding	Probably	van den Bosch et al. (1988)
2. Egg-killing	Probably	van den Bosch et al. (1988)
3. Superparasitism	Probably	Taylor (1988)

design: uncaged trees with and without a refuge and caged trees with and without a refuge.

In spite of the strong a priori evidence in favor of the refuge hypothesis, our analyses to date suggest the refuge was not stabilizing: temporal variability of the exterior population was not increased when the refuge population was removed (W. W. Murdoch, *unpublished data*). There also appears to have been no effect of metapopulation dynamics: temporal variability in the exterior was not increased by isolating the tree. These results, although negative, provide the crucial information that, whatever mechanism is stabilizing the interaction, it operates on a very local spatial scale.

These studies also failed to find evidence for temporal density dependence in the rates of parasitism, host-feeding, or predation (Murdoch et al. 1994). Thus, an additional four hypotheses have been rejected. Finally, J. D. Reeve et al. (*unpublished manuscript*) show that variation in vulnerability among immature immobile prey can lead to stability. Such variation occurs in scale, but the refuge-removal experiment suggests that it does not affect stability in this case.

Our failure to find evidence for temporal density dependence in mortality of scale (Table 1) is surprising, given that *Aphytis* is widely credited with being the control agent and that the populations are so invariant. Detection is undoubtedly made more difficult by the restricted variation in density, overlapping generations, and complex time delays.

An obvious solution to this problem, in principle, is to perturb scale density. This is not easy in practice, however, with a pest insect! This problem has been made easier since the refuge experiment showed that the stabilizing mechanism(s) operate(s) on a small scale. We are in the middle of a pilot experiment in which scale density has been greatly increased in cages, and it appears that parasitism may indeed be responsive to scale density.

Scale size-structure and size-dependent attacks

The eight hypotheses tested and rejected in the first two phases of the study appear to exhaust the main ones that have been proposed to account for stability. In the third and present phase of the study we have turned therefore to a different class of explanation, which involves the life history details of the scale and the behavior of *Aphytis*.

Table 2 presents a daunting list of mechanisms that might affect the stability of the interaction and summarizes their current status. I will give some detail on those mechanisms we have studied intensively and merely comment on the remainder.

Invulnerable adult stages.—We first examined the idea that an invulnerable adult stage in scale might be stabilizing (Murdoch et al. 1987). *Aphytis* attacks virtually all stages of scale except for the adult females (Fig. 3a), and these might act as a sort of temporal refuge and so stabilize the interaction. The initial stage-structured model collapsed the scale life history into two stages, vulnerable immatures and invulnerable reproductive adults, and recognized immature and adult parasitoids; in each case development to the adult stage takes a fixed time, while the adults die off at constant rates. Each parasitized scale becomes a juvenile female parasitoid; males are ignored since they do not kill scale.

The invulnerable adult stage is indeed potentially stabilizing, but unless the adult scale stage lasts more than twice as long as the immature stage, the equilibrium is unstable for a set of parameter values corresponding roughly to conditions in the field (the single

point in Fig. 5a). In reality the two stages are of roughly equal duration and it seems likely that the invulnerable adult stage may contribute to, but cannot explain stability. Instability is manifested as large-amplitude limit cycles with a Lotka-Volterra-like period of ≈4 times the duration of the immature stage. There is no evidence for such cycles in field populations.

We recently added to this model the invulnerable mature but prereproductive stage (scale that are developing crawlers, Fig. 3a). This is the longest prereproductive stage and, although the model above suggests that invulnerable immature stages should have little effect on stability, our preliminary simulations suggest that it may contribute. This effect, therefore, needs to be investigated in detail.

Size-selective host-feeding, parasitism, and sex allocation.—The basic model pretends that *Aphytis* treats all immature hosts equally, but in fact the parasitoid is highly responsive to differences in size among potential hosts. In particular, young (small) immature scale typically are host fed or, at the upper end of the range, receive a male egg, whereas older (larger) hosts receive mainly female eggs (Fig. 3a). In host-feeding, the scale body fluids are eaten, thus providing protein for maturation of parasitoid eggs, and the scale is killed. The next model incorporates this size-selective parasitism and sex allocation by distinguishing between old and young immature scale (Murdoch et al. 1992*b*). The key to the dynamics of this model is that attacks on old hosts lead to new searching female *Aphytis*, while attacks on young hosts result in host death but do not contribute to the searching (female) parasitoid population (Fig. 3b). We thus treat host-feeding and male production as a single phenomenon, and assume there is no gain to the female parasitoid population from either behavior.

This size-selective behavior also has dramatic dynamical effects because it leads to a kind of delayed density dependence in the parasitoid population. Consider Fig. 3b. As the number of searching females increases, they kill more young scale, fewer survive to become old immatures some time later, and therefore the future per head birth rate of the parasitoid decreases. The number of female parasitoids recruiting per parasitoid to the searching population T_p d later will consequently be reduced. Such density dependence in the parasitoid recruitment rate is potentially stabilizing. However, because the density dependence operates with a time delay, it can also be destabilizing if it is strong enough. The strength of the density dependence is measured by the mortality imposed on young immatures relative to that on old immatures, namely $a_y T_y / a_o T_o$.

Fig. 5b shows that the range of parameter space now showing Lotka-Volterra-like cycles is reduced, giving local stability over a much wider range of parameter values. Given our guesstimate of other parameter values in the field, the real population would now be well into the stable region. It thus seems likely that this mechanism contributes to the absence of Lotka-Volterra-like instability. However, the delay in the density dependence can also lead to a new kind of instability,

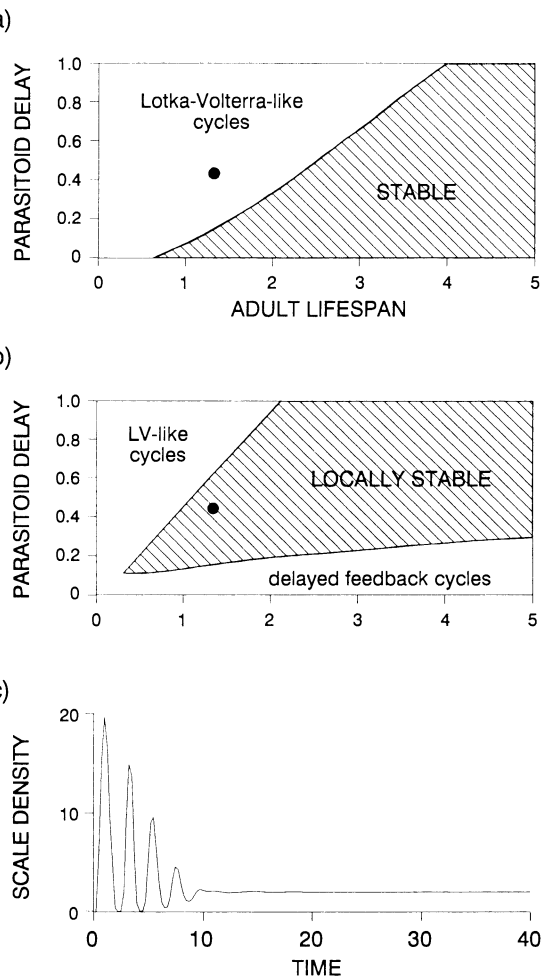

FIG. 5. (a) and (b) show the stability properties of the equilibria in two stage-structured models of the red scale/*Aphytis* interaction as a function of the duration of the adult invulnerable stage and the parasitoid time lag, each expressed as a multiple or fraction of scale development time (modified from Murdoch et al. 1987 and 1992*b*). (a) In the 1987 model there is only a vulnerable immature scale stage and an invulnerable crawler-producing female stage. Typically, stability is more likely the longer the adult stage lives; the limit cycles in the unstable region have a Lotka-Volterra-like period. The dot represents a best guess at where the real system lies in parameter space. (b) In the 1992 model small immatures are host fed by *Aphytis* or receive male eggs, only large immatures receive female parasitoid eggs (see Fig. 3b). The attack rate on small immatures is only 25% of that on large immatures ($a_y T_y / a_o T_o = 0.25$). The unstable area with Lotka-Volterra-like limit cycles has been reduced but a new kind of instability has been created. Over much of the locally stable region large perturbations from equilibrium lead to limit cycles rather than a return to equilibrium. (c) At the point indicated by the dot in (b) perturbations from equilibrium are followed by damped oscillations. Time in degree-days has again been normalized by scale development time.

delayed feedback cycles (Fig. 5b). Furthermore, there is a multiple attractor in part of the locally stable region where large perturbations lead to delayed-feedback cycles, a result seen in an analogous single-species model with cannibalism (Hastings 1987). As the delayed density dependence becomes stronger the area of parameter space occupied by the feedback cycles increases.

If density dependence is weak in the field, the real red scale/*Aphytis* system may lie in a region of parameter space that is both locally and globally stable, as indicated by the point in Fig. 5b. This would lead to damped delayed feedback cycles with a period about twice the immature development period (Fig. 5c). Stronger density dependence would lead to large-amplitude undamped feedback cycles. There is evidence for small-amplitude cycles with almost exactly the predicted period ($\approx$800 degree-days) in field populations, but these appear to exist also in some situations lacking *Aphytis* (Murdoch et al. 1994).

The real red scale/*Aphytis* interaction is apparently stable under a wide range of conditions, whereas stability in the above model seems fragile and holds for only a fairly narrow range of parameter values. It thus seems likely that there are other mechanisms that contribute to the observed stability.

Aphytis responds to scale size not only by varying the kind of attack and sex of egg laid, but by laying more eggs in larger scale (e.g., Luck and Podoler 1985). We have not investigated this effect.

Finally, results of Smith and Mead (1974), Briggs et al. (1993), and others suggests that variation in stage durations of the host should be stabilizing. This is borne out in preliminary modeling of the red scale/*Aphytis* system, though the effect appears to be weak.

Flexible attack behavior by Aphytis

Models of size-structured interactions between hosts and parasitoids have assumed fixed behavior by the parasitoid. However, *Aphytis* responds differently to a given size of host under different circumstances. For example, we have shown in the laboratory that the fraction of attacks on larger scale that lead to host-feeding rather than parasitism decreases as the number of mature eggs carried by the attacking female *Aphytis* increases (Collier et al. 1993). We have also shown that the response to a scale of a given stage or size, e.g., whether the *Aphytis* host-feeds or parasitizes the encountered scale, depends on the presence of other stages or sizes of scale (S. L. Swarbrick and W. W. Murdoch, *unpublished data*). There is therefore potential for complex dependence on both current and previous densities since scale abundances in the field have been influenced by previous scale and parasitoid densities. Flexibility in size-dependent responses may therefore have substantial dynamical effects (Table 2). This work ties nicely into recent ideas on state-dependent optimal behavior by parasitoids (Mangel 1989, Collier et al. 1993).

It appears that flexible host-feeding in response to a single size class of scale, and its physiological consequences such as the gain in future egg production and longevity, do not affect stability (Kidd and Jervis 1989; C. J. Briggs et al., *unpublished model results*). However, I suspect the dependence of the *Aphytis* response on the presence of other scale size classes probably also depends on egg load and may be stabilizing. In addition, T. Collier (*unpublished data*) has shown that clutch size as a function of scale size also depends on egg load. We have not investigated the potential dynamical effects of this flexibility.

"Cannibalism" by Aphytis *(attacks on already-parasitized scale)*

My graduate student Tim Collier has discovered that *Aphytis* is cannibalistic in the laboratory: it may host-feed on already parasitized scale (Table 2). Preliminary data suggest it does so at the same rate as it host-feeds on unparasitized scale. Preliminary modeling shows this to be strongly stabilizing, as might be expected from other models (van den Bosch et al. 1988). We have also recently discovered that *Aphytis* sometimes stabs and kills eggs previously laid in a scale and suspect that this also is potentially stabilizing. Both of these behaviors have shown up in the pilot experiment, mentioned above, in which scale density was locally increased. Finally, superparasitism is a well-known phenomenon with stabilizing potential, and has been seen in the laboratory in *Aphytis*. We have observed it only rarely in the field, however, and the main issue is not its stabilizing capacity but whether it is an ecologically significant event.

Two issues are raised by the evidence for cannibalism. First, to further complicate matters, it is possible that the rate of cannibalism depends on the state of the parasitoid, for example, on its egg load, though we have no evidence on this question.

The second issue has broader implications. While cannibalism is potentially stabilizing, since it induces density dependence in the parasitoid population, this density dependence will likely lead to a lowered per head efficiency as the parasitoid population increases. But as noted in the *Introduction,* the problem we have to account for is the coexistence of stability and severe host suppression, which presumably requires strong responses by the parasitoid population to increases in the host when the latter is at low density. Whether density dependence in the parasitoid can achieve this is not yet clear.

Consideration of the mechanisms discussed in the last three sections and summarized in Table 2 has led us to recognize the existence of many features of the red scale/*Aphytis* interaction that are potentially stabilizing. Indeed, from a dearth of possible explanations for stability at the end of the experimental phase of this work, we have moved to a plethora of possible explanations.

The richness and nature of the possible stabilizing mechanisms, however, raise an interesting problem. We were able previously to test hypotheses singly, either by analysis of sampling data or by simple manipulative field experiments (Table 1). But these approaches probably will not suffice for the mechanisms listed in Table 2. For example, it is not likely to be feasible to alter the vulnerability of various stages or the way in which individual *Aphytis* respond. It is also possible that several processes together act as a sort of "fail-safe." Both of these difficulties suggest the need for well-parameterized models. However, it also seems essential to test predictions of the models, and measure behavior in the field, which can best be done by field experiments such as the pilot mentioned above.

Ratio dependence in the Aphytis *functional response*

I close this section with a brief comment on one potential explanation not mentioned above. Recently it has been suggested that ratio dependence in the consumer provides a universal explanation for the stability of consumer–resource interactions (Berryman 1992), including biological control (Arditi and Berryman 1991). A major difficulty with this idea is that it is purely phenomenological and it has not yet been demonstrated that it arises from biologically reasonable mechanisms; indeed there seem to be severe problems in deriving it from known behavior. It does not seem to arise from interference among individuals in a homogeneous environment (Ruxton et al. 1992), and seems to require, among other problems, contradictions in time scales when based on arguments about spatial heterogeneity (Persson et al. 1992, Oksanen et al. 1992; P. A. Abrams, *unpublished manuscript*).

It is of course possible that a broader phenomenon, namely a functional response that is dependent on predator density (Beddington 1975), can explain stability. This is a phenomenon that does arise as a limit from both individual interference (Ruxton et al. 1992) and from certain kinds of spatial heterogeneity (R. M. Nisbet et al., *unpublished manuscript*), and Ruxton and Gurney (1992) show that the laboratory experiments of Arditi and Saiah (1992) may be consistent with such a predator-dependent functional response. Density dependence in the predator's recruitment or death rates is also a feasible explanation, and indeed I provide evidence above for mechanisms leading to the former.

While ratio dependence and these other biologically more likely predator-dependent mechanisms can in principle account for stability, they raise the difficult dilemma I mentioned above: the price of stability is a decrease in predator efficiency as predator density increases. This in turn leads ineluctably in rich environments to denser prey populations than would otherwise be the case, which may not be consistent with the severe prey suppression observed in systems such as the red scale/*Aphytis* interaction. W. W. Murdoch et al. (*unpublished manuscript*) for example, show for the interaction between *Daphnia* and algae that observed mean algal densities in rich environments are too low to be consistent with the strong consumer density dependence needed to yield stability. This incompatibility between strong resource suppression and consumer density dependence may turn out to be a generic problem.

Size-structured models, life history theory, and ecological processes

Models of size- and stage-dependent behavior of the type discussed above provide a framework for bringing together ideas about the evolution of life history strategies, and their ecological consequences. I illustrate this next with reference to the red scale/*Aphytis* interaction.

The *Aphytis* behavior discussed above seems broadly consistent with Charnov's evolutionary sex-determination theory (Charnov 1982): as predicted, female eggs are laid preferentially in large hosts and male eggs in small hosts. Although the threshold host size for a female egg may vary with the context, there seems to be a strict lower limit to the size of scale that can produce a viable female *Aphytis*. This limit appears to be the key to explaining one of the best documented cases of competitive displacement.

Luck and Podoler (1985) recount how, in less than a decade after its introduction, *Aphytis melinus* replaced *Aphytis lingnanensis* in citrus groves throughout southern California. This rapid replacement was accompanied by improved control of red scale. These authors showed in the laboratory that *A. melinus* can produce viable female offspring in slightly smaller hosts than can *A. lingnanensis*. They argued on the basis of optimality considerations that this should make *A. melinus* the superior competitor, and could therefore explain why it replaced *A. lingnanensis*.

My colleagues and I are investigating the Luck and Podoler (1985) hypothesis in a modification of the model containing size-selective host-feeding and sex allocation. We have added a second parasitoid, *Aphytis lingnanensis*, and divided the immature part of the life history into three stages. The smallest receives male eggs from both parasitoids, the intermediate stage receives females from *A. melinus* but only males from *A. lingnanensis*, while the largest immature receives females from both parasitoids. Thus, for each parasitoid species the model has the form in Fig. 3b, but the threshold age (or equivalently, size) distinguishing "young" and "old" immatures is later for *A. lingnanensis*. We then ask the standard question: When one parasitoid species is at equilibrium, can the other invade and replace it? The answer is, the model predicts that *A. melinus* should win unless *A. lingnanensis* has a much higher attack rate, and furthermore that red scale density should be reduced as a consequence (Fig. 6), which is precisely what the biological control literature claims to have been the case.

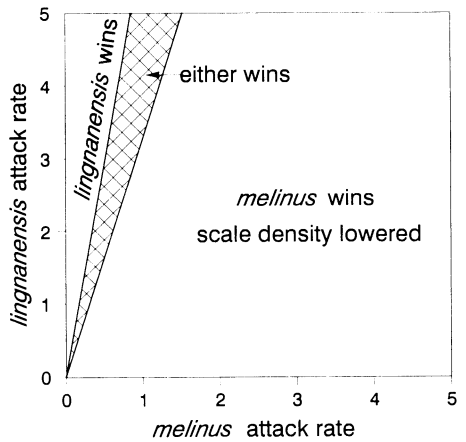

FIG. 6. The outcome of competition between *Aphytis melinus* and *A. lingnanensis* predicted by a stage-structured model.

FUTURE DIRECTIONS

I have tried to show that the decades-old controversy about regulation has been resolved in recent years. Both sides were to some extent correct. The Nicholson school was right that regulation via stabilizing density-dependent processes is essential to account for species persistence: we cannot simply appeal to a concatenation of random events. On the other hand, under very broad conditions that lead to equilibria, the local randomness and spatially out-of-phase dynamics emphasized by the Andrewarthan school can create the necessary stabilizing density dependence. The issue remaining from the controversy is the relative prevalence of locally vs. regionally derived stabilizing mechanisms.

This residuum points to the need to discover the mechanisms responsible for regulation, and for the variety of dynamics shown by regulated populations. In fact, in recent years theory incorporating mechanisms has been developing apace by dealing explicitly with two basic elements: spatial processes and the properties of individual organisms. Yet there are still surprisingly few instances in the field for which we have an unambiguous demonstration of the mechanism(s) leading to regulation. For example, I know of no field example where metapopulation dynamics have been demonstrated experimentally to be essential to regulation. The major challenge for the future is thus to close the gap between theory and experimental test.

Our work on *Aphytis* and red scale, described above, is such an attempt. Unfortunately, its successes so far lie in rejecting, rather than confirming, mechanisms to account for stability.

The red scale/*Aphytis* interaction also illustrates the special problem alluded to earlier: *Aphytis* suppresses red scale density far below the limits set by red scale resources, yet, contrary to the predictions of much resource–consumer theory, does not induce large-amplitude fluctuations in abundance. This problem is not confined to biological control systems. Briggs (1993) has shown experimentally that parasitoids of a gall midge on a coastal scrub plant keep the midge density remarkably constant and at $\approx 1/1000$ of its potential level. Nor is it confined to insects. My colleagues and I have been investigating similarly strong suppression of edible algae by *Daphnia,* an interaction that is also stable (Nisbet et al. 1989; W. W. Murdoch et al., *unpublished manuscript*). Several of the potential stabilizing mechanisms we have discovered in red scale involve density dependence in the parasitoid population, and we need to determine whether they are compatible with the observed strong suppression of red scale.

Finally, the length and particularity of the list of potentially stabilizing mechanisms we have discovered in *Aphytis* (Table 2) raise the issue of generality. We surely hope to find commonality in the explanations for stability coexisting with strong prey suppression across the systems mentioned: scale, gall midges, and *Daphnia,* and in yet other systems that manifest this phenomenon. Yet a number of mechanisms listed hinge, for example, on host stage structure and size-dependent interactions that have no or only partial applicability to these other systems. It would be extremely helpful to get well-tested answers for several of these systems to determine whether generality is likely.

I end with comments on two lines of investigation that show promise for further progress in developing a testable theory of regulation.

Linking population dynamics and evolution through a focus on individuals

Evolutionary studies often investigate how the properties (behavior, physiology, life history traits) of individuals should change in response to selection. But of course these properties are precisely the determinants of a population's vital rates and hence of its dynamics, and ecologists for some time have hoped to incorporate such evolutionary considerations into population models (e.g., Abrams 1982, Sih 1984). In the previous section I used the red scale/*Aphytis* interaction to illustrate that individual-based population models, defined to include size-structured models of the sort described above, provide a particularly suitable framework for this purpose.

This seems to be an area ripe for exploitation, especially through incorporating into population models the results of the dynamic state-variable approach to optimality (e.g., Mangel and Roitberg 1992). The state-variable approach emphasizes the role of the current state of the organism in determining its decisions. For example, a parasitoid's attack rate, sex allocation, and tendency to host-feed rather than oviposit, may all be influenced by its egg load. Egg load in turn is determined by the number and quality of hosts the parasitoid has encountered, and the latter is determined in part by the abundance of parasitoids; there is thus feed-

back between population densities and the state variable controlling behavior. It is straightforward to incorporate an index of the organism's current state into size- and stage-structured models of population dynamics (Gurney et al. 1986).

There is now also opportunity for feedback in the opposite direction, from population dynamics to evolutionary studies. For example ESS (Evolutionary Stable Strategy) and other life history models typically have assumed a stationary population (Eadie and Fryxell 1992, Murdoch et al. 1992*b*) even though populations often show cyclic and perhaps even chaotic behavior. Metz et al. (1992), however, argue that the standard ESS approach can be extended to the latter situation. The same criterion (the geometric mean rate of growth at low frequency or density) can be used to explore fitness of a rare phenotype, and coexistence of competing populations, when the resident population is non-stationary.

Spatial processes

I noted above the need for empirical studies to test metapopulation theory. Here I want to draw attention to a recent innovation in the study of spatial dynamics and the empirical challenge it poses.

Most existing metapopulation theory assumes that populations consist of spatially distinguishable subpopulations. Key stabilizing features of metapopulations seem to be (1) out-of-phase fluctuations generated by local spatial differences, which may be fixed as in early models (e.g., Murdoch and Oaten 1975) or random as in later models (e.g., Reeve 1988), and (2) restrictions on the "mass action" assumption that individuals can instantly and without constraint move anywhere in the region. Hassell et al. (1991) use the same "local" model for within-subpopulation dynamics as Reeve (1988), namely the Nicholson–Bailey parasitoid–host model, but they achieve regional persistence without imposing persisting local spatial differences. The key here seems to be that different subpopulations are initially out of phase, i.e., we still need at least an initial set of spatial differences. It is clearly a major empirical challenge to determine whether real populations have the spatial structure, and dynamics, found in these models.

Recent work by DeRoos et al. (1991), however, suggests that the distinction between local and regional dynamics may be even more difficult to operationalize than is suggested by earlier metapopulation theory. They develop models in which individuals occupy explicit points in space and have restricted movement. Individual vital rates have a random component, which I interpret as causing spatial differences in vital rates and thus playing an analogous role to the spatial differences in parameter values among subpopulations in more standard theory. They show that, even though there is no imposed patch or subpopulation structure, locally different dynamics develop and lead, through out-of-phase fluctuations, to stability in an otherwise unstable predator–prey interaction. (There is also locally acting density dependence induced by the structure of the system; even so, persistence is impossible if there is unrestricted movement of individuals.) The authors define a "coherence length," which is the spatial extent of collections of individuals that fluctuate in a locally distinct way and out of phase with other local collections. It appears to be determined by a combination of the distance moved by individuals and their vital rates. These models thus produce an outcome similar to metapopulation dynamics, but in a spatially undifferentiated population occupying continuous and undifferentiated space.

Again, a major challenge is to develop empirical approaches to testing these ideas in real systems. It might be useful to isolate different-sized segments of the population and determine their stability, or at least their temporal variability. Models of the system might predict the rate at which temporal variability of an isolate decreases with its size, or threshold sizes at which qualitative changes in stability or variability occur. However, this may be altogether too naive an approach, and it would be useful for modelers to give thought to critical tests of their models.

I began by noting that regulation is central to ecology but, in the U.S., has fallen far down the research agenda. This perhaps reflects both previous uncertainty as to the nature and prevalence of regulation, and the difficulty of studying it. I have tried to show that the problem is in fact well defined and subjectable to experimental investigation and that recent developments in modeling have made regulation highly amenable to theoretical treatment and have created powerful tools for incorporating evolutionary ideas into the analysis of population dynamics. I believe the further development of our discipline requires that we place a high priority on the study of regulation. Conceptual development apart, we certainly need to do so to deal with the rapidly expanding environmental problems that beset us. Fortunately, we are increasingly better equipped to face this challenge.

Acknowledgments

Roger Nisbet and Allan Stewart-Oaten helped clarify a number of issues and Ilkka Hanski and Don Strong provided useful comments on the manuscript. Cherie Briggs, Bob Luck, Roger Nisbet, Sue Swarbrick, and Sandy Walde kindly allowed me to summarize some of our unpublished joint research. Finally, Sue Swarbrick helped with most of the analyses. The work discussed here was supported by several grants from NSF, by USDA grant 90-37153-5237, and by grant DE-FG03-89ER60885 from DOE, which does not necessarily agree with the results or conclusions.

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