13

Population dynamics and the evolutionary stability of biological control

ROBERT D. HOLT, MICHAEL E. HOCHBERG AND MICHAEL BARFIELD

Introduction

An appealing feature of classical biological control is that it has the potential to be self-sustaining, following initial establishment of the control agent, without the need for perpetual, large-scale human intervention. Most theoretical studies relevant to biological control (as in many chapters in this volume) explore population dynamics, making the quite reasonable assumption that basic biological properties of pest species and control agents are constant. Yet, over sufficiently long time-scales, neither target pests nor control agents are likely to have fixed properties. A truism of evolutionary biology is that all species harbor genetic variation for most characters (Lewontin, 1974) and so can respond *via* changes in genetic composition to environmental change. Introducing a biological control agent that limits pest numbers below economically significant levels surely counts as a significant environmental change, one that might be expected to have the potential to evoke an evolutionary response in the target pest species. This raises the question of what circumstances foster sustained biological control over evolutionary time-scales. Given strong selection, evolutionary time-scales permitting substantial changes in traits relevant to biological control could well be of the order of years or tens of years, not eons, given the high potential growth rates and population sizes, and short generation times, of many insect species. We suggest that the best biological control is that which can be evolutionarily stable, as well as ecologically persistent, and that a useful role of theory is to clarify when such evolutionary stability might be expected.

Recently (Holt & Hochberg, 1997) we have observed that while there are numerous instances of reduced pest control due to the evolution of resistance to chemical control agents, relatively few clear-cut examples exist for evolved breakdown in biological control. The few examples of evolved resistance that are reasonably unequivocal involve internal pathogens (e.g., myxomatosis, Dwyer *et al.*, 1990), rather than parasitoids or predators. Although for reasons discussed in detail in Holt & Hochberg (1997) it is difficult to quantify the seeming discrepancy in evolutionary responses to chemical *versus* biological control agents, there is little reason to doubt that it is real. This raises an interesting evolutionary puzzle.

Given that both chemical and biological control are likely to lead to novel selection pressures on target pest species, why is evolved resistance, leading to weakened control, not as obvious in biological as in chemical control systems? We (Holt & Hochberg, 1997) suggested that a number of complementary factors may contribute to this pattern. These include (i) the potential for greater costs of resistance to biological agents; (ii) the importance of behavioral plasticity in the control agent; (iii) the likelihood of coevolution, in which control agents evolve counter-adaptations to improved defenses in target species, and (iv) the role of spatial and temporal heterogeneity in weakening selection on target species. Scant data exist to assign relative importance to these factors; any may pertain to a given system.

Here, we use a simple theoretical model to examine the most novel of these four factors, the suggestion that spatial heterogeneity can weaken the evolutionary responses of a target pest species to an introduced control agent. The theoretical results reported below suggest that the constraining effects of spatial heterogeneity on host evolution can be greatly magnified by unstable population dynamics, which are likely to typify many biological control systems (see

Chapter 2). We should emphasize at the outset that our aim here is not to champion a particular model, but rather to use it to illustrate some of the dramatic effects spatial heterogeneity and population dynamics may have on evolutionary responses by host species to biological control agents. The particular conclusions we reach, drawn from a particular model, help highlight the need to examine the evolution of host resistance in a much wider range of models and ecological scenarios.

Sustained biological control requires that the population of the control agent persist, with oscillations in abundance bounded away from zero. Yet, an important aspect of natural enemy–victim interactions is their propensity towards instability when victim populations are limited to low levels by effective natural enemies (see Chapter 4). This observation has motivated the search for mechanisms stabilizing enemy–victim interactions, either at local equilibria or more broadly at landscape or regional scales (e.g., Hochberg, 1996). It is widely believed that spatial heterogeneity in one guise or another often helps to promote the persistence of strong natural enemy–victim interactions (e.g., Beddington *et al.*, 1978; Taylor, 1991). As we shall see, given that a host–parasitoid interaction persists because of spatial heterogeneity, the existence of unstable host–parasitoid population dynamics can have profound consequences for the rate of host evolution and the likely evolutionary stability of biological control.

A refuge model

We examine host evolution under biological control by parasitoids in situations where heterogeneity in parasitism is the ecological factor that permits persistence. Heterogeneity in parasitism may arise from a variety of sources, and can be broadly classified as involving individual-level phenomena (e.g., phenological and phenotypic variability in pests and/or natural enemies) or ensemble/population-level phenomena (e.g., density-dependent spatial variability). Such heterogeneities are generally recognized as stabilizers in host–parasitoid interactions, at least if the parasitoid limits host numbers to levels where direct density-dependence (namely self-limitation) is weak (Beddington *et al.*, 1978; see Chapter 2 and Chapter 12).

The mathematical model we employ is the 'generalized proportional refuge model' (Holt & Hassell, 1993). Although the model was originally intended to reflect spatial protection for a fraction of the host population, the same model form can be interpreted as describing other host refuge/resistance mechanisms where host individuals are in two discrete classes – essentially either vulnerable to, or protected from, parasitism throughout their lives, and in which the density of hosts in either class plays no role in their vulnerability. Possible examples include the encapsulation of juvenile parasitoids by larval hosts, and the concealment of larval hosts within plant structures from adult parasitoid attack.

For the sake of clarity, however, we will refer to the refuge in spatial terms, such that a fraction of hosts reside in an 'exposed' habitat and the remaining hosts are in the 'refuge' habitat. Refuge and exposed habitats may arise because of barriers to parasitoid activity, or by behavioral preferences on the part of the adult parasitoid to search exclusively for hosts in certain areas. The fraction of the host population residing in the refuge reflects individual host behavior, e.g., the probability that a maternal host will lay an egg in one habitat, rather than another. This probability of refuge use can be viewed as a measure of habitat preference, a behavioral trait which can evolve if genetic variation exists in individual host preferences (Jaenike & Holt, 1991).

Several evolutionary questions naturally arise about this system. Under what circumstances does selection favor refuge use by the host? Given that a fixed fraction of hosts use a refuge, what is the strength of selection acting on other parameters (e.g., habitat-specific growth rates, or parasitoid attack rates) in the system? We show that the answers to the above evolutionary questions may be substantially influenced by dynamical properties of the system, suggesting an important function of population dynamics in modulating the rate of host evolution in biological control. Our ultimate goal is to assess the interplay of population dynamics, host evolution, and the effectiveness of biological control.

Elsewhere we (Hochberg & Holt, 1995, and

unpublished results) have examined a rather different and more complex model for coevolution in a host–parasitoid system with a refuge. Here, we deliberately examine a system with both a simple ecology (the parasitoid is assumed to be effective enough so that the host does not experience direct density-dependence), and a simple genetics (we assume clonal or haploid variation in the host species). Moreover, rather than examine the more complex scenario of coevolution between parasitoid and host, we concentrate on how population dynamics influence evolution in the host, with a genetically fixed parasitoid, and the consequences of such evolution for the effectiveness of biological control. An important task for future work will be to examine a broader range of host genetic architectures and permit coevolutionary responses by the natural enemy, in a wider range of ecological models for interacting hosts and parasitoids.

Each generation, a fraction, ε_i, of hosts in clone i are exposed to parasitism; the remaining fraction, $1-\varepsilon_i$, are completely protected in the refuge habitat. In the absence of parasitism, hosts within and outside the refuge may have different expected net growth rates. *A priori*, it is reasonable to expect that host growth rates will tend to be smaller in the refuge than in exposed habitats; if this were not the case, then evolution would irrevocably be towards increased use of the refuge (barring other costs to protection from parasitism). Smaller refuge growth rates could be interpreted therefore as general 'costs' to protection from parasitism.

The model (from Holt & Hassell, 1993, generalized to multiple clones) is as follows:

$$\begin{aligned} N_i(t+1) &= N_i(t)[\varepsilon_i \lambda_{1i} f_i(P(t)) + (1-\varepsilon_i)\lambda_{2i}] \\ &= N_i(t) W_i(t) \end{aligned} \qquad (13.1)$$

$$P(t+1) = c\sum_i N_i(t)\varepsilon_i(1-f_i(P(T)))$$

Here, t denotes generation t, $N_i(t)$ is the abundance of host clone i in generation t, and $P(t)$ is the abundance of the parasitoid in generation t. In effect, in each generation a given clone experiences two environments – one (a refuge) in which it has a constant growth rate, and the other in which it may have either a variable growth rate, varying with fluctuations in parasitoid density, or a constant growth rate, if the system is at its demographic equilibrium and parasitoid numbers are constant.

In the remainder of this chapter, the quantity $f_i(P)$ is taken to be $e^{-a_i P}$, the usual Nicholson–Bailey form (e.g., Hassell, 1978). The quantities λ_1 and λ_2 are the growth rates of exposed and protected hosts, respectively. The quantity α_i is the attack rate, per parasitoid, on host clone i; c is the number of parasitoids emerging per parasitized host. For convenience, we set c equal to 1. The quantity $W_i(t)$ is the realized growth rate, or absolute fitness, of host clone i in generation t. As will be examined in more detail below, this fitness varies both with exposure to each habitat, and with parasitoid abundance. For notational clarity, in the following text the index i is suppressed except when referring explicitly to the dynamics of multiple clones.

As mentioned above, this model has previously been used to explore the importance of spatial heterogeneity in host growth rates for the persistence of the host–parasitoid interaction (Holt & Hassell, 1993). Here we recapitulate some key results. At equilibrium, parasitoid and host densities are, respectively:

$$P^* = \frac{1}{a}\ln\left[\frac{\varepsilon\lambda_1}{1-(1-\varepsilon)\lambda_2}\right] \qquad (13.2)$$

$$N^* = \frac{1}{ac}\ln\left[\frac{\varepsilon\lambda_1}{1-(1-\varepsilon)\lambda_2}\right]\left(\frac{\lambda_1}{\lambda_T - 1}\right)$$

where

$$\lambda_T = \varepsilon\lambda_1 + (1-\varepsilon)\lambda_2$$

is the host rate of increase at low densities, averaged over both habitats, in the absence of parasitism. For an equilibrium with positive densities to exist, two conditions must hold. First, $\lambda_T > 1$. Otherwise, the host goes extinct, dragging along with it the specialist parasitoid. Second, $(1-\varepsilon)\lambda_2 < 1$. The quantity $(1-\varepsilon)\lambda_2$ is the maximum rate of growth for those hosts residing in the refuge. If this quantity exceeds unity, these hosts (and hence the entire host population) grow geometrically, regardless of the fate of hosts exposed to parasitism. That is, the parasitoid cannot limit host numbers, and so there is no control.

The local stability analysis of this model is laid out fully in Holt & Hassell (1993), to which we refer the reader for details. Often, it is reasonable to assume that the growth rate of protected hosts differs from that of exposed hosts (e.g., because the two host classes experience different microclimates). The key factor determining stability in this model is the growth rate of hosts in the refuge, weighted by the fraction of hosts residing there. Even if hosts have a high rate of increase (averaged over both habitats), the system may be stable if $(1-\varepsilon)\lambda_2 < 1$. However, if protected hosts have a very low rate of increase, or a very low fraction use the refuge (leaving most hosts exposed to parasitism), although the system may persist it is likely to exhibit cyclic or chaotic dynamics, often with large-amplitude fluctuations. Thus, the message for sustained biological control from the proportional refuge model is that protection from natural enemies should be costly for target host species – but not too much so – if control is to persist without large-scale outbreaks over ecological timescales.

Host evolution in the proportional refuge model

Assume that a host is strongly limited by a parasitoid, and the interaction persists because the host uses a proportional refuge. How does host evolution modify the degree of control, and the importance of the refuge in allowing the interaction to persist, either at a stable point equilibrium or with bounded oscillations? There are two complementary techniques for examining such evolutionary questions. First, for multiple host clones, iterating the above equations allows one to analyze directly changes in the relative proportions of different clones in the total host population, and conditions for persistent polymorphism. Second, for any single clone the expression for W_i in eqn (13.1) defines a 'fitness generating function' (Brown & Vincent, 1992), which describes host fitness in generation t as a function of lower-level parameters, such as the attack rate. (Below, we will use 's' to denote an arbitrary parameter upon which W_i functionally depends). Manipulating the expression for W_i permits one to examine how small changes in parameter values translate into changes in host fitness. This analytic approach is typically difficult and indeed usually impossible when population dynamics are unstable (basically because one cannot solve for the time-series of population densities, given fluctuating populations, and so cannot analytically describe temporal variation in host fitness). In this chapter, we report the result of numerical studies of evolution in the system defined by eqn (13.1). (Similar results emerge from more detailed approximate, analytical studies of unstable systems: unpublished results).

We consider the direction of parameter evolution, first for stable population dynamics, and then for unstable population dynamics.

Stable population dynamics

Not surprisingly, the realized fitness of clone i declines with increasing parasitoid density. Because parasitoid density is determined by parasitoids emerging from all host clones, the fitness of one clone indirectly is depressed because other clones are present – an example of 'apparent competition' (Holt & Lawton, 1994). Holt & Lawton (1993) analyzed general models of apparent competition under the assumption that the system settles into a point equilibrium (i.e., with unvarying host and parasitoid densities). The stable coexistence of alternative clones sharing a parasitoid is usually impossible. The dominant clone out of any given clonal array is the one which sustains, and persists in the face of, the highest parasitoid density. This 'rule-of-thumb' provides a unifying principle for several of the following results.

Evolutionary biologists often assume that mutations causing a small change in a given character arise more frequently than mutations of large effect. Assume that fitness, W, is an arbitrary function of a parameter, s, and the initial population is fixed with $s = s'$. The fate of a mutation with a slightly different value of s, $s'' = s' + \delta s$ is determined by the difference between $W(s')$ and $W(s'')$; the greater this difference, the greater the selection favoring (or disfavoring) the new mutation. Using a Taylor expansion, we estimate the change in clonal fitness as $W(s'') = W(s') + \delta s(\mathrm{d}W/\mathrm{d}s|_{s=s'})$. The quantity $\mathrm{d}W/\mathrm{d}s$ is the 'strength

of selection' on a given parameter s; this quantity measures the marginal effect of a small increase in an arbitrary parameter s on fitness W. If the strength of selection is near zero, clones differing by a small amount in the value of s from s' are effectively selectively neutral. If, by contrast, the strength of selection is large in magnitude, selection more strongly favors (or disfavors) small deviations in s.

We consider the host parameters ε, a, λ_1, and λ_2 in turn, and assume that clonal variation exists in one parameter, while the other parameters remain fixed.

Evolution of host protection, $1-\varepsilon$

The strength of selection on use of the refuge is:

$$\frac{dW}{d(1-\varepsilon)} = -\lambda_1 e^{-aP} + \lambda_2 \tag{13.3}$$

After substituting $P=P^*$, we find that $dW/d(1-\varepsilon)>0$ if $\lambda_2>1$. As makes sense, hosts are selected towards increasing use of the refuge, where their positive growth rate permits them to grow, escaping control (until limited by other factors, beyond the scope of the model).

By contrast, $dW/d(1-\varepsilon)<0$ if $\lambda_2<1$. In this case, hosts are selected to avoid the refuge, regardless of the presence of the parasitoid. As refuge use evolves toward lower values for ε, the inherent instability of the interaction can emerge, creating host outbreaks.

It would appear that in either case, host evolution tends toward lower levels of effective control (either failed limitation of the target host by the parasitoid, or episodic pest outbreaks). Below, we examine in more detail how selection acts on ε, given population dynamic instability.

Evolution in the attack parameter, a

In contrast to evolution in refuge use, which includes an explicit cost ($\lambda_1>\lambda_2$), we can examine how exposed hosts may evolve cost-free resistance to parasitism *via* reductions in the parasitoid attack parameter, a. The strength of selection on the attack parameter is:

$$\frac{dW}{da} = -Pe^{-aP}\varepsilon\lambda_1 < 0 \tag{13.4}$$

which is always negative. Therefore, as expected from the assumed lack of costs, the host should evolve so as to lower attack rates. By inspection of eqn (13.2), this implies higher host density, and hence lowered control. It is interesting that all else being equal, the more effective the parasitoid is at attacking exposed hosts (higher a), the *weaker* is selection on the host to evolve counter-adaptations. Moreover, the magnitude of eqn (13.4) declines with increasing density of parasitoids (as long as $aP>1$). Basically, if total attacks are high enough, very few hosts escape parasitism at all, so small changes in the parameter a do not greatly reduce net parasitism rates and thus are not strongly favored.

Evolution in the basic growth rates, λ

Assume that use of the refuge is fixed, and host productivity within it is permitted to evolve. The strength of selection on growth in the refuge is simply:

$$\frac{dW}{d\lambda_2} = 1-\varepsilon \tag{13.5}$$

Thus, the strength of selection relevant to adaptation to the refuge is directly proportional to the magnitude of use of the refuge, but is independent of the absolute growth rates in either habitat.

Strength of selection on growth in the exposed habitat is:

$$\frac{dW}{d\lambda_1} = \varepsilon e^{-aP} = \frac{1-(1-\varepsilon)\lambda_2}{\lambda_1} \tag{13.6}$$

In contrast to eqn (13.5), larger growth rates in either habitat weaken selection on growth rate in the exposed habitat. The strength of selection for improved adaptation to the refuge exceeds that for improved adaptation to the exposed habitat (namely $dW/d\lambda_2>dW/d\lambda_1$), provided that:

$$\lambda_1+\lambda_2>\frac{1}{1-\varepsilon}$$

The reason for the effect of host growth rate on the strength of selection on exposed hosts is that large host λ values tend to sustain large parasitoid populations, hence few hosts outside the refuge are likely to

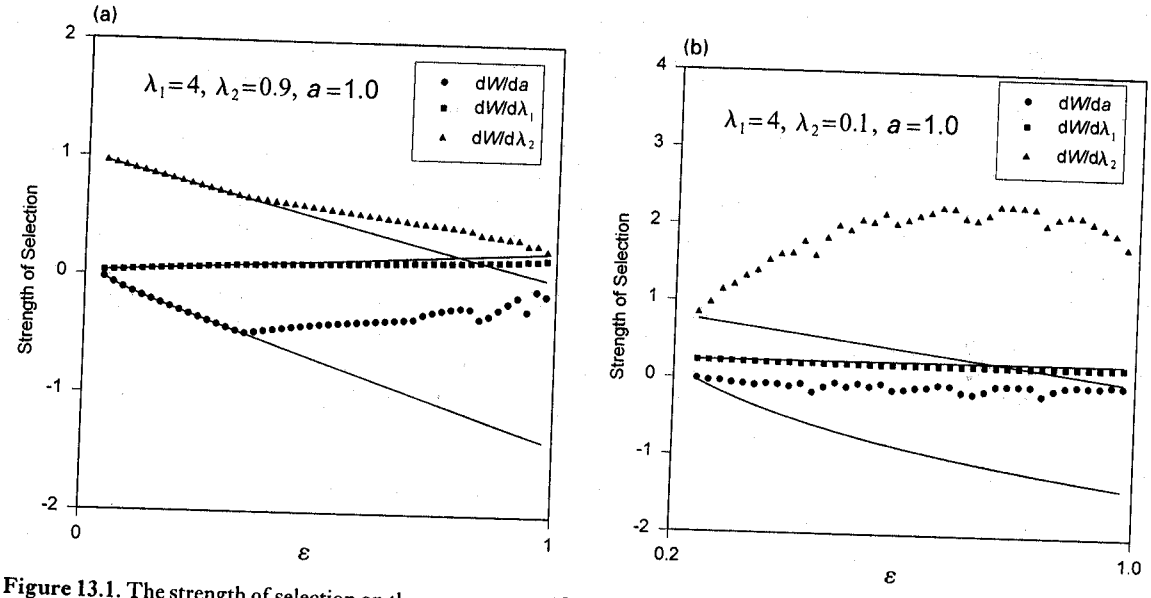

Figure 13.1. The strength of selection on three parameters (●, attack rate (a); ■, growth rate in exposed habitat (λ_1); ▲, growth rate in protected refuge (λ_2)) as a function of preference for the exposed habitat (ε). The parameters used are shown in the boxes. (a) moderate-quality refuge; (b) low-quality refuge. At low ε, the populations show stable dynamics. The continuous lines depict the expressions for strength of selection, assuming equilibrium, derived in the text. The divergence of the symbols from the lines indicates effects of population dynamics on the strength of selection. For unstable dynamics, the strength of selection was calculated as follows. First, a single clone is simulated using the parameters indicated in the graphs for 90 000 generations. For the next 10 000 generations, the values of $P(t)$ were recorded. This was then used to calculate the geometric mean fitness for a rare clone, over this same timespan. If sufficiently rare, the invading clone should have no effect on parasitoid dynamics. The rare clone was chosen to have parameters slightly different from the resident clone. This was done for two rare clones, each deviating by 1% in its parameter value (above and below, respectively) from the resident clone. To estimate dW_g/dq, the difference in these clones' realized value for W_g was divided by the difference in the parameter values. This process was repeated for ε in increments of 0.02. This numerical procedure agrees with the theoretical results, in the stable region. The parameters in (b) lead to much more unstable dynamics than those in (a) (see Holt & Hassell, 1993). The figure shows that there are correspondingly larger deviations in selection from equilibrial expectations.

escape parasitism and benefit from an increase in λ. This weakens selection for improved adaptation to the non-refuge habitat. It is useful to recall that the strength of selection is a measure of the net effect of a small change in a trait, averaged over all environments experienced by individuals expressing that trait. Because the model assumes that reproduction follows parasitism, high parasitism rates (which in this model are indirectly generated by high host growth rates) imply that most exposed individuals never live to realize a potential increase in fitness.

If potential pest populations have high intrinsic capacities for increase in the absence of biological control, given that refuges stabilize the host–parasitoid interaction, there appears to be a bias in host adaptive evolution towards conditions within the refuge, relative to conditions outside.

Pulling these results together, the continuous lines of Fig. 13.1 depict for a particular combination of parameters how strength of selection on the various model parameters varies with ε.

In short, selection on the refuge use parameter of the host always tends to weaken biological control. By inspection of eqn (13.2), we see that host evolution on the attack parameter, a, always leads to higher host numbers, thus less effective control. One interesting

result for stable systems is that control from very effective parasitoids (high a) might decay less rapidly than control from less-effective parasitoids (low a). There are two reasons for this: (i) as noted above, the strength of selection favoring a lower a, weakens with increasing a; and (ii) host abundance (and hence the likely pool of genetic variation) declines with increasing a. Finally, from eqn (13.2) it can be seen that host densities increase (albeit weakly) with increasing host growth rates, so selection on these parameters also implies somewhat lowered control.

Unstable population dynamics

As noted earlier and explored more fully in Chapter 4, a hallmark of host–parasitoid dynamics where hosts are limited to low densities by parasitoids is a propensity towards dramatic instability, with potentially damaging host outbreaks (see Chapter 12). We have seen that if a host–parasitoid interaction is stabilized because of a refuge, there is a tendency for parasitoid-dominated limitation to disappear over evolutionary time. In particular, if the refuge is a sink (i.e., $\lambda_2 < 1$, so that the host would go extinct were it restricted there), hosts are selected to avoid the refuge. Host evolution in general thus would appear to be inimical to long-term biological control. However, this particular conclusion assumes that the host–parasitoid system remains dynamically stable, as the host evolves.

We now turn our attention to evaluating selection when refuge use is low enough for parasitoid-driven instability. The basic question we ask is how such instability influences selection on the host. If dynamical instability weakens selection on hosts to escape parasitism, then systems that are to a degree unstable may evolve less rapidly than systems that are stable, and so retain control for longer spans of time. As before, we examine evolution in each parameter separately, but instead of reporting analytical results we now use numerical studies.

Evolution of habitat preference

We assume that the refuge is a sink habitat ($\lambda_2 < 1$), so that selection favors hosts that avoid the refuge if populations attain constant levels. Our first procedure is to compete pairs of clones against each other, using numerical simulations of eqn (13.1). The clones have similar parameter values, except that they differ in their habitat preferences (measured by ε). Simulations were run until one clone dominated the host population (to within machine error), or for 100 000 generations. Figure 13.2 shows the outcome of these competitive trials for a number of parameter combinations.

It is useful to walk through the example of Fig. 13.2(a). To save space, this figure presents the results of two different scenarios in one frame: one of relatively high productivity of exposed hosts ($\lambda_1 = 10$, the upper triangle) and one of low productivity ($\lambda_1 = 4$, the lower triangle). In both cases the refuge habitat is assumed to be a slight sink, or $\lambda_2 = 0.9$. The two scenarios are illustrated on opposite signs of a diagonal line of slope 1. Because ε_i is bounded between 0 and 1, the upper and lower triangles can denote any possible pair of competing clones differing solely in habitat preference; above the line of slope 1, we call the clone with higher preference for the exposed habitat, clone 2 (namely $\varepsilon_2 > \varepsilon_1$), and below the line, the clone of higher preference for the exposed habitat is clone 1 (this is just a re-labeling). In zones denoted ε_j (where $j = 1$ or 2) that clone wins; in zones denoted C, there is clonal coexistence (= protected genetic polymorphism).

We demonstrated above that given stable dynamics, selection should favor avoiding the refuge if the absolute growth rate there is less than 1. Figure 13.2 shows that if one starts at high refuge use (low ε, implying stable dynamics), selection indeed favors higher exposure to the parasitoid; clones with higher values of ε replace clones with lower values of ε.

However, at higher exposure, the population dynamics become unstable. Figure 13.3 displays typical patterns in mean host abundance and coefficient of variation in abundance, as a function of ε. For sufficiently high ε, the dynamics are unstable and display increasingly large variance in numbers as ε increases. Figure 13.4 depicts an example of how the frequency of excursions to high levels (i.e., potentially worrisome pest outbreaks) tends to increase with ε.

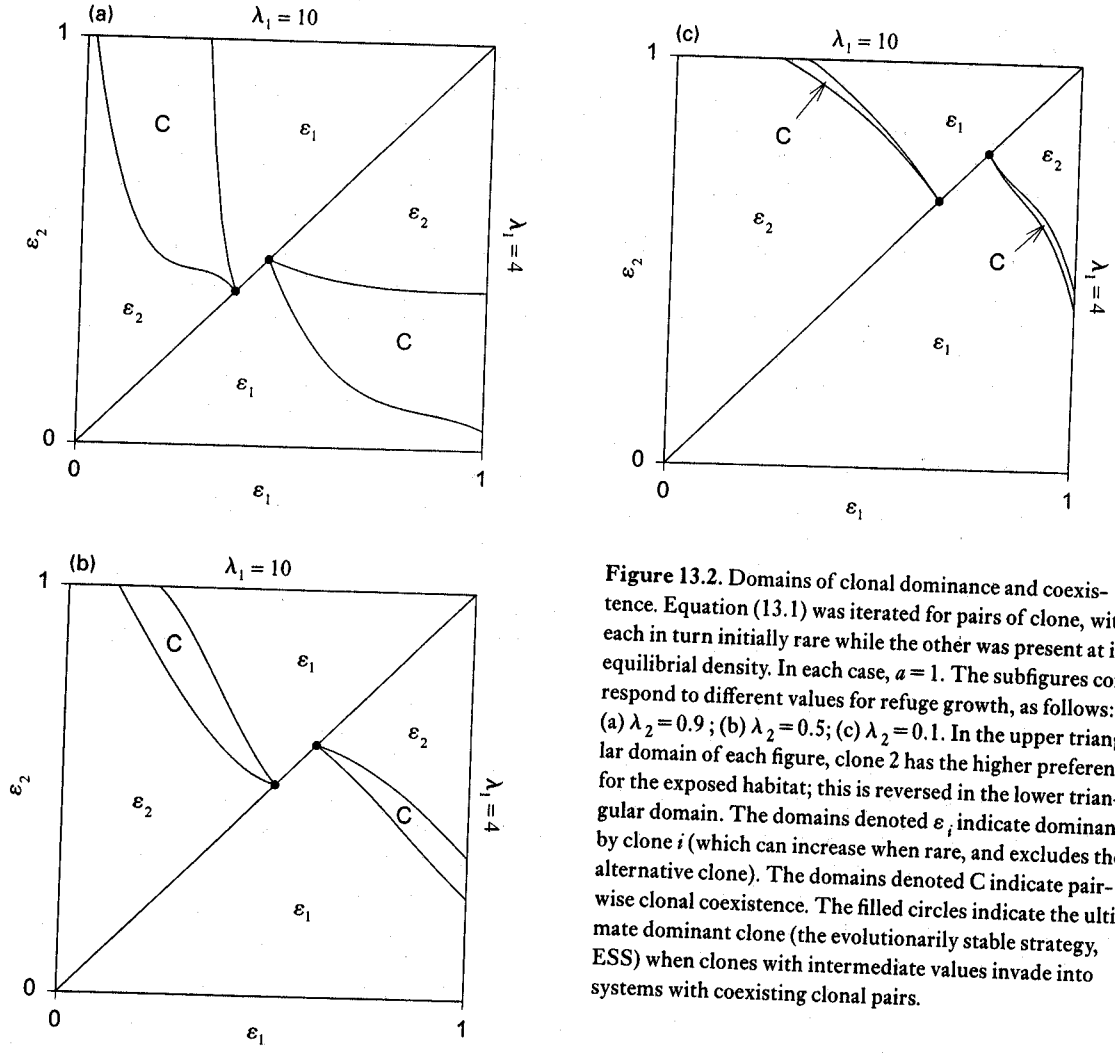

Figure 13.2. Domains of clonal dominance and coexistence. Equation (13.1) was iterated for pairs of clone, with each in turn initially rare while the other was present at its equilibrial density. In each case, $a = 1$. The subfigures correspond to different values for refuge growth, as follows: (a) $\lambda_2 = 0.9$; (b) $\lambda_2 = 0.5$; (c) $\lambda_2 = 0.1$. In the upper triangular domain of each figure, clone 2 has the higher preference for the exposed habitat; this is reversed in the lower triangular domain. The domains denoted ε_i indicate dominance by clone i (which can increase when rare, and excludes the alternative clone). The domains denoted C indicate pairwise clonal coexistence. The filled circles indicate the ultimate dominant clone (the evolutionarily stable strategy, ESS) when clones with intermediate values invade into systems with coexisting clonal pairs.

Population dynamics play a crucial role in determining the direction of evolution of refuge use. In particular, population instability tends to put a brake on the evolutionary decline in use of the refuge. From Fig. 13.2, we see that at high current values of exposure, *lower* values for exposure are favored. Thus, unstable population dynamics fosters use of an (on average) intrinsically costly, or low quality, refuge habitat.

If a pair of clones differ substantially in their use of the refuge, sustained coexistence can occur (area C of Fig. 13.2). However, if one starts with such a coexisting pair, and introduces a clone with an exposure level intermediate to those of the two resident clones (iterating the above model for three clones simultaneously), one of the original pair inevitably disappears. In our simulations of such three-way clonal competition, with repeated introductions of clones with intermediate habitat use (compared to a coexisting clonal pair) the system moves towards a single, intermediate value for ε (indicated by filled circles in the figure), a frequency of refuge use which

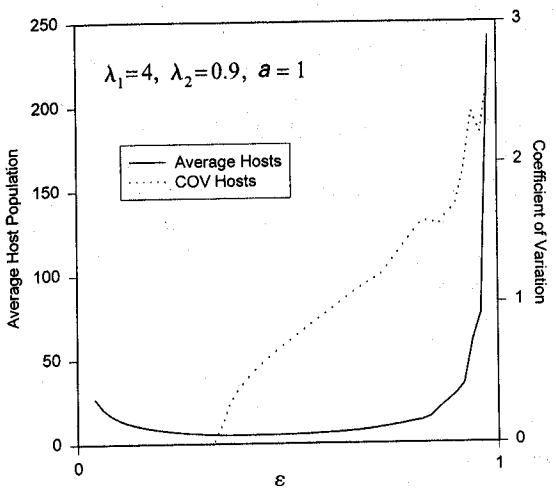

Figure 13.3. Average host abundance, and coefficient of variation in numbers, as a function of ε. —, average hosts; ·····, coefficient of variation in hosts.

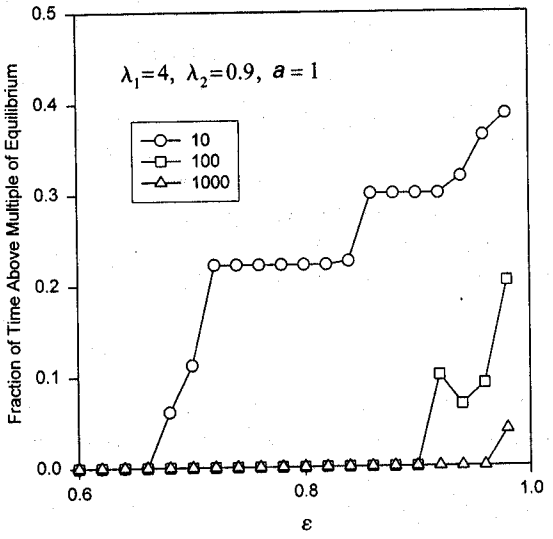

Figure 13.4. Fraction of time population trajectories reach a magnitude of $Y\times$ equilibrial values, where $Y = 10$ (circles), 100 (squares), and 1000 (triangles). The incidence of extreme population values increases with increasing ε.

defeats all lower or higher values for ε. This defines an evolutionarily stable strategy (ESS) refuge level. As the cost to being in the refuge increases (or quality there decreases), λ_2 declines, and the domain of parameter space permitting clonal coexistence decreases. Along with this shift in the domain of coexistence, the evolutionarily stable refuge level shifts towards lower levels (compare Figs 13.2(a), (b) and (c)).

The importance of initial conditions

The effect of refuge evolution upon the magnitude of realized control depends upon the starting conditions. If originally the refuge was not greatly utilized by the host, the dynamics are highly unstable. An evolutionary increase in refuge levels tends to greatly lower variability in host population abundance, measured for instance by the coefficient of variation or the frequency of excursions to a multiple of equilibrial host population size. In this case, host evolution can lead to more sustained control (namely fewer and smaller outbreaks). By contrast, if the initial population was heavily protected by the refuge, a decay in refuge level leads to increased mean abundance and an increase in variability, thus less effective control. The magnitude of this effect depends upon the cost of being protected or alternatively by the inherent quality of the refuge; the higher the cost or lower the quality of the refuge, the more the magnitude of variability in abundance is observed to increase at the ESS (our unpublished results). Moreover, once the dynamics become unstable, selection favors continued refuge use, helping to prevent yet further decay in control.

Evolution of the attack parameter

As a second approach to analyzing parameter evolution, given population fluctuations, we empirically computed a strength of selection. In the numerical simulations we introduced clones differing by a small amount in a given parameter at very low densities into populations fluctuating around their equilibrium. The strength of selection was determined as the average change in geometric mean fitness over a specified time period, per unit change in the parameter (see the legend to Fig. 13.1 for more details on our protocol).

Figure 13.1 (filled circles) shows how population

fluctuations influence the strength of selection on the attack parameter, compared to expectations based upon equilibrial assumptions (when the symbols are coincident with the straight lines in the figure, the population dynamics are stable). The effect of instability is, in general, to weaken the strength of selection on a. Indeed, when refuge hosts have a low intrinsic growth rate (see Fig. 13.1(b)), the strength of selection on a is not markedly different from zero, regardless of the proportion of protected hosts.

This suggests that if a host–parasitoid interaction persists with bounded fluctuations in host numbers, because of the high costs of protection or use of a low-quality refuge, then this ecological mechanism fostering persistence simultaneously hampers the evolution of resistance by the host to the parasitoid.

Evolution of growth rates

Our numerical studies suggest that population instability may at times have a small effect on the strength of selection towards improved adaptation to the exposed state (e.g., Fig. 13.1, filled squares). By contrast, such instability increases the strength of selection towards improved adaptation to the refuge state (Fig. 13.1, filled triangles). This is particularly notable in Fig. 13.1(b) (where the parameter choices lead to large fluctuations in P), which shows that the strength of selection to the refuge actually *increases* with decreasing proportion of hosts protected by it – opposite the expectation derived given an assumption of equilibrial populations.

Our analysis of the proportional refuge model shows that combining spatial heterogeneity and unstable population dynamics can lead to what, at first glance, are counterintuitive effects on selection. To make the results more intuitive, it is helpful to recall some general effects of heterogeneous environments upon evolutionary dynamics. Considering spatial heterogeneity alone, one expects an automatic bias in selection towards habitats in which a species is most common, and in which they are most successful (see Holt, 1996). Considering temporal variability alone, because selection is governed by geometric mean fitness across generations (Seger & Brockmann, 1987), there is an automatic bias towards generations of low, rather than high, fitness. In the proportional refuge model, unstable dynamics leads to periods of low host fitness (generations of high parasitoid abundance), during which the only reproductively successful hosts are those found in the refuge, where fitnesses are constant. Small changes in attack rates or intrinsic growth rates on exposed hosts are in effect devalued by selection, because most exposed hosts are lost to parasitism in any case during these years of low fitness. The greater the amplitude of variation in parasitoid numbers, the stronger this bias towards the refuge is likely to be.

Discussion

Effective biological control usually mandates that one must attempt to limit target pest species to densities at which the inherent instability of natural enemy–victim interactions is likely to be important. Often, spatial factors such as refuges may be required for such strong interactions to persist. Given that strong limitation of a target pest species by an introduced control agent may generate strong selection on the former species, potentially leading to the breakdown of control, it is of considerable interest to understand how spatial and temporal heterogeneities can modify such evolutionary dynamics. In Holt & Hochberg (1997) we examined a number of factors that influence the evolutionary stability of biological control. Here, we have demonstrated that the existence of unstable population dynamics can dramatically change the likely direction of evolution in a target host species, in a system which persists because the host can utilize a refuge. In particular, our theoretical results suggest that unstable dynamics can make it more difficult for pest species to evolve counter-adaptations to parasitism. Moreover, unstable dynamics foster the continued use of costly/low-quality refuges, which can be the key factor permitting the sustained persistence of inherently unstable host–parasitoid interactions. The general conclusion we draw is that for the purpose of sustained biological control, aiming for the tight regulation of the target pest by the control agent may in the end be self-defeating.

Holt (1997) explores why use of a suboptimal sink habitat (in this case, the refuge) can be favored by selection if populations have unstable dynamics. The basic idea is that unstable dynamics in one habitat necessarily mirrors temporal variation in fitness there. All that is needed for use of a second, low-quality habitat to be favored by selection is that in some generations, fitness there exceeds fitness in the (on average) higher-quality habitat. Utilization of low-quality habitats can be a form of 'bet-hedging' (Seger & Brockmann, 1987) fostered by variance in fitness in high-quality habitats. Weak use of the refuge leads to population fluctuations driven by the unstable host–parasitoid interaction, automatically paving the way for increased use of lower-quality refuge habitats lacking the parasitoid.

This suggests that the maintenance of biological control over evolutionary time-scales may reflect the interplay of a number of factors. Refuges are important ecologically in reducing the magnitude of population fluctuations, thus permitting the persistence of tightly-coupled host–parasitiod interactions. However, given stable population dynamics, the evolution of refuge use is in the direction of less effective limitation and regulation by the parasitoid (Hochberg & Holt, 1995). Indeed in some systems the very existence of unstable population dynamics may be needed for refuge levels to remain at an intermediate level (rather than all hosts being exposed, or protected) over evolutionary time-scales. Natural selection tends to ensure that some hosts will remain exposed to parasitoids, only if there are costs to protection in refuges (e.g., if refuges are intrinsically lower in quality than exposed habitats). Partial refuge use is evolutionarily stable, given unstable host–parasitoid dynamics. Highly effective control that leads to tight host regulation solely by parasitoids may thus be evanescent, given evolutionary responses to parasitism by hosts (Hochberg & Holt, 1995).

Our investigation considered the strength of selection, one parameter at a time. More generally, one could imagine that trait evolution involves trade-offs among parameters. Moreover, it is likely that there will be concurrent parameter evolution (i.e., coevolution) in the parasitoid, as well. It is also important to examine a much wider range of ecological models. A full examination of all these possibilities is beyond the scope of this chapter. However, the insights drawn from the proportional refuge model are likely to apply much more broadly. Evolution by natural selection tends to favor the optimization of any character that has a large strength of selection upon it, at the expense of characters with a low to zero strength of selection. Overall, even with trade-offs among parameters and parasitoid coevolution, unstable host–parasitoid dynamics should foster the employment of costly/low-quality refuges by hosts, and diminish the effectiveness of selection on the host to withstand parasitism by other mechanisms outside the refuge.

We suggest that a modicum of instability in biological control over ecological time-scales may in some circumstances facilitate the persistence of biological control over evolutionary time-scales. The conventional wisdom that one should seek natural enemies that lead to stable population dynamics in biological control may, in the end, lead to self-defeating efforts in control. This evolutionary perspective complements discussion of the utility of natural enemies that are local exterminators of target species – rather than regulators – in biological control programs (see Chapters 2 and 4). We caution that this potential long-term role of dynamic instability in facilitating long-term control has been demonstrated only for a rather simple model (the above proportional refuge model). We would like to leave the reader with two main messages. First, it is important that biological control theory add an evolutionary dimension to the existing rich, sophisticated literature on ecological mechanisms fostering persistence and stability of biological control systems. Second, one issue that warrants particular attention is the role of spatial heterogeneity and temporal variability in constraining evolutionary responses by target pest species to effective biological control agents. The long-term effectiveness of biological control may require either a finely balanced coevolutionary arms race, or a kind of conservatism in the basic ecological niche (broadly defined, Holt, 1996) of the pest. Spatial heterogeneity and unstable dynamics can foster such conservatism.

References

Beddington, J. R., Free, C. A. & Lawton, J. H. (1978) Modelling biological control: on the characteristics of successful natural enemies. *Nature* **273**: 513–19.

Brown, J. S. & Vincent, T. L. (1992) Organization of predator–prey communities as an evolutionary game. *Evolution* **46**: 1269–83.

Dwyer, G., Levin, S. A. & Buttel, L. (1990) A simulation model of the population dynamics and evolution of myxomatosis. *Ecological Monographs* **60**: 423–47.

Hassell, M. P. (1978) *The Dynamics of Arthropod Predator–prey Systems*. Princeton: Princeton University Press.

Hochberg, M. E. (1996) An integrative paradigm for the dynamics of monophagous parasitoid–host interactions. *Oikos* **77**: 556–61.

Hochberg, M. E. & Holt, R. D. (1995) Refuge evolution and the population dynamics of coupled host–parasitoid associations. *Evolutionary Ecology* **9**: 633–61.

Holt, R. D. (1996) Demographic constraints in evolution: towards unifying the evolutionary theories of senescence and niche conservatism. *Evolutionary Ecology* **11**: 1–10.

Holt, R. D. (1997) On the evolutionary stability of sink populations. *Evolutionary Ecology* **11**: 723–32.

Holt, R. D. & Hassell, M. P. (1993) Environmental heterogeneity and the stability of host–parasitoid interactions. *Journal of Animal Ecology* **62**: 89–100.

Holt, R. D. & Hochberg, M. E. (1997) When is biological control evolutionarily stable (or is it)? *Ecology*.**78**: 1673–83.

Holt, R. D. & Lawton, J. H. (1993) Apparent competition and enemy-free space in insect host–parasitoid communities. *American Naturalist* **142**: 623–45.

Holt, R. D. & Lawton, J. H. (1994) The ecological consequences of shared natural enemies. *Annual Review of Ecology and Systematics* **25**: 495–520.

Jaenike, J. & Holt, R. D. (1991) Genetic variation for habitat preference: evidence and explanations. *American Naturalist* **137**: S67–S90.

Lewontin, R. C. (1974) *The Genetic Basics of Evolutionary Change*. New York: Columbia University Press.

Seger, J. & H. J. Brockmann. (1987) What is bet-hedging? *Oxford Surveys in Evolutionary Biology* **4**: 182–211.

Taylor, A. D. (1991) Studying metapopulation effects in predator–prey systems. *Biological Journal of the Linnaean Society* **42**: 305–23.