Concepts

Emphasizing new ideas to stimulate research in ecology

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WHEN IS BIOLOGICAL CONTROL EVOLUTIONARILY STABLE (OR IS IT)?

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Abstract. The evolution of resistance by insect and weed pests to chemical pesticides is a problem of increasing importance in applied ecology. It is striking that the evolution of resistance by target pest species in biological control is much less frequently reported, particularly in control involving parasitoids and predators, rather than pathogens. Although it is conceivable that this reflects biases in reporting or frequency of application, we suggest that there is a puzzle here worthy of scrutiny, and we outline several potential underlying causes. In order of discussion (not necessarily of importance), these are: (1) lack of genetic variation; (2) genetic constraints on selection; (3) weak selection; (4) temporally varying selection; and (5) coevolutionary dynamics. We, in particular, focus on the potential for weak selection on the host for increased resistance, despite effective control. The very spatial mechanisms (e.g., refuges, metapopulation dynamics) believed to facilitate the persistence of many natural enemy–victim systems with strong biological control may also incidentally provide an environment where selection is weak on target pests to evolve improved resistance to control agents, thereby biasing coevolution toward the enemy. The basic insight is that in a spatially heterogeneous environment, a strong *limiting* factor on a population can be a weak *selective* factor. The hypotheses presented here provide ingredients needed to predict which biological control systems might be evolutionarily stable, and which not. Our aim in this thought piece is to stimulate more attention to the evolutionary dimension of biological control systems.

Key words: coevolution; evolution of pest species; genetic constraints on selection; niche conservatism; resistance to biological control.

INTRODUCTION

One of the most enduring and societally important enterprises in applied ecology throughout this century has been the control of populations of economically damaging species, particularly of agricultural weed and insect pests (DeBach 1974). The war against pests has employed a vast array of tactics, including the application of a diverse array of chemicals, and the introduction of predators, parasitoids, and pathogens in biological control programs. ''Control,'' as employed here, is the persistent reduction of the target pest below a target level (usually involving an economic threshold). Often (if not always), economically successful control mandates that the pest not greatly affect the levels of its own resource (e.g., annual production of an agricultural crop). A simple measure of the effectiveness of control is the ''q value'', where $q = N^*/K$, N^* is the equilibrium or time-averaged abundance of the pest in the control situation, and K is the pest's average abundance in the absence of the control agent (Beddington et al. 1978). ''Biological control'' in this paper denotes control from self-sustaining populations of an introduced control agent that limits populations of its prey or host species, the target pest (Waage and Mills 1992). ''Effective'' biological control agents cause substantial, persistent reductions in pest abundance.

As the human population continues to increase, managing pest problems for both the short and long term can only become more urgent. Control programs, whether emphasizing chemical or biological control agents, often implicitly assume that target species are evolutionarily static, fixed targets. Yet all biological species harbor genetic variation and can respond to environmental change by shifts in their genetic composition. Because many pest species have short gen-

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eration lengths and large population sizes, if control agents exert strong selection one expects substantial evolutionary change over short time spans (Gould 1991).

Given these basic facts, a striking contrast appears to exist in evolutionary responses by target pest species to chemical and biological pest control: in chemical control, the evolution of resistance to an economically troublesome level has occurred repeatedly, and in many habitats and taxa (Gould 1991), whereas in biological control, examples of the evolutionary breakdown of effective control are much less frequently reported (Croft 1992).

In this thought piece, we do two things. First, we sketch our rationale for suspecting the existence of a broad disparity between the average evolutionary responses (as manifest in control levels) of target pest species to chemical vs. biological, control, and point out potential biases in the available data. Then, we present hypotheses for why evolved resistance to biological control agents may not be as pervasive as is evolution to withstand chemical control. These hypotheses include the potential for greater costs of resistance to biological control agents, and the greater importance of behavioral plasticity, population dynamics, spatial heterogeneity, and coevolutionary dynamics in biological control. These hypotheses are complementary, not competing; all could contribute to the observed pattern.

The Pattern

Chemical control: rampant evolutionary decay

There are now a very large number of compelling examples of the evolution of resistance to pesticides by arthropods and weeds, leading to reduced control (e.g., Georghiou 1986, Denholm and Rowland 1992). Indeed, populations of some insect species have developed resistance to essentially *all* available insecticides (e.g., the diamondback moth, *Plutella xylostella,* Cheng 1988).

It is difficult from available data to develop a statistically robust measure of the incidence of evolved resistance. Yet there are numerous persuasive indirect indications that the phenomenon of evolved resistance to chemical control is widespread. May and Dobson (1986) observe that the percentage of agricultural production lost to insect pests, disease, and weeds doubled between the 1940s and 1980s, despite the great increase in pesticide application over this same period. This observation provides a kind of summary statistic across many crops and pest taxa, suggesting the frequent, independent emergence of evolved resistance (although it is difficult to separate the effects of evolved resistance from the many changes in agriculture in the last 50 yr, including decreasing genetic diversity of crops, increased farm sizes, intensive crop breeding, and so on). Georghiou (1986) states that ''whereas the presence of resistance was a rare phenomenon during the early 1950s, it is the fully susceptible population that is rare in the 1980s,'' including serious cases of resistance in plant pathogens (toward fungicides and bactericides) and in weeds (toward herbicides).

Indeed, the urgency of the problem of evolved resistance has led to a growing attention to ''insecticide resistance management'' (Green et al. 1990, Denholm and Rowland 1992), where aspects of the ecology of the target pest species (e.g., its habitat distribution, phenology, and mobility) are exploited to conserve susceptibility. There now exists a sophisticated theoretical literature that uses population genetic theory to predict the selective environment arising from different application regimes, leading to concrete suggestions for the evolutionary management of target pest species (e.g., Comins 1977, Taylor and Georghiou 1979, Tabashnik 1986, Gould 1991, Gould et al. 1992, Ives 1996).

Biological control: sporadic evolutionary breakdown

There have cumulatively been hundreds of successful establishments of insect natural enemies, particularly insect parasitoids (Waage and Greathead 1988; J. K. Waage, *personal communication*), leading to the lasting control of arthropod and plant pests, with some examples now spanning many decades (e.g., red scale [*Aphytis*], Murdoch et al. 1985). One might imagine effective control would often induce strong selection on the pest to rapidly evolve mechanisms of escape or tolerance to attacks by the control agent, leading to weakened control. The existence of geographical variation in the virulence of parasitic natural enemies and in the susceptibility of their hosts (e.g., Briese 1986*a*, *b*, Carton and Nappi 1991) makes it reasonable to expect evolved resistance in biological control systems.

Yet there are few clear-cut examples of the erosion of biological control where evolution appears to be the culprit (as opposed to, say, environmental change). Moreover, most of the good examples to date appear to involve microbial control agents (e.g., viruses, bacteria, protozoa), some of which have been applied in an augmentative or inundative manner to control insects (e.g., Channer and Gowen 1992; reviews in Briese 1986*a, b*).

One possible case of the erosion of biological control due to evolution in the host involves the rhinoceros beetle, *Oryctes rhinoceros,* and a baculovirus pathogen. *Oryctes* is a serious pest of coconut and oil palms in Southeast Asian islands, and has been successfully controlled on many of these islands over the past two decades (Young 1986). On certain islands, Java and South Sulawesi in particular, continuous outbreaks of the pest occurred in the mid-1980s, raising the possibility that the beetle had evolved resistance to its pathogen. Laboratory trials employing various strains of the virus from other areas showed that pathogen virulence was much lower in beetle populations from agricultural

habitats than in populations from native habitats, consistent with a hypothesis of evolved resistance (Zelazny et al. 1989). However, it is not known whether the apparent resistance of the beetle evolved in response to its native pathogen strains.

Other potential examples of evolved host resistance come from biological control of introduced weeds. For instance, Burdon et al. (1981) reported an increase in resistance in *Chondrilla juncea* following successful biological control by the rust fungus *Puccinia chondrillina*. However, the effect on overall control is unclear. Burdon and Marshall (1981) reviewed the available literature and showed that asexual weeds are often more effectively controlled by biological control agents than are sexual weed species. Yet there could be overall effects of initial levels of genetic variation on the effectiveness of the original control efforts, without an evolutionary increase in resistance subsequent to the initiation of control.

The best documented example of evolved host resistance and coevolution in a host–parasite biological control system is the famous case of the interaction between rabbits and the myxomatosis virus in Australia. After initial mortality rates approaching 100%, new strains of the virus increased rapidly in frequency, in concert with the breakdown of control provided by the virus (Fenner 1983). Dwyer et al. (1990) have examined this system in a population dynamic, evolutionary framework, and argued that host–pathogen coevolution, including increased host resistance, is likely to have occurred.

Yet despite these few familiar examples, the evolutionary decay of control in biological control systems does not appear to be the ubiquitous phenomenon so throughly documented in the literature of chemical control, particularly if one considers control agents other than internal pathogens. For instance, in a literature review, Waage and Greathead (1988) could find no clear field evidence for a decrease in parasitoid effectiveness during the course of classical biological control. If anything, parasitoids may become *more effective* after establishment (Waage and Greathead 1988) (though many examples cannot exclude alternative, nonadaptive explanations [Weis et al. 1989, Hopper et al. 1993]).

We have not been able to find published or unpublished unequivocal accounts of the evolutionary decay of control due to evolved resistance to parasitoids or predators used as biological control agents. Henter and Via (1995) recently independently surveyed the literature, and found no clear cases in which an insect has evolved in response to parasitoids in biological control. We by no means claim that such examples do not exist (that would indeed be surprising), but do stress that there is a substantial disparity in reports of evolved resistance leading to greatly reduced control in chemical, vs. biological, control systems. Informal consultation with a number of students of biological control employing parasitoids and predators (see *Acknowledgments*) supports this tentative generalization.

One possible case for host evolution due to the presumed selection pressure caused by a parasitoid under field conditions involves the larch sawfly (*Pristiphora erichsonii*) and the ichneumonid wasp *Meoleius tenthredinis*. This parasitoid was imported from England to America in 1910 to control the sawfly, but almost three decades later an outbreak of the pest was observed in Manitoba (Ives and Muldrew 1981). A few years later (1944) it was discovered that these outbreak hosts were encapsulating the parasitoid's eggs (Muldrew 1953). By the early 1970s, resistant populations had been identified across the northern United States, from Minnesota to New York. This looks at first glance like a firm case of evolved resistance to control by a parasitoid in field populations. However, an alternative reasonable explanation is that instead of in situ evolution, there were additional introductions of resistant strains across the host species' range (Ives and Muldrew 1981).

In short, a signature of chemical control of pest species is that it often leads to evolution in the target pest that erodes control, whereas such an erosion of control in biological control systems seems much more sporadic, and may be largely restricted to host–pathogen examples. We suggest there is a puzzle here. In the remainder of this thought piece, we address two issues. First, are there reasons one might suspect the very existence of this pattern? Second, given that the pattern exists, are there generic features of biological control systems, particularly those involving parasitoids and predators, which might explain a lack of manifest evolutionary changes in control in comparison with the ubiquity of such changes in chemical control?

Is the pattern real?

Before considering possible explanations for this discrepancy, it is useful to ask if the pattern really exists. It is reasonable to surmise that many more pest species, and more local populations of any given pest species, have been exposed to chemical control compared to biological control programs. If so, the prominence of documented cases of evolved responses to chemicals in the literature may simply reflect a difference in sample size.

Such a difference in sample size could arise for several reasons. First, if more species are targets of chemical control than biological control, there would simply be more opportunities to observe dramatic evolutionary responses to chemicals than to biological control agents. It is difficult to discount entirely the influence of such a statistical bias in the reported incidence of strong evolutionary responses by target pest species. Second, there may be biases in reporting, given that there is little economic incentive for the close moni-

toring of successful biocontrol systems. Such systems could exhibit slow decay in levels of control without exciting close attention, and thus be systematically underreported. Moreover, if the target pest evolves very rapidly, thereby escaping control, the attempt at biological control would be deemed a failure and so would not likely enter the published record. Indeed, many introductions of biological control agents have been successful, in that the population of the natural enemy persists, without leading to economic or effective control (D. Pimentel, *personal communication*). It is an important, if difficult, task to account for such biases in comparing chemical to biological control. Nonetheless, our interpretation of the literature is that the difference is indeed real and not a statistical artifact. In the remainder of this piece, we presume that the difference in evolutionary decay for the two kinds of control is genuine.

Hypotheses to Explain the Pattern

Imagine a grand-scale comparative experiment in pest control, where in region X, a chemical is applied annually, leading to an economically desirable value for q, whereas in region Y (distant from X) an effective biological control agent is established, also reducing the pest to q. Based on the pattern posited above, as time goes on one should see a decay in pest control in X, due to evolved resistance to the chemical control agent, whereas there should be less (or no) change in control in Y. But why? In general, slow (to nonexistent) evolutionary responses to environmental change (e.g., introduction of a control agent) are expected if any (or all) of the following is true: (1) there is no relevant genetic variation in the focal species; (2) there are countervailing selective pressures arising via negative genetic correlations among fitness components (tradeoffs); (3) selection is weak; (4) selection is inconsistent in direction. Finally (5), character evolution in a species may be present but masked in population-level parameters (e.g., q), because of coevolutionary responses by the control agent. Reasons 1–4 can, in principle, also constrain the evolution of resistance to either pesticides (Georghiou and Taylor 1986) or biological control agents. However, for reasons laid out below, we suggest 2, 3, and 4 should be more prevalent in biological control. Reason 5 is unique to biological control. We emphasize that these are not competing hypotheses, but instead are complementary causal factors, several (or all) of which could enter into explaining weak evolutionary responses by target pest species, as measured by shifts in q.

Lack of genetic variation.—The simplest explanation for a lack of evolutionary response following a change in the environment is a lack of relevant genetic variation (Bradshaw 1991). There is considerable suggestive evidence that this cannot account for the broad pattern identified above.

Many of the studies of evolution by natural selection in natural populations compiled by Endler (1986: Table 5.1) involved responses by prey species to predation. Although poorly studied in the field, studies in the laboratory and greenhouse of interacting host–parasitoid systems have shown evolutionary reductions in parasitoid reproductive rate suggesting the evolution of increased resistance in hosts (Takahashi 1963, Pimentel and Al-Hafidh 1965, Olson and Pimentel 1974, Pimentel et al. 1978, Bouletreau 1986), genetic variation in host susceptibility (Carton and Nappi 1991), and the evolution of lowered host susceptibility (Zareh et al. 1980). A recent study of the pea aphid *Acyrthosiphon pisum* and its dominant parasitoid *Aphidius ervi* showed substantial clonal variability in host susceptibility to the wasp (Henter and Via 1995), and additive genetic variation in the ability of the wasp to parasitize its host successfully (Henter 1995). Likewise, laboratory assays suggest that insects often possess considerable genetic variation in resistance to viruses, within and between populations (Briese and Podgwaite 1985, Channer and Gowen 1992).

Fueled by these studies and noting the multifaceted avenues by which a pest species *could* evolve resistance to a natural enemy (e.g., the many forms of refuges in time or space; Gross 1993, Hochberg and Holt 1995), we suggest that a simple lack of genetic variation cannot, in general, explain why so many pests appear not to have evolved resistance to introduced control agents, though it may well apply in particular cases.

Constraints on selection.—Henter and Via (1995) describe a fascinating case study of an interaction between a host (the pea aphid) and a parasitoid, in which genetic variation in resistance was shown to exist in the host, yet no evolutionary increase in resistance was observed over the course of six clonal generations. Furthermore, field data showed that parasitism was a common source of mortality, sufficiently intense that a significant response should have been observed. Henter and Via (1995) note several possible constraints on selection that may explain this puzzle, potentially relevant to all biological control cases in which one does not observe the evolution of enhanced resistance: (1) Resistance to the parasitoid may involve costs (negative genetic correlations in fitness components). For instance, ability to escape the parasitoid may reduce ability to acquire a limiting resource, or make the host more vulnerable to other mortality agents such as generalist predators or fungal pathogens. If such costs are sufficiently great, they may preclude a response to selection for resistance to the control agent (see Godfray 1994: 347). (2) There may be pest-by-control-agent genotype interactions, leading to frequency-dependent selection, and a reduced overall rate of selective advance. We will return to this issue below.

One hypothesis that could explain the difference in evolved resistance in chemical vs. biological control is

that negative genetic correlations in fitness components (i.e., trade-offs) are larger for traits providing resistance in biological control. The genetic architecture and interaction mechanisms involved in chemical and biological control may be different enough to imply different magnitudes of costs. Seger (1992) usefully distinguishes between qualitative and quantitative natural enemy–victim interactions. The former involve defenses like passwords (which have no direct costs in energy or time), whereas the latter are like bulwarks, diverting resources from reproduction or other fitness-related functions.

One might conjecture that the evolution of resistance to pesticides may often be password like; for instance, a single enzymatic change may hamper the biochemical mode of action of a pesticide. A lack of correlated functions (viz., costs) of such adaptive responses to chemicals may make host resistance evolutionarily labile. By contrast, evolution of resistance to biological control agents, in particular predators and parasitoids, might involve shifts in the optima of polygenic, quantitative characters (e.g., body size, phenology) under stabilizing selection in the absence of the control agent. This suggests implicit costs to the evolution of resistance to the control agent. Host–pathogen systems are believed to often have password-like elements in their coevolutionary race (e.g., gene-for-gene systems; Frank 1996, Rosenzweig 1996); this may explain why there appear to be more cases of evolved resistance in biological control with pathogens than with other natural enemies.

It is impossible to assess these suggestions at present, given the paucity of evidence regarding the genetics of resistance to natural enemies.

Strong population limitation need not imply strong selection.—Pest control requires that pest numbers be limited to sufficiently low q's by mortality from the control agent. A fair statement of the usual intuition about the relation of limiting factors and selective factors is provided by the following quotation from David Pimentel (1986:8):

> *Most insect pests produce from 100 to 1000 progeny per female. Thus, if the pest population is to be kept under control, from 98 to 99.9% of each generation have to be destroyed before they reproduce. If parasites provide this amount of control, clearly the intensity of parasite selection on the host will be intense . . . [thus] the parasite population must be the major cause of host mortality and therefore the major selection imposed on the pest population.*

But whether or not a high average mortality factor translates into strong selection depends upon heritable variance in fitness in the controlled population. Spatial structuring can lead to situations where strong limitation is decoupled from strong selection, averaged over the entire target population (Vermeij 1985).

This decoupling is currently being exploited in managing the evolution of resistance to chemical pesticides. For instance, Alstad and Andow (1995) recently developed a detailed simulation model showing how coupling treated and untreated fields in a patchwork could reduce the evolution of resistance by insect pests to insecticidal proteins (derived from *Bacillus thuringiensis* genes) that are expressed in transgenic plants. (Though transgenic plants and inundated agents are often discussed under the rubric of ''biological control,'' in some important respects these systems resemble chemical control. In particular, these control strategies target the pest via single or multiple applications, with little or no recourse to population responses via time-delayed, density-dependent feedbacks. Moreover, these systems have little opportunity for evolutionary responses by the control agent to the pest.) The reason selection is weak in these scenarios is that a substantial fraction of the pest population in the control situation resides in ''refuge'' habitats, where they are not exposed to the pesticide.

We suggest that effective biological control systems, in which natural enemies stably regulate target pests at low densities, may have as a generic feature the spatial structure that Alstad and Andow (1995) suggest is needed to hamper evolution of resistance to pesticides (see also Bouletreau et al. 1986). A conundrum arising in biological control is that, in the absence of complete extirpation of the pest, one must ensure the persistence of the natural enemy population. Yet the essence of strong predator–prey and other natural enemy–victim interactions is that they are dynamically unstable, often strongly so, so that the control agent is at risk of extinction. The resolution of this dilemma may often involve spatial heterogeneity.

The population dynamics of host–parasitoid systems have received much more attention than other classes of natural enemy–victim interactions relevant to control (Murdoch 1990), so we concentrate on them. One conclusion that emerges from many lines of theoretical work is that spatial heterogeneity in attacks may be key to the effective regulation of hosts at low q values by parasitoids (e.g., Beddington et al. 1978, Hassell 1978, May and Hassell 1988). Two basic stabilizing mechanisms involving space, bracketing a broader range of possibilities, are: (1) within-population spatial heterogeneity; (2) metapopulation (colonization–extinction) dynamics. These involve different spatial scales and assumptions about the mechanisms permitting population persistence, yet both illustrate how spatial patterns in attacks that lead to persistent host–parasitoid interactions may also foster evolutionarily stable biological control.

1. *Within-population heterogeneity.*—Consider first a simple yet instructive model in which a species with haploid genetics occupies two habitats, A and B. Each generation, individuals sort out with a fixed fraction e

of individuals in habitat A (each with an expected fitness of W_A); the remainder are in habitat B (with expected fitness W_B). Mean fitness across the two habitats is $W = eW_A + (1-e)W_B$.

Fitnesses are determined by a phenotypic variable v; the original population is fixed at $v = v'$. A small increase in v is selectively favored if it increases fitness averaged over both habitats, or $dW/dv = e\ dW_A/dv + (1-e)dW_B/dv > 0$. If v is expressed just in A, the strength of selection on v is directly proportional to the population-wide exposure of individuals to habitat A, i.e., e. If few individuals occupy habitat A, the strength of selection for improving habitat-specific adaptation there (or removing mutations with habitat-specific deleterious effects) is necessarily weak. With trade-offs in phenotypic effects between habitats, such that if $dW_A/dv > 0$ then $dW_B/dv < 0$, an increase in v enhancing fitness in habitat A will be actively disfavored if e is sufficiently small.

In effect, natural selection is automatically biased toward the habitats most often experienced by the members of a population. This theme has been explored recently by several authors examining evolution in spatially heterogeneous environments, using different assumptions about dispersal (Brown and Pavlovic 1992, Holt and Gaines 1992, Kawecki 1995, Holt 1996). The basic message is that demographic asymmetries induce ''weighting'' in natural selection, favoring adaptation in habitats harboring the largest fraction of a breeding population. Models for the evolution of quantitative characters expressed in each of two habitats, with a strong genetic correlation between the character values, often show a long transient phase in which a population is maladapted to one of the two habitats, before finally settling into an equilibrium where the phenotype is at the local optimum in each habitat (Via and Lande 1985). This phase can be particularly long for the habitat that contributes least to the overall population.

The spatial structure in these evolutionary models is broadly comparable to the spatial structures believed to ensure both system persistence and effective host population control in host–parasitoid systems. For instance, host–parasitoid systems with ''proportional refuges'' (Hassell 1978, Holt and Hassell 1993, Hochberg and Holt 1995), have a fraction e of hosts exposed to parasitoids each generation, whereas $1-e$ are in the refuge. Proportional refuges are particularly likely to permit persistent host–parasitoid interactions when the refuge is a sink for the host (Holt and Hassell 1993). In the above model for adaptive evolution over two habitats, ''habitat type'' may be identified with being within, vs. outside, the refuge. If increased ability to escape parasitism outside the refuge comes at too great a cost in reduced fitness in the refuge, the net effect may be that selection will not improve adaptation by the target pest to the control agent. Even without trade-offs, a weaker strength of selection outside the refuge leads to a reduced ability to purge the population of deleterious mutations, reducing host fitness outside the refuge (Kawecki 1995, Holt 1996*a*, *in press*). By contrast, selection on the parasitoid is entirely focused in the habitats it occupies. Simulation studies suggest that unstable (but bounded) population dynamics weakens selection on exposed hosts to escape parasitism (Holt et al., *in press*).

2. *Metapopulation dynamics.*—Host–parasitoid systems with strong top-down control might persist, not because of local stability, as in proportional refuge models, but because of regional dynamics, with local extinctions in occupied patches balanced by colonization of empty patches (e.g., Hassell et al. 1991, Taylor 1991). Assume a large number of patches are available in a landscape. Each patch can be qualitatively characterized as empty, with the host alone, or with both the host and parasitoid. Assume the parasitoid reduces host numbers in jointly occupied patches so effectively that the risk of host extinction (followed by parasitoid extinction) is greatly increased, with a probability of one over some short time period. Because of this strong ''top-down'' limitation in local abundance, host populations in patches containing parasitoids should contribute relatively few individuals toward colonization of empty patches; the majority of propagules instead should come from patches with just the host.

Assume that in each patch with both host and parasitoid, there is the potential for evolution of resistance by the host. In each patch there is a race between two processes: a demographic process, namely rapid extinction due to strong limitation by the parasitoid, and an evolutionary process of local adaptation by natural selection (Gomulkiewicz and Holt 1995). If extinctions take place too rapidly, then whatever evolutionary progress was made in those patches experiencing extinction is expunged. Because empty patches are largely colonized from patches without the parasitoid, patches in which local coevolution is occurring may make little overall contribution to the total breeding population of the host, relative to patches containing the host but no parasitoid (where there is no opportunity for the evolution of resistance). Selection should in this case be weighted toward adaptation to conditions in transient refuge patches without parasitoids, thereby making less likely the evolution of host adaptations to escape parasitism. Put another way, by virtue of being a very effective limiting factor on a host, and persisting via a regional balance of local extinctions and recolonizations, a natural enemy may automatically render itself into an ineffective *selective* factor on its host (despite being both locally and regionally a very effective limiting factor). This makes less probable evolution of increased resistance in the overall host population.

This scenario warrants more detailed theoretical attention. Preliminary results (R. D. Holt and R. Go-

mulkiewicz, *unpublished data*) suggest the general conclusions are sound, given realistic assumptions about within-patch evolutionary dynamics.

These scenarios suggest that persistent biological control at low q may often necessarily have, as an emergent property, the spatial structure believed to retard the evolution of resistance to pesticides (May and Dobson 1986, Alstad and Andow 1995), particularly when biological control is via specialist natural enemies. However, in some circumstances effective biological control may be stable without refuges or metapopulation dynamics (D. Pimentel, *personal communication*); for instance, small rates of augmentation may stabilize strong, spatially homogeneous host–parasitoid interactions at low q. If biological control is achieved without producing a heterogeneous selective environment, the above logic will not apply. We predict that such examples of biological control may be vulnerable to the breakdown of control due to evolved resistance in the target pest species.

We are unaware of data that directly allow us to assess the expectation that effective chemical agents have on average been more uniformly applied over the agricultural landscape, compared to the spatial patchiness emerging from the population dynamics of biological control. However, the ubiquity of evolved resistance to chemical control suggests to us that heterogeneous selective environments hampering evolved resistance by pests have historically been the exception, not the norm, in chemical control.

Temporally variable selection pressures in biological control.—Natural enemy dynamics can be influenced by many factors other than host availability, such as the weather, which varies in space and time. Moreover, the dynamical instability inherent in strong natural enemy–victim interactions may translate into temporally varying selection on target species. Theoretical analyses suggest that unstable population dynamics can weaken selective responses by target host species to effective control agents (Holt et al., *in press*). However, because chemical application and persistence also vary temporally, it is difficult to make firm a priori predictions about whether chemical control or natural enemies might provide the more consistent force of selection.

Predators and parasitoids may have plastic behavioral responses to changes in their prey or hosts, for instance by focusing search efforts in whichever microhabitats are currently most profitable. This behavioral plasticity implies that these classes of natural enemies do not, in fact, present a single selective force on a target pest, but instead a shifting suite of selective forces, which vary qualitatively in direction among host generations. This inconsistency in selection arising from labile enemy behavior could weaken evolutionary responses by hosts to introduced control agents. By contrast, chemical pesticides have a fixed physiological mechanism by which they harm pest fitness and so are likely to provide a rather constant direction for selection.

Selection that does not depend upon genotype frequency tends to increase either the average growth rate or the equilibrial abundance of the species experiencing selection (Roughgarden 1979). In the context of pest control, both these effects translate to weakened control of the pest species. If instead selection is frequency dependent, there is no necessary relation between selection and these mean population level parameters. Chemical agents, which are behaviorally and evolutionarily static, may be less likely to induce frequency-dependent selection on target pests than arise from biocontrol agents, which may be behaviorally plastic and also have their own evolutionary dynamics. An abstract, general reason why biological control may exhibit less evolutionary breakdown than chemical control is that the former should more often involve frequency-dependent selection. Little formal theory exists to date that bears on these suggestions.

Balanced coevolutionary dynamics.—The above hypotheses emphasize host genetics (e.g., negative genetic correlations) and the influence of behavior and population dynamics on host evolution and consequent control. One additional key difference between biological and chemical control is that biological control agents are living organisms capable of evolutionary responses. One intuitively plausible explanation for the widespread absence of apparent evolutionary change in biological control systems is that rapid evolutionary responses by the control agent effectively counterbalance evolutionary change in the host (Zareh et al. 1980, Pimentel 1986). Rather than no host evolution, evolution could either be constantly ongoing (as in an arms race) or reach a coevolutionary equilibrium, but in such a way that there is no net effect on the level of control. In this scenario, for biological control to be evolutionarily stable, each time the pest becomes better at avoiding the control agent, and hence increases in abundance, the control agent should be selected to more effectively capture the pest, reducing its numbers.

There are two potential limitations to coevolution as a full explanation for the absence of decayed resistance in biological control. First, it is not necessarily the case that improved ability by the pest to withstand the control agent automatically leads to stronger selection on the agent to capture the pest, nor that prey will respond so as to counter increases in predator capture abilities. Abrams (1986, 1990, 1991) sketches a number of plausible theoretical counterexamples, hinging upon correlated effects (see also Vermeij 1994). Second, given that the system tends towards a coevolutionary equilibrium, it may not necessarily be an equilibrium where the host is limited to a low value of q.

A detailed analysis of the influence of evolutionary dynamics on the level of control requires one to con-

sider models that simultaneously track population dynamics and character evolution. Several authors (e.g., Rosenzweig 1973, Abrams 1986, Rosenzweig et al. 1987, Saloniemi 1993) have addressed such issues in the general literature on predator–prey coevolution. However, there have been relatively few attempts to address the evolution of control using models tailored to biological control systems since the pioneering studies by Pimentel and his associates (e.g., Pimentel et al. 1975, Pimentel 1984), with the exception of vertebrate host–pathogen systems (Dwyer et al. 1990). We (Hochberg and Holt 1995) have recently examined host–parasitoid dynamics, using a relatively realistic model for the interacting populations, but permitting as well evolution in phenotypic characters that influence the exposure of hosts to attack. This model predicts that in a wide variety of reasonable circumstances, initially effective biological control may be evolutionarily transient. Whether or not this conclusion is robust across systems remains to be seen.

Despite these caveats, it seems likely to us that predator–prey coevolution in the end will prove to be an important (and even essential) part of the explanation for the apparent discrepancy in the persistence of biological, vs. chemical, control. However, we also suggest that coevolutionary checks on the decay of control are likely to be more effective, when combined with the other factors discussed above. For instance, the weakening of selection on prey arising from the spatial and temporal heterogeneities inherent in persistent, strong predator–prey interactions (and which may often be absent in chemical control) may systematically bias coevolutionary dynamics in favor of the control agent.

The famous ''life–dinner principle'' (Dawkins and Krebs 1979) states that because a rabbit runs for its life, whereas a fox runs merely for its dinner, the coevolutionary race should be biased in favor of the rabbit. This is not always to be expected (Abrams 1986). In particular, if foxes are so effective in exposed habitats that most rabbits are restricted to warrens—refuges where they happily live out their lives and are exposed to local selection pressures, without encountering a fox—the average rabbit isn't even in the race, whereas all the foxes still are: the coevolutionary race is then automatically tilted toward the fox.

Discussion

We hope that this thought piece will motivate a more determined search for instances of evolved host resistance and coevolution in biological control, and further theoretical and empirical explorations of the environmental and biotic conditions in which biological control is likely to be evolutionarily stable, rather than transient. Few data currently exist to assess the relative importance of the hypotheses we have presented (e.g., regarding correlated effects of resistance). We conclude with a brief discussion of evolutionary patterns one might expect to see in biological control systems.

If our hypotheses are reasonable, we suggest that one might expect the following patterns in the ordering of prevalence of evolved resistance:

1) with respect to the control agent, *chemicals* > *microparasites* > *macroparasites* > *predators*;

2) with regard to application strategy, *inundative* > *augmentative* > *single release* (''inundative'' denotes periodic releases of the control agent from stock populations, usually over large areas; ''augmentative'' refers to more modest releases);

3) with respect to the areal scope of application, *geographical* > *regional* > *local.* These axes are not independent: chemicals tend to be applied inundatively; predators and parasites by contrast are released once or a few times, and usually only at local or regional scales. Assembling data to assess these hypotheses and disentangling the relative contributions of these three axes to the evolutionary dynamics of pest control is a worthy challenge for future work.

The arguments of this paper help clarify some factors that may foster, or hamper, evolutionary shifts in biological control of pest species.

When biological control is ineffective to start with, it is not surprising that there might be little evolutionary change in the pest. It is more counterintuitive that highly effective control may also be correlated with rather ineffective selection on the pest to avoid parasitism. The reason is that if effective control is stabilized by spatial heterogeneity, there may be relatively few host individuals that actually encounter parasitoids, at least among those hosts that contribute to future generations (e.g., because these hosts live in patchy refuges). Because evolution is automatically weighted toward patches with most hosts, there may be only weak selection to improve adaptations to withstand parasitism. Biological control is most likely to go awry because of evolutionary dynamics fostering increased host resistance in situations where ecological stability arises from factors other than spatial heterogeneity (see Hochberg 1996).

As noted earlier, similar conclusions apply to the evolution of resistance to chemical pesticides (e.g., Comins 1977, Taylor and Georghiou, 1979, 1982, May and Dobson 1986, Alstad and Andow 1995). For instance, Croft (1992) suggests as a principle for limiting resistance, ''Leave unselected populations of pests in refugia or maximize immigration and hybridization of susceptible biotypes with selected populations.'' Our basic point is that the spatial structures needed for highly successful biological control to persist over ecological time, may as a byproduct, automatically generate the scenario espoused by Croft, and articulated more recently by Alstad and Andow (1995) and others, for reducing evolved resistance to chemical control.

The experience of chemical control is that strong

limitation of pests by chemicals is vulnerable to decay because of evolution in the pest. It is possible that some successful biological control systems will also prove ultimately to be evolutionarily transient (as in the control of rabbits by myxomatosis). We suggest that evolutionary stability in biological control is most likely, given synergistic combinations of the processes discussed above. For instance, a parasitoid that can effectively limit its host (the target pest) after introduction may be more likely to maintain this control over evolutionary time spans if ecological stability arises from spatial heterogeneity (leading to weak selection on the host), host counteradaptations are costly, and the parasitoid can mount effect coevolutionary responses to host changes.

The management of pest evolutionary responses is an exciting chapter in the emerging field of ''applied evolutionary biology'' (Holt 1996*b*). This field will provide a testing ground for studies of the evolution of adaptation to tolerate severe, novel environmental stresses (Denholm and Rowland 1992). We suggest that in sculpting control programs to minimize evolved resistance, either to chemical or biological control, it is essential to consider spatial and temporal heterogeneity in the selective environment imposed on the target species, the costliness of potential defenses, and potential behavioral and coevolutionary responses by the control agents. Scant data exist at present to assess the relative importance of these factors.

One general reason to prefer biological over chemical control strategies, beyond the usual reasons, is that effective biological control may in the end be more evolutionarily stable, by virtue of the basic ecology of and evolutionary constraints on strong, persistent natural enemy–victim interactions. If effective biological control indeed tends to be evolutionarily stable, whereas chemical control is evolutionarily evanescent, this to us provides a compelling reason for emphasizing biological control in integrated pest management schemes.

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