

Microevolution in biological control: Mechanisms, patterns, and processes

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Abstract

Microevolution may determine both the safety and efficacy of classical biological control. Despite a growing body of literature, there are several key unanswered questions regarding the role of evolution in biological control: (1) How common is local adaptation of natural enemies to their hosts or the environment in the native range? How critical is it for success of biological control to find locally adapted agents for importation? (2) Does adaptive evolution following introductions play an important role in biological control? (3) Do introductions of biological control agents impose bottlenecks in population size that reduce genetic variation, and is reduced genetic variation associated with low fitness and poor performance? (4) How great is the risk of evolution of host range of biological control agents? (5) What is the risk of target pests evolving resistance to biological control agents? If pests evolve increased resistance, will biological control agents evolve mechanisms to overcome that resistance? Here, we review the four fundamental processes of microevolution, and discuss how they interact in the context of biological control. We discuss our current state of knowledge regarding the outstanding questions, highlight the types of experiments that can address them, and suggest ways to use microevolution to define risks, and enhance efficacy and safety of biological control.

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1. Introduction

In the field of biological control we apply ecological theory concerning population dynamics to the control of pest populations. Links between biological control and ecology are strong, and important developments in ecology have emerged from biological control, such as Huffaker's (1958) classic work on predator–prey population cycles. The links between biological control and evolutionary biology are less developed. For classical biological control, where specialized natural enemies from the native range of an exotic pest are introduced to control that pest,

the main role of evolutionary biology has been to delineate phylogenetic relationships of taxa (Beard, 1999; DeBach, 1964). Such relationships are critical both for identifying pests and biological control agents and for ascertaining which species are relatives of target pests for use in host-range testing (Briese and Walker, 2002; Wapshere, 1974).

The importance of ongoing microevolution in biological control is less certain, despite a growing body of literature. The work to date includes conceptual application of population genetics to biological control (e.g., Force, 1967; Hopper et al., 1993; Lucas, 1969; Remington, 1968; Roderick, 1992; Roush, 1990), development of hypotheses (Blossey and Nötzold, 1995; Müller-Schärer et al., 2004), and models (Holt and Hochberg, 1997), genetic surveys of biological control

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agents (e.g., Baker et al., 2003; Briese et al., 1996; Coll et al., 1994; Espiau et al., 1997; Follett and Roderick, 1996; Hufbauer et al., 2004a; Krafur et al., 1997; Myers and Sabath, 1981; Vaughn and Antolin, 1998) and targets of biological control (e.g., Bruckart et al., 2004; Burdon and Brown, 1986; Gaskin and Schaal, 2002; Hufbauer et al., 2004b; O'Hanlon et al., 1999), experimental work on the evolution of interactions between biological control agents and their targets (Hufbauer, 2001, 2002; Lemasurier and Waage, 1993; Salt and van den Bosch, 1967; van den Bosch, 1964), and reviews (Hopper et al., 1993; Roderick and Navajas, 2003; van Klinken and Edwards, 2002).

Although much has been learned from this research, vital questions regarding the role of microevolution in safe and effective biological control remain unanswered. In particular, the following five sets of questions warrant further attention: (1) How common is local adaptation of natural enemies to their hosts in the native range? How critical is it for success of biological control to find locally adapted agents for importation? (2) Does adaptive evolution following introductions play an important role in biological control? (3) Do introductions of biological control agents impose bottlenecks in population size that reduce genetic variation, and is reduced genetic variation associated with low fitness and poor performance? (4) How great is the risk of evolutionary change of host range of biological control agents? (5) What is the risk of the evolution of resistance in target pests? If pests evolve increased resistance, will biological control agents evolve mechanisms to overcome that resistance?

Here we evaluate the potential roles of microevolution in biological control. We focus on classical biological control, though much of the discussion is relevant to other modes of biological control. We first lay some groundwork by reviewing the fundamentals of evolution and the four main processes that lead to microevolutionary change: mutation, genetic drift, selection, and gene flow. Then, we underscore how these processes interact and give rise to population structure and local adaptation. Finally, we present evidence bearing on the five sets of questions outlined above in the context of biological control, highlighting gaps in the state of our knowledge and suggesting types of research that could help fill them.

2. An evolution primer

Evolution is defined simply as change over time of the proportion of individual organisms that differ genetically in one or more traits (Futuyma, 1998), or from a microevolutionary perspective, change in the genetic composition of a population (Hartl and Clark, 1989). A common misconception is that new mutations or recombination are required for evolutionary change. This is

not the case. As long as standing genetic variation exists, both adaptive and neutral evolution can proceed without novel mutations. Four major processes lead to microevolution: mutation, genetic drift, selection, and gene flow.

2.1. Mutation

Mutation is the fundamental source of all genetic variation. Before the development of molecular genetics, mutations were identified by their effects on the phenotype. Currently, mutation typically is defined more narrowly as an alteration of DNA sequence. DNA mutations include both point mutations where one base pair of DNA is substituted for another, and insertions and deletions (indels) where one or more base pairs of DNA are inserted into, or deleted from, a DNA sequence. Recombination between DNA sequences also can generate novel combinations of sequences. Recombination is not mutation as it is currently understood at the molecular level, however recombination can give rise to phenotypic effects that may be interpreted as mutations. Estimates of mutation rates are often smaller than 10^{-6} per base pair each generation, but vary by orders of magnitude, depending upon both the taxa and the region of the genome in which they are measured (Futuyma, 1998; Lande, 1995). Mutations that increase fitness are even rarer: most mutations are thought to range from neutral to deleterious in their effects on fitness, with only a very small proportion being advantageous. Despite the infrequency of mutations, particularly beneficial ones, the chance that a mutation will occur that will influence some aspect of a complex trait is increased because life history traits and other complex ecologically important traits are affected by a large number of loci (Houle et al., 1996; Houle, 1998). However, new mutations exist at low frequency (on the order of $1/2N_e$, for diploid organisms with effective population size N_e), and are unlikely on their own to influence microevolution in classical biological control, as long as population sizes are large and selection is not particularly strong (Lande, 1995).

2.2. Genetic drift

Genetic drift is change in the frequencies of alleles in a population due to random sampling effects. Genetic drift reduces variation within populations, and can increase differences between populations as alternate alleles become fixed (i.e., present in all individuals) or lost. The rate at which alleles go to fixation or are lost depends strongly upon effective population size. In small populations, the chance sampling effects that constitute genetic drift can have a larger proportional influence on the next generation than in large populations. Simply due to chance alone, alleles can become fixed even if they

are maladaptive, and lost even if they increase fitness. Thus, small isolated populations are influenced more strongly by drift than large populations, leading to a situation where the individuals may not be well adapted to their environment. Because of the relationship between genetic drift and population size, genetic drift underlies our understanding of the genetic consequences of bottlenecks in population size and our understanding of founder events. Bottlenecks in population size are known to reduce variation at neutral loci (Nei et al., 1975), and are associated with inbreeding depression (Frankham, 1998; Woodworth et al., 1994). However, bottlenecks may actually increase variation in quantitative traits through a proposed complex process that effectively converts epistatic variation to additive genetic variation (for detailed explanations and discussion see Cheverud et al., 1999; Goodnight, 1987, 1988; Pray and Goodnight, 1997). However, the strength and importance of such an effect is debated (Knowles et al., 1999; Lopez-Fanjul et al., 2000, 2002; Naciri-Graven and Goudet, 2003; Regan et al., 2003; Saccheri et al., 2001; Whitlock and Fowler, 1999).

2.3. Selection

Selection is the differential survival and reproduction of phenotypes, and is the process typically leading to adaptation. The importance of selection in natural populations varies tremendously, but can be very strong (Endler, 1986). The strength of natural selection can be measured by comparing the fates of discrete phenotypes or by estimating the relationship between fitness and continuous phenotypes. For the latter situation selection gradients estimate selection acting directly on a single trait of interest, while selection differentials estimate both direct selection acting on a trait, and indirect selection acting on correlated traits. Estimates of linear (i.e., directional) selection gradients (β) are fairly intuitive: β is the slope of the line describing the relationship between the value of a trait (in units of standard deviations of that trait) and relative fitness. Steep slopes indicate strong directional selection, and shallow slopes indicate weak selection. In a recent review on the strength of selection in natural populations, Kingsolver et al. (2001) found that linear selection gradients were approximately exponentially distributed, with a mean of 0.22. The median value was a relatively modest 0.16, but there was a long tail of larger values (i.e., steeper slopes), with 13% of the studies having $\beta > 0.5$.

Selection acts on phenotypes, not genotypes, and it will only lead to adaptive evolution when there is a genetic basis to differences between phenotypes. When natural selection differs across environments, populations may become adapted to their local environment (local adaptation), such that individuals in the populations have higher average fitness in their home environ-

ment than alternate environments. As discussed below, local adaptation plays an important role in ideas about the efficacy and safety of biological control.

2.4. Gene flow

Gene flow is the exchange of genes among populations. Gene flow homogenizes populations through keeping them genetically similar to each other. With no or limited gene flow populations can diverge from each other via mutation, genetic drift and/or selection. Gene flow also can be a creative force by bringing new alleles into populations (Lenormand, 2002; Slatkin, 1987). Estimates of gene flow can be used to describe genetic differences among populations as well as to infer the rate of migration among populations long term (Roderick, 1996). Recently developed approaches based on assignment tests and Bayesian approaches can be used to infer more recent movements of individuals (Davies et al., 1999; Roderick, 2004). Estimating rates of movement from data on gene flow in the native range may help in planning and implementation of biological control. If movement is high, then a single release may be sufficient to cover a wide area, while if movement is low, then more active redistribution will be needed. However, rates of gene flow rarely have been studied prior to an introduction (but see Briese et al., 1996; Michalakis et al., 1993). Work on gene flow following introduction suggests that biological control agents can exhibit restricted movement (e.g., Follett and Roderick, 1996; Vaughn and Antolin, 1998) or frequent dispersal (e.g., Coll et al., 1994; Krafur et al., 1997).

2.5. Interactions between evolutionary processes

The patterns we see around us—how neutral variation is structured within and among populations, whether populations are adapted to their local environment—are influenced by interactions between these processes. Natural selection, genetic drift, and gene flow, together shape the structure of adaptive and neutral genetic variation within and among populations and local adaptation is due to genotype by environment interactions for fitness. Whether populations become locally adapted depends upon the balance between selection, genetic drift, and gene flow. If populations are small, alleles favored by selection may be lost by genetic drift and selection must be quite strong to overcome chance events, while if populations are large, then even weak selection can lead to adaptive evolution, as only the rarest alleles are likely to be lost through drift. Local adaptation generally is favored by low gene flow, but if selection is strong enough, local adaptation can occur even in the face of high gene flow. However, particularly at the edges of a species range, if populations receive a high proportion of migrants that are adapted to other

environments, then they may be maladapted to their local environments (Lenormand, 2002).

3. Outstanding questions

3.1. How common is local adaptation of natural enemies to their hosts or the environment in the native range? How critical is it for success of biological control to find locally adapted agents for importation?

Whether or not a potential biological control agent is locally adapted in the native range is a vital piece of information. It often is assumed that agents are locally adapted to the climate in the native range. Rarely are experimental data on adaptation to climate collected (but see Mason and Hopper, 1997), but it is nonetheless sensible to export agents from regions of the native range that match the climate of the release area in the introduced range reasonably well (e.g., van Klinken et al., 2003). Climate data along with the physiological tolerances of biological control agents often are used in evaluating limits to the success of biological control following a release (e.g., Dennill and Gordon, 1990; Julien et al., 1995; Lactin et al., 1997; McClay and Hughes, 1995; Nowierski and Fitzgerald, 2002; Scott, 1992). These studies and others, suggest that adaptation to climate regimes is common, and therefore that prudent climate matching is important in the success of biological control.

What is less clear is the importance of finer scale adaptation of biological control agents, in particular adaptation to genotypes of the target pest. Here, the literature on host–parasite interactions provides a resource, because most specialist biological control agents (pathogens, parasitoids, and insect herbivores) can be considered parasites. Empirical work illustrates that parasites often are adapted to the local populations of their hosts, such that they perform better on genotypes from their local population than others (e.g., Burdon and Jarosz, 1991; Lively and Jokela, 1996; Parker, 1985; Via, 1991a,b; papers in Cory and Myers, 2004; Kawecki and Ebert, 2004; Mopper and Strauss, 1998), but this is not always the case (Hufbauer, 2002; Imhoof and Schmid-Hempel, 1998; Kaltz et al., 1999), in part because a state of local adaptation can fluctuate through time and space (Dybdahl and Lively, 1998; Lively, 1999; Mopper et al., 2000; Thrall et al., 2002).

Three factors emerge as important to the evolution of local adaptation of parasites to their hosts: parasite host range, relative life span of host and parasite, and relative migration rates and metapopulation structure of host and parasite.

- (1) Parasites with a narrow host range are more often locally adapted than parasites with a broader host

range (Lajeunesse and Forbes, 2002), perhaps due to time-lags in adaptation of parasites with broad host ranges.

- (2) Kawecki and Ebert (2004) summarize work on the relative life-spans of hosts and parasites: short-lived parasites attacking long-lived hosts (e.g., perennial plants) tend to be locally adapted, while parasites that have similar life spans as their hosts do not. This is thought to be because short-lived parasites have time to adapt to particular host genotypes (Edmunds and Alstad, 1978, papers in Mopper and Strauss, 1998). At the other extreme, with host–parasite systems such as insects and parasitoids, the parasite develops on a single host per generation, and its offspring develop in different host individuals. In this case, selection to perform well on a host genotype may differ from selection the generation before. This situation can lead to negative frequency dependent selection (rare host genotype advantage) (Dybdahl and Lively, 1998; Lively and Dybdahl, 2000), and dynamically changing patterns of local adaptation and maladaptation of the parasites (Thompson et al., 2002).
- (3) Metapopulation dynamics, and partially overlapping distributions of parasites with their hosts (Gandon, 2002; Nuismer and Kirkpatrick, 2003) also can influence whether and when local adaptation will occur. Relative rates of dispersal of hosts and parasites across a metapopulation shape patterns of local adaptation in host–parasite systems (Gandon and Michalakis, 2002). If host migration rates are lower than those of the natural enemies, then the host population represents a stable environment to which the natural enemy can adapt. If host migration rates are higher than those of natural enemies, then models suggest that parasites may not be able to keep up with the new host genotypes, and are less likely to be locally adapted.

Thus, though parasites are commonly locally adapted to their hosts, they are not always, and it is not safe to assume that a potential biological control agent will be adapted to the target pest in the native range. Indeed, either the parasite or the host may be locally adapted to the other and thus, in the language of an evolutionary arms race, “winning” the interaction.

If there is local adaptation of a natural enemy to invasive pest genotypes, will using the locally adapted genotypes or population lead to more effective biological control? For the answer to this question to be yes, the locally adapted natural enemies should have higher rates of population increase or be more damaging to their local hosts. Dybdahl and Storfer (2003) make a clear distinction between the ability of a parasite to infect or develop on its host (infectivity), and the degree of damage done to the host by a parasite (virulence). Often the

two are related biologically because a faster individual or population growth rate of the parasite is associated with greater damage to the host. However, infectivity and virulence are not always correlated.

Particularly relevant to biological control is that parasites can infect hosts but have low virulence on those hosts. This rarely occurs with parasitoids, because most parasitoids kill their hosts on emergence (though see English-Loeb et al., 1990), and so the ability of a parasitoid to successfully ‘infect’ a host and its virulence on that host are generally treated together. Infectivity associated with low virulence is not uncommon in plant pathogens, where avirulent forms of a pathogen can infect their hosts without negatively affecting plant fitness (e.g., Saunders and O’Neill, 2004). In the plant–insect literature this issue is often viewed from the perspective of a host plant; among plants that are susceptible to a particular herbivorous insect species, some genotypes may be tolerant, while others incur fitness costs when fed upon. Thus, the insects can ‘infect’ both categories of genotypes, but only are virulent on the latter. Clearly, a biological control agent must be able to develop on a target pest (i.e., must be infective), but the ability to develop does not guarantee virulence. Unfortunately, most work on local adaptation of parasites either conflates infectivity and virulence in the measures of adaptation, or explicitly focuses on infectivity as a measure of adaptation, leaving the question of whether local adaptation is positively or negatively associated with virulence unanswered (but see Ebert, 1994). However, much can be learned from examining work on the evolution of virulence apart from local adaptation.

Models of the evolution of virulence suggest an important role for the mode of parasite transmission (Bull, 1994; Day, 2001; Ebert, 1994). Vertical transmission occurs when hosts pass their parasites directly to their offspring, while horizontal transmission is the movement between different host individuals, and can range from transmission only between closely related individuals, to transmission between different species of hosts.

The simplest models of the evolution of virulence predict that decreased virulence may evolve in systems with vertical transmission because if the host dies before the parasite reproduces then the parasite will not be transmitted to a new host. Free-living parasites sometimes may have life cycles that are akin to vertical parasite transmission, when the parasite is short-lived and entire populations develop on a long-lived host, the offspring of a single parasite are found on the same host (e.g., as can occur with natural enemies of long-lived plants). This also may lead to reduced virulence. Selection for decreased virulence is predicted to be weaker in systems with horizontal transmission because host reproduction is not required for successful parasite transmission (Bull, 1994; Day, 2001). However, the evolution of attenuated virulence can occur in horizontal transmission systems

as well. A classic example of this phenomenon comes from the biological control of rabbits introduced to Australia with myxoma virus (Fenner, 1983). The virus initially lead to death in close to 100% of infected rabbits. When the population density of the rabbits had decreased, those forms of the virus that were acutely fatal were not transmitted as successfully to a new host and declined in frequency in the population, while less virulent forms allowed for more time for contact between sick and healthy rabbits (via mosquito and flea vectors), and therefore increased in frequency.

A recent theoretical advance in understanding the evolution of virulence is the explicit incorporation into models that parasites may attack more than one variety or species of host (Gandon, 2004). With more than one host, levels of virulence in horizontally transmitted parasites will depend upon several factors, most importantly, whether virulence on the different hosts is influenced by the same, or physiologically related traits, or is independent. When virulence on two hosts is influenced by the same or physiologically related traits, the evolution of increased virulence generally is expected.

In the context of biological control, the tendency of vertically transmitted parasites (or, potentially, highly specialized and sedentary parasites) to evolve high infectivity associated with attenuated virulence suggests that the most damaging parasites may not have the narrowest host ranges (Hufbauer et al., unpublished).

The research described above leads to two main conclusions regarding the local adaptation of parasites to hosts within their native range: (1) Local adaptation of parasites to hosts is common, but not universal. (2) The relationship between local adaptation and virulence requires further study. In some parasites with narrow host ranges where local adaptation is found, virulence may be attenuated.

Despite the unknowns, ideas about local adaptation have refined the idea that finding the region where a target pest species evolved (the center of origin; McClay et al., 2004) may aid in finding successful biological control agents. Evidence for local adaptation suggests that narrowing the search from the evolutionary center of origin of an invader to the locations that were the source of an introduction may improve efficacy. This has led to the use of historical records and molecular markers to identify the provenance of invasions (Nissen et al., 1995; Roderick, 2004). Assuming that natural enemies at the area of provenance will be locally adapted to the particular genotypes of the target pest that are invasive is based on the underlying idea that there is a coevolutionary arms race and that the natural enemies are “winning” that race rather than the hosts. Targeting the area of provenance of an invasion also assumes that the invasive genotypes of the pest have native counterparts, and are not a consequence of hybridization, severe bottlenecks, or rapid adaptive evolutionary changes.

While assuming that biological control agents are locally adapted, and that locally adapted agents are more effective may always not be valid, it nonetheless is clear that genotypes of invasive pests do vary in their susceptibility to their natural enemies (e.g., Bruckart et al., 2004; Burdon et al., 1981; Henter and Via, 1995; Lym et al., 1996; Nahrung and Murphy, 2002; Sobhian et al., 2003), and that genotypes of actual or candidate biological control agents vary in their ability to attack different genotypes and species of target pest (Fumanal et al., 2004; Henter, 1995; Messing and Aliniaze, 1988; Peng et al., 2004; Volchansky et al., 1999) and this can influence the level of control achieved. Because of this variation, it is becoming common practice to evaluate whether a candidate biological control agent is able to develop and is damaging to the genotypes of a pest in locations where it is targeted for control.

In practice, most biological control agents are sought where feasible both geographically and financially. Only rarely are the genetic data available to pinpoint the area of provenance of an invasive species, and thus using such information to target the search for biological control agents is also rare. We are aware of only one case where evidence for local adaptation of an agent to invasive genotypes was studied prior to release for biological control: that of an eriophyid mite (*Floracarus perrepae* Knihinicki and Boczek) on Old World climbing fern (*Lygodium microphyllum* (Cav.) R. Br.) (Goolsby et al., 2003, 2004a). Goolsby et al. (2004a) used cpDNA sequence data to find populations in the native range related to invasive populations in Florida, then compared the performance of mites from that location and others on genotypes from the native and invasive range. They found strong evidence of local adaptation of the mite to genotypes of the plant. The host range of this mite is quite narrow, and it does appear to damage its host plants (Goolsby et al., 2004b), thus the appropriate steps are being taken to evaluate the benefits and risks of releasing it into the United States.

Ideas about local adaptation have influenced the regulatory side of biological control in that permits for the release of new arthropod agents of invasive weeds are now commonly given for a single population on which host specificity tests have been performed, to avoid introducing biological control agents that may differ in their host use. For pathogens of both weed and insect pests, permits are given for single isolates. This approach differs dramatically from that once promoted by DeBach (1964), who suggested that individuals from many populations should be introduced. This change in perspective has come about largely through the recognition that separate populations may differ in their ecologies due to the evolution of local adaptation. In addition, bringing together individuals from separate locally adapted populations may lead to outbreeding depression (e.g., Kazmer et al., 1996; Keller et al., 2000; Wu et al., 2004) or have other unintended consequences (Hoffmann et al., 2002).

3.2. Does adaptation following introductions play a critical role in successful biological control?

It is thought that biological control agents may become adapted to the new environment and the particular genotypes of the target pest in the new range where they have been introduced. Indeed, it has been assumed that when populations of a newly introduced biological control agent remain small for some time and then outbreak, that adaptation has occurred (Lawton, 1990; Murray, 1985). However, such a pattern can be due simply to Allee effects and to exponential population growth from a small founding population, rather than to adaptation (Crooks and Soule, 1999; Hopper and Roush, 1993; Lawton, 1990; Murray, 1985; Roderick and Howarth, 1999).

Some of the strongest support for adaptation following introduction comes from biological control agents of insect pests in cropping systems. The use of pesticides likely imposes strong selection for natural enemies to evolve resistance to pesticides. Indeed, assays of field populations of several different biological control agents have revealed correlations between resistance and pesticide use, strongly supporting that resistance to pesticides evolved in the field (Baker and Weaver, 1993; Rathman et al., 1995; Rosenheim and Hoy, 1986; Xu et al., 2001). Artificial selection also has been used to increase resistance in natural enemies in efforts to integrate biological control with chemical control (Hoy and Knop, 1981; Rosenheim and Hoy, 1988; Roush and Hoy, 1981). Artificial selection also has been used successfully to alter biological control agents with respect to other characteristics, notably diapause induction (van Houten et al., 1995). Thus, with strong enough selection, adaptive evolution in biological control agents following introduction appears likely.

There is less evidence for biological control agents adapting to particular genotypes or species of the target pest(s) following introductions. Local adaptation following an introduction may have occurred in the ichneumonid parasitoid *Bathyleptes curculionis* (Thompson) (Salt and van den Bosch, 1967). This parasitoid has two main hosts, *Hypera brunneipennis* (Boheman) and *Hypera postica* (Gyllenhal) (Curculionidae). Parasitoids collected from an area with *H. brunneipennis* and from an area with *H. postica* were caged with each species. They were encapsulated less often in the host from which they were collected than the alternate host. Another case suggesting local adaptation following an introduction comes from *Cotesia glomerata* (Linnaeus) (Braconidae) (Lemasurier and Waage, 1993). This parasitoid was introduced from *Pieris brassicae* (Linnaeus, 1758) in Great Britain for the control of *Pieris rapae* (Linnaeus, 1758) in the US About 100 years following its release, rates of parasitism of a British and a American population of *C. glomerata* were evaluated on *P. rapae*, and

exhibited 16.8 and 31.3% parasitism, respectively. Both these studies strongly suggest that the biological control agents adapted to their local hosts following introductions. However, though ground-breaking, neither study was replicated at the population level, making the inference of local adaptation weaker. In one case where a reciprocal transplant was performed that was replicated at the population level, the introduced populations of the biological control agent were less fit than their native counterparts on the target hosts in the new range, suggesting maladaptive evolution associated with the introduction, rather than local adaptation (Hufbauer, 2002).

With only three studies that we know of where biological control agents were evaluated, we must look elsewhere for evidence of adaptation following introductions. There has been an explosion of research on the evolution of biological invaders, largely spurred by the “evolution of increased competitive ability” (EICA) hypothesis (Blossey and Nötzold, 1995). There is strong evidence that invasive plants can become locally adapted to their new environment fairly rapidly (e.g., Blair and Wolfe, 2004; Maron et al., 2004; Siemann and Rogers, 2001), suggesting that introduced biological control agents may adapt as well. We simply do not have enough experimental data to conclude whether or not adaptation in the new environment is an important part of efficacious biological control.

3.3. Do introductions of biological control agents impose bottlenecks in population size that reduce genetic variation, and is reduced genetic variation associated with low fitness and poor performance?

The effects of introductions on neutral variation has been the subject of research for several decades (Baker and Stebbins, 1965). Recent work has focused on invasive species (e.g., Tsutsui and Case, 2001; Zeisset and Beebee, 2003), but some have examined biological control agents. At least three studies (e.g., Baker et al., 2003; Hufbauer et al., 2004a; Lloyd et al., 2005) suggest that biological control agents do indeed pass through bottlenecks in population size that reduce variation in neutral loci as predicted on theoretical grounds (Hopper et al., 1993).

The consequences of reduced variation in neutral loci in classical biological control agents have not been studied directly. However, two studies on *Aphidius ervi* Haliday (Braconidae) show that populations with reduced neutral variation also have reduced fitness (Hufbauer, 2002; Hufbauer et al., 2004a). Here, this pattern is correlational and cause and effect cannot necessarily be inferred. The existence of a positive relationship between genetic diversity and fitness is supported by a meta-analysis performed by Reed and Frankham (2003). They examined 34 studies that included measures or proxies of genetic diversity and fitness for at least three separate populations and found a significant positive correlation

($r = 0.43$) that explained 19% of the variation in fitness. This suggests that genetic diversity can be a valid indicator of fitness, and that low genetic diversity is likely associated with inbreeding and inbreeding depression. In an experimental examination of the effects of breeding history on the ability to adapt to a novel stressful environment, inbred populations of *Drosophila* had lower fitness and were less adaptable than outbred populations (Reed et al., 2003). Concern about inbreeding is particularly great when population sizes remain small for long periods. However, if bottlenecks are of brief duration, even if they are severe, relatively little variation will be lost (Nei et al., 1975). With introductions of biological control agents that are successful in founding populations, bottlenecks may well be brief. Indeed, one goal to strive for to avoid potentially detrimental inbreeding effects is to minimize the period that populations remain small.

While Reed and Frankham's (2003) study suggests that reduced genetic variation is important for fitness, the invasion biology literature shows that this is not universal. For example, the green spruce aphid (*Elatobium abietinum* (Walker)), an introduced pest in New Zealand appears to be genetically monomorphic (Nicol et al., 1998). While a lack of genetic variation may retard the evolution of resistance to control methods, it has not prevented this aphid from reaching pest status. A similar lack of variation has been found in invasive populations of Japanese knotweed (*Fallopia japonica* (Houtt.) Ronse Decr.) (Hollingsworth et al., 1998), and low genetic variation may actually facilitate invasion of the Argentine ant (*Iridomyrmex humilis* (Mayr)) (Tsutsui et al., 2003). Certainly not all biological invaders lack variation, but some species are clearly able to attain high population sizes even with little genetic variation. At this point, there is no reason to suppose that biological control agents, a taxonomically diverse group, would be more or less prone to inbreeding depression.

To evaluate the consequences of genetic bottlenecks for biological control, experimental introductions could be performed where populations are established with agents ranging from inbred to outbred, crossed with release sizes from small to large. This would allow genetic variation to be disentangled from simple demographic factors. Although it is difficult to sacrifice extremely valuable biological control agents to experiments at the outset of a new introduction, it is at this stage that experimental manipulations would be most illuminating because discrete introductions could be made and tracked over time with respect to both population growth rate and other measures of fitness and variation in neutral loci.

3.4. How great is the risk of evolution of host range of biological control agents?

van Klinken and Edwards (2002) review and analyze the literature on the potential for evolutionary changes

in the host range of phytophagous insects used for biological control of weeds. As they point out, there is not a single well documented case of the evolution of host range following a biological control introduction, not even the infamous *Rhinocyllus conicus* (Frölich) (Curculionidae) (Arnett and Louda, 2002). Although this is quite heartening, adequate experimental tests required to document the evolution of host range to our knowledge have not been done. Like with studying other forms of local adaptation, reciprocal transplant experiments are one of the best experimental tests of the evolution of host range. Individuals from replicate populations of the nontarget host and the target host could be reared on both the nontarget and the target host. Evidence of local adaptation to the nontarget would suggest that host use had evolved. Data from such experiments could give us greater confidence in current host-specificity testing protocols.

A small set of experiments of this sort have been done, not with biological control agents, but with herbivorous insects with a fairly broad host range that include a new host in their diet. For example, *Euphydryas editha* (Boisduval, 1852) (Nymphalicae), a polyphagous butterfly has populations that have become locally adapted to plants introduced from Europe (Singer et al., 1992; Thomas et al., 1987). Similar experiments need to be performed with biological control agents, which typically have a much narrower host range, and therefore may behave differently.

Despite ongoing protests, widespread concern about the impact of classical biological control agents on native arthropods is a relatively recent development (Howarth, 1991). To our knowledge, no one has yet examined whether the host-range of biological control agents of arthropods has evolved following introductions, leaving a vast gap in our knowledge that only can be filled with experimental evaluations.

In addition to exploring whether host range has evolved following biological control introductions, taking a microevolutionary perspective to host-range testing could enable us to begin to estimate the risk of evolution prior to new introductions, as suggested by Secord and Kareiva (1996) and Schaffner (2001). By measuring genetic variation in traits that influence host use we can begin to estimate the short term risk of evolution of host range.

3.5. What is the risk of target pests evolving resistance to biological control agents? If pests evolve increased resistance, will biological control agents evolve mechanisms to overcome that resistance?

The evolution of resistance to pesticides is a common phenomenon in insects, weeds, and plant pathogens (Jasieniuk and Maxwell, 1994; Leroux and Gredt, 1997; Rosenheim et al., 1996). Pest population sizes are typi-

cally quite large, which increases the chance that a mutation influencing resistance will occur, and selection in the form of the application of pesticides is quite strong, driving mutations or standing genetic variation that enables survival and reproduction towards fixation quickly. Such dramatic evolution of resistance to natural enemies due to novel mutations has not occurred in biological control systems (Holt and Hochberg, 1997), probably because selection imposed by natural enemies is unlikely to be as strong as that imposed by the use of pesticides and may be both spatially and temporally heterogeneous. In addition, the genetics underlying resistance to a pesticide often may be simpler than that underlying resistance to a biological control agent, making it more likely that a single mutation of large effect can increase fitness when pest populations are suppressed with pesticides (Groeters and Tabashnik, 2000) than when they are suppressed through biological control.

However, evolution can proceed without new mutations if there is standing genetic variation. Quantitative genetic variation in resistance or tolerance to biological control agents is likely to exist in most pests. For example, cordgrass (*Spartina alterniflora* Loisel.) harbors genetic variation in both tolerance of and resistance to *Prokelisia* hoppers that have been introduced for the biological control of invasive western populations (Garcia-Rossi et al., 2003). The pea aphid (*Acyrtosiphon pisum* Harris, 1776) likely contains variation in resistance to parasitism by the parasitoid *A. ervi* both within (Henter and Via, 1995) and between (Hufbauer and Via, 1999) populations. The chestnut blight fungal pathogen (*Cryphonectria parasitica* (Murrill) Barr) shows variation in tolerance to strains of the hypovirus (Peever et al., 2000). Research from systems outside of biological control shows that standing genetic variation for resistance to natural enemies is common. This has been particularly well studied in plants (Fritz and Simms, 1992), but also has been found in insect–parasitoid systems (Kraaijeveld and Godfray, 1999), insect–pathogen systems and pathogen–enemy systems (Roderick and Navajas, 2003).

The other component necessary for resistance to biological control agents to evolve is selection. Holt and Hochberg (1997) argue that the spatial and temporally heterogeneous nature of selection imposed by biological control agents reduces the potential for the evolution of resistance. However, Thompson and co-workers (Nuismer et al., 1999; Nuismer and Kirkpatrick, 2003; Thompson, 1994; Thompson et al., 2002) argue that spatial and temporal heterogeneity leads to evolutionary “hotspots” and “coldspots.” In the hotspots, selection is strong, and rapid adaptive evolution is expected.

There are several examples from biological control that provide evidence of the evolution of resistance to biological control agents. One is from the classic work on the control of rabbits with myxoma virus in Australia noted above (Fenner, 1983). In addition to the

evolution of attenuated virulence among the virus strains, rabbits appear to have evolved greater resistance. This is shown through a clear relationship between rabbit susceptibility to a myxoma strain of intermediate virulence and the number of outbreaks of myxoma a rabbit population had experienced. Another classic example is the biological control of rush skeleton weed (*Chondrilla juncea* L.) in Australia with the rust *Puccinia chondrillina* Bubak & Syd. (Chaboudez, 1994). In Australia, three genotypes of this apomictic weed have been identified: one susceptible to the imported strain of the rust, and two resistant. After release of the rust, the spatial distribution and frequency of the genotypes shifted so that the susceptible genotype became rare, and the average level of resistance increased (Burdon et al., 1981). Recall here that evolution is simply change over time of the proportion of individual organisms that differ genetically in one or more traits and does not require mutation or recombination. Given this understanding of evolution, it can be seen that rush skeleton weed exhibited rapid evolutionary change in response to biological control. A third example suggesting the evolution of resistance to a biological control agent comes from an insect–parasitoid system, in which the host (the weevil *H. postica*) has exhibited an increase in encapsulation rate of the parasitoid *B. curculionis* through time (Berberet et al., 2003).

Thus, while evolution of resistance to biological control is not likely to be as rapid as evolution of resistance to pesticides, it can occur, and it can influence the efficacy of biological control. However, the biological control agents may evolve as well, which may make complete failure of control due to resistance a rare phenomenon. In situations like that seen in the control of rush skeleton weed, importing additional strains of the pathogen may shift the genotype frequency away from the forms that are resistant to the original strain, and lead to better control.

4. Conclusions

The above review underscores that relatively few data are available to address the questions we pose. Local adaptation of candidate or actual biological control agents within the native range to their hosts or to the environment rarely has been studied. Theoretical work on local adaptation suggests it will be common, though not ubiquitous. Reasonably strong evidence suggests that adaptation to climatic regimes supports the need for sensible climate matching. With respect to adaptation to host genotypes, recent theoretical developments linking rates of dispersal of host and parasite to local adaptation are relevant to biological control. However, the degree to which local adaptation to host genotypes influences successful biological control following an introduction is

largely unknown. It will enhance the success of biological control most if local adaptation to host genotypes is associated with increased rather than reduced virulence. Even with reduced virulence, if local adaptation to host genotypes increases population size, it may enhance control.

After agents are introduced, they may adapt to the new environment. Some biological control agents of insects in cropping systems have adapted to pesticide regimes, but the role of ongoing adaptation to host genotypes or other aspects of the environment is less obvious. Introductions of biological control agents can impose bottlenecks in population size that are revealed in reduced variation in molecular loci of biological control agent populations. However, the link between molecular variation and fitness is somewhat tenuous, and has not been scrutinized in biological control agents. Of vital importance to long-term safety of biological control is the risk of the evolution of host range. This is one of the issues that strikes the most fear in biological control practitioners and members of regulatory agencies, yet is one with the least relevant data. Both retrospective studies of past introductions, and prospective evaluations of genetic variation for host use are needed urgently. For biological control to be an evolutionary stable means of managing pests, there needs to be a balance between the evolution of resistance of target pests to their biological control agents, and evolution in biological control agents to overcome resistance. While we know both pests and agents can evolve with respect to their interaction, the importance of ongoing coevolutionary dynamics are unknown.

If we are to understand better the role of microevolution in biological control, the simple fact is that we need more data. With more data it may be possible to manipulate biological control systems to facilitate or regulate adaptive evolution in biological control agents that leads to more effective control, while minimizing maladaptive changes through drift, evolution of host range in biological control agents that reduces the safety of biological control, and evolution of resistance in target pests. In risk assessment, it is what we do not know that hurts us. Only with a greater understanding of the role of microevolution in biological control, can the field move efficiently towards minimizing the risks and maximizing the benefits.

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