# REVIEW

# Is host-specificity of weed biological control agents likely to evolve rapidly following establishment?

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## Abstract

Over 352 herbivore species have now been intentionally introduced into new regions as weed biological control agents. Recent evidence shows that rapid and significant evolution in host-specificity can occur. The risk of non-target use by biological control agents increasing to unacceptable levels through rapid evolution therefore needs to be considered. In addition, weed biological control offers many as yet largely unexploited opportunities for improving our basic understanding of host-specificity and its evolution. We therefore evaluate the evidence that rapid evolution (1) alters the use of existing hosts, and (2) alters the fundamental host-range. Most cited examples of so-called host shifts from weed biological control were not the result of genetic change. There was only limited evidence of genetically altered performance on a non-target host and no evidence of altered fundamental host-range. We conclude, from both theory and the available data, that only altered use of existing hosts (through quantitative genetic changes) needs be considered when evaluating the risk of rapid evolution. Host-specificity testing methodologies can be improved and adapted to better assess the risk of occurrence of post-release evolution.
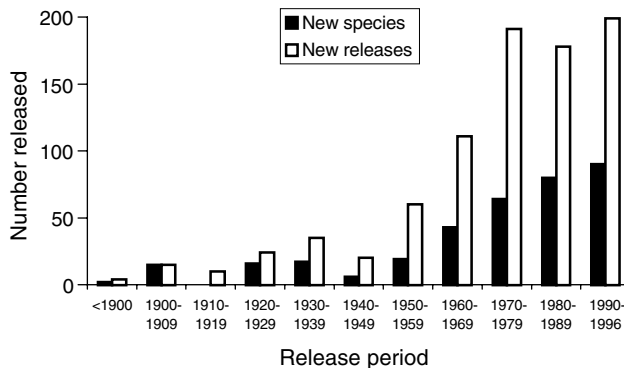
## INTRODUCTION

Although herbivore–host relationships traditionally were thought to have evolved slowly, recent evidence demonstrates that significant changes in host use can occur in the field within 10's or 100's of generations (Thompson 1998). Examples of so-called rapid evolution remain rare and it is not yet known whether it occurs only under exceptional circumstances, or whether host use by herbivores is more fluid than previously thought. The possibility of rapid evolution nonetheless raises important issues for the practice of classical weed biological control, where herbivores (both insects and pathogens) are released into totally new environments free of specialist natural enemies and often exposed to potential new hosts and different selection pressures.

The long-held and most widely accepted view among biological control practitioners is that there is essentially no risk of post-release evolution resulting in increased non-target attack because evolution will favour increased specialization (Huffaker 1959; Harris & Zwolfer 1968; McFadyen 1998), particularly if "host-range is integrated with many features of biology" (Zwolfer & Harris 1971). This position has, however, been challenged recently by those questioning the "green credentials" of biological control (Howarth 1991; McEvoy 1996; Secord & Kareiva 1996; Simberloff & Stiling 1996a, b), and does not necessarily match the modern understanding of herbivore–host interactions (Courtney & Kibota 1989; Brooks & McLennan 1993; Futuyma 2000). Potential consequences of improperly evaluating the risk of evolution of host-specificity are two-fold: (1) potentially damaging agents might be released, and (2) potential agents might be rejected unnecessarily because of unfounded fears of post-release evolution.

In many ways biological control of weeds offers an ideal opportunity for assessing the risk of rapid evolution in host-specificity following the colonization of new environments. Over 352 exotic organisms have been intentionally released since 1832, many of them into more than one country (Fig. 1). Most introductions are of relatively specialized herbivores and are typically well documented, with details including the origin of source populations, the timing and size of release populations, and predictions of non-target plant species that might be at risk (Julien & Griffiths 1999; references therein). Host-specificity is described, at least in general terms, for most agents, with detailed studies typically including the target weed as well as both closely and distantly related plants.

**Figure 1** Number of weed biological control agents released through time. New releases are classified as introductions into new countries. Data from Julien & Griffiths (1999).

Here we review the evidence for rapid evolution in the host-specificity of herbivores, looking particularly at evidence from weed biological control, and discuss implications for the practice of weed biological control. We approach the problem from the perspective of evolutionary constraints inferred by host-specificity itself and discuss whether the type and degree of host-specificity predisposes a herbivore to evolutionary change. This approach has the added advantage that host-specificity is relatively easily described and quantified. Rapid evolution also requires genetic variation and an ecological context in which directional selection can occur. These requirements are discussed elsewhere (e.g. Courtney & Kibota 1989; Via 1990; Roderick 1992; Carroll & Dingle 1996; Holt & Hochberg 1997; Ronce & Kirkpatrick 2001).

## TYPES OF HOST-SPECIFICITY

Host-specificity is an intrinsic property of an insect, and is the consequence of behaviour, post-ingestive constraints, nutritional needs and morphology (Zwolfer & Harris 1971; Scriber 1984; Slansky & Rodriguez 1987; Courtney & Kibota 1989; van Klinken 2000a). As such, it is a function of genotype and can therefore vary considerably between individuals within a species (Wiklund 1981; Singer *et al.* 1993) and between populations (Funk 1998), all else being equal.

Host-specificity is a combination of host-range breadth, the level of preference for each host for feeding or oviposition, and the rate of growth and reproductive performance on each host (van Klinken 2000a). Thus two herbivores with identical host-ranges could differ dramatically in their host-specificity because one species does not distinguish between hosts whereas the other species utilizes one in strong preference to the remainder. Likewise, evolution in host-specificity can result in a change in the pattern of use of existing hosts, or a change in host-range through the addition, replacement or loss of hosts.

The expression of host-specificity will often depend on the environment (van Klinken 2000a). For example, many herbivores are capable of finding and utilizing more hosts than they do in the field, most often because of host-availability. It is therefore valuable to distinguish between the fundamental host-range of a herbivore, the limits of which are determined genetically, and the realized host-range, which is how the fundamental host-range is expressed within a particular environment (Nechols *et al.* 1992; van Klinken 2000a; Schaffner 2001). Fundamental host-range can be determined in no-choice tests under optimal physical conditions, using healthy (and usually naïve) insects, and run for the duration of the insects' life to exclude time-dependent effects (van Klinken 2000a).

Fundamental host-range therefore includes all those plant taxa that can be used as a host by a particular genotype, whereas the realized host-range is all those actually used in a particular environment. Similarly, the relative use of potential hosts will depend both on intrinsic properties (such as preference for, or performance on each host, and how the insect responds to experience) and on the environment (in particular the relative availability of hosts) (Courtney & Kibota 1989).

Host-specificity can be considered as a subset of niche, although there are important conceptual distinctions. Host-specificity can be described precisely, whereas niche is typically a multidimensional construct describing the relationship between populations (or species) and the environment (and populations from other species) (Hengeveld 1988). In addition, host-specificity is defined as a property of genotypes rather than populations. For herbivore populations (or species), the fundamental and realized host-range becomes the sum of host-ranges for the constituent genotypes. The fundamental host-range includes all hosts upon which the combined fitness of the population is greater than "0", even though some of these hosts do not necessarily support viable populations.

Evolution of host-specificity has received attention from others (Zwolfer & Harris 1971; Weidermann 1991; Marohasy 1996; Secord & Kareiva 1996; Futuyma 2000). However, confusion remains regarding the different ways in which host-specificity can change, particularly between genetically and ecologically induced changes in host use, and between genetic change resulting in the altered use of existing hosts and genetic change resulting in the addition, replacement or loss of hosts. We consider evidence for each of these, and discuss how evolution can be better predicted through host-specificity testing.

## EVIDENCE OF EVOLUTION OF HOST-SPECIFICITY

The weed biological control literature contains many reports of genetic change in the host-specificity of released agents

(Secord & Kareiva 1996), but the evidence for genetic change is in most cases weak. Terminology which has specific evolutionary meaning, such as "host-shift" and "host switch", is used frequently within the weed biological control literature to describe an observed change in the pattern of host use in the field (see Marohasy 1996). For example Dennill *et al.* (1993) use the term "host-shift" to describe the colonization of two non-target species by an introduced gall wasp in South Africa. However, genetic change was never implied, with one host being explained as a "new association" and the other explained as the consequence of intraspecific competition on the original host. In addition the same authors state that "20 host shifts of introduced weed biocontrol agents have been noted to date", but again never imply genetic change as a mechanism. Nonetheless these examples have subsequently, and perhaps incorrectly, been used in the literature as purported examples of rapid evolution among weed biological control agents (Marohasy 1996; Secord & Kareiva 1996; Simberloff & Stiling 1996a).

There are, however, also examples in the literature for host-specificity changes that are likely to have a genetic basis. The subtlest way in which host-specificity can change genetically is through a *change in the pattern of use of hosts* that are already included within the fundamental host-range. Adaptations in host use can potentially be expressed in one or more ways, such as altered acceptability for particular behaviours within the host location and acceptance sequence, and altered performance for pre-reproductive and reproductive development (Courtney & Kibota 1989; Marohasy 1998; van Klinken 2000b). Behavioural responses to hosts can also vary with deprivation and prior experience, and the nature and degree of such behavioural plasticity can therefore also change genetically.

More drastic changes in host-specificity can occur because of *alterations to the fundamental host-range*. The fundamental host-range can change either through the addition of new hosts (for example through the loosening of constraints), loss of existing hosts (for example through extreme shifts in preferences) or, much less often, through the replacement of existing hosts. Host replacement in a herbivore is more likely to occur if high host fidelity leads to reproductive isolation of the populations on each host (Funk 1998).

Genetic constraints to host-specificity changes will differ between species depending on the number of traits that result in differential preference for, and/or performance on, non-targets, and whether or not they are under independent genetic control. When traits controlling host-range are governed by a number of genes (quantitative traits), altered selection pressures in the environment may be sufficient to alter host-specificity. When one or only a few genes are controlling host-range, novel mutations are probably necessary for the fundamental host-range to change.

Examples from the literature for genetic changes in host use will now be characterized as to whether they provide evidence for (1) changes in quantitative traits in response to altered environmental selection pressures, or (2) changes in traits arising from mutation.

## Changes in quantitative host use traits

There are several examples of adaptations in the pattern of host use over a relatively short time (Thompson 1998). One of the most dramatic examples is of an endangered checkerspot butterfly in the United States (Singer *et al.* 1993). A European weed was introduced into some of the sites where this butterfly occurs. Oviposition preference of individual females was measured over an 8-year period to compare relative preferences for the native, ancestral host and the introduced host. Over that period the proportion of females preferring the new host increased steadily from under 10% to over 50%. Other examples include the soapberry bug (altered beak-length and juvenile growth parameters, Carroll *et al.* 1997), *Papilio* butterflies (altered degree of oviposition preference, Thompson 1993), and the apple maggot fly (altered oviposition behaviour, Prokopy *et al.* 1988). In fact, changes like these in the host-specificity of herbivores are likely to be quite common (Thompson 1998).

In cases like these, novel mutations are probably not necessary to allow these changes in host use patterns. Rather, they are more likely the result of selection acting on characters (e.g. oviposition preference, larval performance) that are controlled by a number of genes. For example, ecological host specialization in the oligophagous pea aphid has resulted from selection pressure acting on two quantitative traits, fecundity and host acceptance, assisted by positive genetic correlations (Hawthorne & Via 2001).

In weed biological control, the concern is that rapid evolution in host use may result in improved performance on non-target host species. No systematic records have been kept of predicted or actual incidences of non-target attack. However, in Australia at least 14.4% of agents released up until 1991 ($n = 125$) had the potential to attack non-targets upon release (A. Willis and T.A. Heard, unpublished). Release requirements in Australia have historically been relatively strict, so this is likely to be a conservative figure for releases elsewhere in the world. More than 51 biological control agents (many of which have been released in more than one geographical region) therefore probably utilize non-target species in the field.

There is, however, only one example where performance by a weed biological control agent on an existing non-target host has been thought to have improved genetically post-release. The chrysomelid beetle *Chrysolina quadrigemina* Forster was released in California to control *Hypericum*

*perforatum* L. in 1946. It was first observed attacking the closely related non-target species *H. calycinum* L. in 1975 (Andres 1985). Although already within the fundamental host-range, laboratory tests suggest larval survival on *H. calycinum* by field populations collected off that host was greater than in populations collected off the target weed ($n = 30$ newly emerged larvae, data not shown; Andres 1985), so it appears that some ecological specialization has occurred (Andres 1985; Ehler 1991; Secord & Kareiva 1996). However, observations of field attack coincided with the increased use of *H. calycinum* in roadside plantings (Andres 1985) and may therefore merely reflect a change in relative host availability (see also Marohasy 1996). Furthermore, performance tests need to be repeated more rigorously to confirm that genetic differentiation between populations has in fact occurred.

The likelihood that host use patterns will change depends on (1) the extent of novel selection pressures, and (2) the level of genetic variation upon which the selection will act. It is difficult to predict a priori the selection pressures for altered host use in a novel environment. Weed biological control examples will usually involve a source population with high average fitness (target) and a sink population with very low average fitness (non-target). In situations like this, there will rarely be selection pressure for improved performance on the non-target (Holt & Gaines 1992; Holt 1996). Whereas in most natural populations genetic variation in preferences and performance is not likely to be limiting (Wiklund 1981; Via 1990; but see Futuyma 2000), in cases of weed biocontrol, host use changes may be limited by the low level of genetic variation in the introduced population.

Quantitative genetic changes in host use traits can potentially result in a change in fundamental host-range, but the likelihood is even more remote. Host loss, although of no concern to the practice of biological control, is the most likely because it can result from strong selection on the alternative host as appears to be happening for checkerspot butterflies (Singer *et al.* 1993). Quantitative changes resulting in the addition or replacement of a fundamental host would require adaptation for improved performance on a "sink" host with an initial fitness of "0". This is highly unlikely (Holt & Gaines 1992; Ronce & Kirkpatrick 2001), and has never been recorded through rapid evolution.

### Changes in host use arising from novel mutation

Mutations can change both host use and fundamental host-range, although there are no documented examples of mutation resulting in the rapid evolution of host-specificity. Changes in host use are generally most easily explained through quantitative genetic changes. The likelihood of mutations changing the fundamental host-range will depend, in part, on the number of traits constraining it.

Fundamental host-ranges can be constrained by a single behaviour or trait (Futuyma 2000). For example, the response of one monophagous flea beetle species to host cues is coded by only a single gene, suggesting that a single mutation could cause a host addition or replacement in this species (Mikheev & Kreslavskii 1986). Similarly a single dominant gene determines the level of sensitivity of different *Yponomeuta* species to chalceneglycoside feeding deterrents (van Loon 1996).

Alternatively, host-range can be limited by numerous traits all governed by different genes, such as various steps within the oviposition sequence, larval development and adult feeding. In these cases, all of these aspects would have to change if the fundamental host-range is to change. For example, the apparent host replacement of the brown planthopper (*Nilaparvata lugens*) in the last 250 000 years from the semiaquatic weed *Leersia hexandra* to the contribal species *Oryza sativa* (rice) has involved changes in larval development, oviposition and adult feeding (Sezer & Butlin 1998a, b). There is no evidence for a genetic correlation between the traits, so it appears that the shift has occurred as a result of coincidental mutations (Sezer & Butlin 1998b). Dominance occurs in all traits towards the novel host, which in the absence of genetic correlations was probably essential for this host replacement to have proceeded (Sezer & Butlin 1998b).

Considering these examples, it should not be surprising that there is some phylogenetic evidence for host replacements in monophagous herbivore species (Menken 1996; Wahlberg 2001). These examples are not, however, the norm; the restricted host associations in the phylogenies of other taxa indicate that the majority of herbivore–host relationships have been relatively immutable (e.g. Hodkinson 1988; Futuyma 2000). There also may be constraints to host replacements: There is evidence that host shifts will more often occur to plants that have been hosts in the species' evolutionary history (Janz *et al.* 2001).

### PREDICTING THE RISK OF INCREASED ATTACKS ON NON-TARGET HOSTS THROUGH EVOLUTION

Changes in fundamental host-range should not be a concern for biological control practitioners. The risk that changes in quantitative host use traits will result in the addition of a previously unusable host are minimal. The risk of host additions or replacements that require specific individual or multiple mutations are even less likely, and are no more likely to occur for an introduced species in the future than in the past or future for a native species. It could be argued that they are less likely to occur because an introduced species is less likely to be exposed to plants that were hosts in the species' evolutionary history (Janz *et al.* 2001). Temporary ecological phenomena associated with biocontrol introductions, such as

high population densities or low host availability, may increase the likelihood that mutant individuals will come in contact with the novel host. However, this increased chance is inconsequential when compared to the minuscule per annum likelihood ($< 10^{-10}$) that a particular amino acid mutation will occur (Hartl 1987). There is only one scenario in which fundamental host-range may change by mutation at a relatively high rate: when the only barrier to host use is a single deterrent chemical. In this scenario any of several mutations could result in the addition of the host. To date there are no examples, but the scenario is feasible and warrants consideration.

Since the fundamental host-range is unlikely to change after release, the first challenge to biological control practitioners should be to describe as completely as possible the fundamental host-range of the introduced population. This is generally accomplished using host-specificity testing (van Klinken 2000a; Schaffner 2001). The fundamental host-range of the test population is often easily determined experimentally by excluding environmental factors that might limit its expression, such as prior experience and time-dependent effects, through the use of naive insects in no-choice long-duration tests (Papaj & Rausher 1983; Szentesi & Jermy 1990; van Klinken & Heard 2000; van Klinken 2000a, b). In many cases it is possible to describe fundamental host-range for particular aspects of the life history (such as oviposition or even specific steps within the oviposition sequence) and thereby describe possible constraints to host change more precisely (van Klinken 2000a). The host-specificity testing conducted by weed biological control practitioners appears to be very effective, because there is no evidence in the literature of an agent attacking a plant outside the defined fundamental host-range.

Changes in the pattern of host use can be greatly impacted by demographic changes such as those experienced by biological control agents (Holt & Gaines 1992; Kawecki 1995; Holt 1996; Ronce & Kirkpatrick 2001). The greatest risk to non-targets in weed biological control is from changes in the pattern of use of hosts identified through host-specificity testing as being within the fundamental host-range of the agent. Thus, the second challenge for biological control practitioners is to predict whether improved host use of non-target species is likely to evolve, and whether those changes are likely to result in non-target impact reaching unacceptable levels. There is no question that the use of any non-target species included within the realized host-range has the potential to improve in the new environment because of new selection pressures. In these cases, the onus is (and should be) placed on the biological control practitioner to put forward cogent arguments why this is not likely to happen.

The exact physiological and behavioural factors that result in differential performance on target and non-target hosts can usually be identified and quantified through host-specificity testing. Generally the greater the differences in host use, the more genetic change is required to improve use of non-targets, and the less likely that rapid evolution will occur. However, a full assessment of risk will require consideration of the genetic variation present (e.g. Karowe 1990), likely gene flow (including the effect of dispersal ability and inbreeding) and likely selection pressures within the ecological context into which releases are made (e.g. the relative distribution and abundance of the target and non-target species).

In some cases, selection experiments may assist in demonstrating that such host-specificity changes are possible (Hill & Caballero 1990; Sheck & Gould 1996). The capacity for change can also be estimated by examining how the pattern of use of existing hosts differs between conspecific populations of the herbivore from areas differing in the expected selection regime imposed by the host plants (Fox & Morrow 1981). The influence of ecological processes on herbivore–host evolution is now better understood (Holt & Gaines 1992; Kawecki 1995; Ronce & Kirkpatrick 2001), which should improve the predictability of host-specificity adaptation after release.

It is also possible to learn from previous experiences. For example, adaptive change in *C. quadrigemina* populations should be confirmed, and the adaptive mechanism that resulted in the specialization of some populations on *H. calycinum* should be investigated. Standard genetic analyses of inheritance patterns in hybrid populations would provide valuable insight, and the importance of novel mutations could be determined by establishing whether the adaptation is reversible when the populations are returned to the original host. More potential case studies are likely to emerge as other weed biological control agents are scrutinized, especially species for which at least limited non-target attack was predicted.

## CONCLUSIONS

All examples of rapid evolution of host-range by a herbivore can be classified as changes in the pattern of host use rather than a change in the fundamental host-range, with the exception of one example of host loss. Furthermore, both theory and available data suggest that the risk that a herbivore will rapidly evolve to expand its fundamental host-range is extremely low. To some degree, the number and nature of constraints to fundamental host-range can be determined through host-specificity testing (e.g. van Klinken 2000b).

There is also only one, unconfirmed, example among weed biological control agents where performance on an existing non-target host has improved. There are, however, several well-documented examples in the ecological

literature of rapid evolution in the use of existing hosts, and theory suggests it might even be relatively common. Once again, host-specificity testing can be used to help evaluate whether the herbivore is likely to be predisposed to evolved host use. The more physiological and behavioural aspects of the agent that are inferior on the non-target, the less likely rapid improvement in host use will occur.

We conclude that the fundamental host-range of biological control agents is, in theory, describable through host-specificity testing, but that altered performance on non-targets is not as predictable. Risk can be assessed for the latter by considering constraints, genetic variation and ecological context, but greater efforts are necessary to do so confidently. More importantly for weed biological control practitioners, recent research is making it easier to predict whether potential evolutionary change will be sufficient to cause increases in non-target attack that results in unacceptable impact. More generally, available evidence from weed biological control suggests that rapid, directional evolution of host-specificity is not a widespread phenomenon among narrowly host-specific herbivores. The risk of rapid evolution of biological control agents resulting in unacceptable non-target impact therefore appears small.

## ACKNOWLEDGEMENTS

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