Chapter thirteen

Invasions and the regulation of plant populations by pathogens

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INTRODUCTION

The potential of pathogens to have dramatic impacts on plant populations is made clear by familiar stories of the Irish potato famine (Fry and Goodwin 1997), the ecological extinction of chestnuts caused by chestnut blight (Anagnostakis 1987), and the transformation of Australian Jarrah forests to scrubland by *Phytophthora cinnamomi* (Weste and Marks 1987). Similarly, the annual worldwide expenditure of over $6.6 billion in fungicide application (Donaldson *et al.* 2002) reflects the toll fungal pathogens alone can take on plant growth and fecundity in agricultural systems. Some of the most notable examples of these impacts arise when pathogens are introduced into novel biological environments; invasive and emergent pathogens continue to frustrate the best efforts of resource managers, conservation biologists, and plant protection agencies (Weste and Marks 1987, Daughtrey *et al.* 1996, Goodell *et al.* 2000, Gordon *et al.* 2001, McDonald and Hoff 2001, Wingfield *et al.* 2001, Gilbert 2002, Rizzo and Garbelotto 2003, Parker and Gilbert 2004). Concurrent with efforts to reduce the impacts of unwanted diseases, there is broad interest among researchers, agriculturalists, and land managers to harness the destructive potential of plant pathogens to control weedy plants (Hasan and Ayres 1990, Charudattan and Dinoor 2000).

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In natural ecosystems, pathogens have great potential to influence the dynamics and composition of plant populations and communities through density-dependent and coevolutionary dynamics (see reviews in Dinoor and Eshed 1984, Burdon 1987, Jarosz and Davelos 1995, Alexander *et al.* 1996, Alexander and Holt 1998, Gilbert 2002, 2004). In many plant communities, plant pathogens may prevent competitive exclusion and thus help maintain species diversity (Gillett 1962, Packer and Clay 2000, Gilbert 2002, Wright 2002, Gilbert 2005). Collective insights from research on epidemic diseases, biological control, and the evolutionary ecology of diseases in natural ecosystems provide a robust basis for identifying when pathogens are likely to be important in regulating plant populations, and their implications for understanding biological invasions.

Here we draw broadly from a diverse literature to place the regulation of plant populations by pathogens into the context of two key, complementary theories about the role of pathogens in biological invasions: Biotic Resistance (Maron and Vila 2001) and Escape from Natural Enemies (Keane and Crawley 2002) (Table 1). We then consider the practical implications for using introduced pathogens for classical biological control of introduced invasive plants (Table 1), illustrated with a detailed case study of the control of *Chondrilla* by rust fungi.

PLANT DISEASES AND POPULATION REGULATION

Pathogens reduce the fitness of individual plants by killing them, reducing growth, impeding competitive ability, or by rotting fruits or seeds (see recent review in Gilbert 2002). The greater probability of pathogen spread between closely spaced host plants means that most fungal plant diseases show density-dependent development. In addition, densely spaced plants may create microclimates that encourage pathogen growth, and hosts stressed by competition may be more susceptible to disease (Burdon and Chilvers 1982, Gilbert 2002). The combination of strong impacts on individual host plants and density-dependent disease development suggests that pathogens should be powerful regulators of plant populations. Nevertheless, aside from epidemics caused by introduced pathogens, there are remarkably few empirical studies showing that plant diseases are responsible for regulating plant population dynamics in natural ecosystems (Gilbert 2002). In large part this absence reflects the difficulties of isolating disease impacts from other factors and the strong focus on diseases with economic importance. But physiological or evolutionary responses by the host may also counteract the regulatory actions of pathogens in natural ecosystems. In particular, plants that survive after disease has killed or stunted competing conspecific neighbors may show a compensatory response that offsets numerical losses from disease at the population level (Friess and Maillet 1996, Alexander and Holt 1998, Alexander and Mihail 2000). In addition, maternally-transmitted induced resistance can generate cross-generational effects that ameliorate the numerical impacts of disease in succeeding generations (Agrawal *et al.* 1999). Finally, the genetics

Table 1 Implications of different features of plant-pathogen interactions for three areas of invasion biology: the two theories of biotic resistance and escape from natural enemies, and the field of classical biological control.

Features of plant-pathogen interactions	Biotic resistance	Escape from natural enemies	Biological control
Impact of pathogen on host	Pre-adapted pathogens in new range are highly virulent on naïve hosts.	Pathogens in native range limit host density or distribution. Introduced plants leave virulent pathogens behind.	To be effective control agents, pathogens must be important in population regulation in native range.
Interactions with environment (disease triangle)	Local pathogens are adapted to local environment.	Plants may escape natural enemies if introduced into an environment not conducive to disease development.	Introduced biocontrol pathogens may fail if poorly adapted to local environment.
Host range of pathogen	Generalist pathogens are more likely to acquire newly introduced host species.	Assumes specialist pathogens play a unique role in regulating plant populations.	Only specialist pathogens can be utilized as control agents.
Natural history/ life history of pathogen	Density-dependent disease development may change impact on invading hosts as invasion proceeds.	Pathogens with resting structures or alternate life history strategies most likely to accompany introduced plants; such pathogens may have greater virulence.	Pathogens with resting structures should show greater success.
Rapid evolutionary changes	Pathogens may acquire introduced host through evolutionary host shift. Virulence may increase quickly when host becomes common.	Host may lose defenses to original pathogens after introduction.	Host may develop resistance to introduced biocontrol agent.

of plant-pathogen interactions can be highly dynamic, with large changes in pathogen virulence or host resistance evolving in a few generations (e.g., Burdon and Thompson 1995, Bishop *et al.* 2000). Such rapid evolutionary changes are expected to strongly influence the impact of pathogens on host numerical dynamics (Alexander *et al.* 1996).

BIOTIC RESISTANCE AND ESCAPE FROM NATURAL ENEMIES

For disease to develop, virulent pathogens, susceptible host plants, and suitable environmental conditions must converge (commonly called the Disease Triangle). Changes in any of these components can reduce or increase how much diseases affect plant population dynamics. If plants are introduced to an environment where virulent pathogens are not present or where environmental conditions do not favor disease development, the plant population may be released from previous regulation by pathogens. Similarly, plants introduced to a new locale with novel, virulent pathogens or where environmental conditions favor disease development may experience unprecedented population regulation by pathogens. Such changes in the prevalence and severity of diseases under different conditions have long been thought to play an important role in the process of biological invasions in two hypothetical ways:

1. In natural habitats, native pathogens colonize and are highly virulent on naïve, introduced plant species and prevent population growth (*Biotic Resistance*).
2. Pathogens were important in regulating the plant population in its native range, but are not in the introduced range (*Escape from Natural Enemies*).

Biotic resistance

The great majority of plant introductions do not result in invasions (Mack 1995, Williamson 1996). Rather, many introduced species either fail to thrive altogether or are restricted to human cultivation, unable to build self-sustaining populations in wild plant communities or even in disturbed rangeland communities. Research attempting to identify plant traits that confer invasiveness (Reichard and Hamilton 1996, Rejmánek and Richardson 1996) shows that our predictive ability is imperfect, and it seems to be more difficult to predict which introductions will fail than which will succeed (Reichard and Hamilton 1996). Biotic resistance is one possible explanation for why some introductions fail when they "should" succeed; native pests and pathogens colonize exotic plants and eliminate them before they can establish a viable population (Elton 1958, Simberloff 1986, Mack 1996).

Pathogens will contribute to biotic resistance only when three conditions are met. First, the pathogens involved must not be narrow host specialists. In plant communities with high host diversity (and corresponding low host density) generalist plant pathogens should dominate (Gilbert *et al.* 2002, Gilbert 2005),

which could contribute to the high invasion resistance of hyper-diverse ecosystems like tropical rain forests (Fine 2002). Second, because densities of the introduced host will usually be low immediately following introduction, pathogens involved in biotic resistance would not be those for which transmission or host switching was strongly density-dependent. Third, the pathogen must exact a high fitness cost on the host. Combinations of generalization and high virulence are not unusual in pathogens, especially for species that have long resting stages or saprophytic ability (e.g., *Phytophthora cinnamomi, Verticillium dahliae, Fusarium oxysporum*). Even pathogens that can attack many hosts have differential impacts on different host species. If a pathogen has a greater negative effect on the competitive ability of the non-native hosts than on the native hosts, then it will contribute to the competitive exclusion of the non-native (Keane and Crawley 2002).

Since even fairly specialized pathogens often infect many members of the same genus or family, it is likely that the number of pathogens competent to infect a novel host depends on whether it is phylogenetically related to native species already in the community. Therefore, biotic resistance should favor invasion by species with no close relatives. Despite this clear prediction, and the accepted practice of using phylogenetic relationships in quarantine and trade policy to target relatives of weedy plants or identify potential carrier hosts, surprisingly few studies of invasion even mention the phylogenetic structure of the invaded communities. In one rare attempt, Mack (1996) found that for 5 out of 6 regional floras, naturalized species were more common in genera with no native congeners than those with native congeners, consistent with the idea of biotic resistance. However, this study was not able to control for the effect of opportunity (whether plants without native congeners have a higher probability of successful invasion given their probability of introduction). Using a different approach, Duncan and Williams (2002) compiled a list of all plant species that have ever been introduced for cultivation in New Zealand. They found that introduced species in genera that already had resident natives were more likely, not less likely, to successfully naturalize. They suggest that species with native congeners may share characteristics that make them more fit in the introduced range, and this factor overwhelms the potential effect of local natural enemies.

There is a striking need for more studies to determine whether and when biotic resistance by native pathogens occurs. Because there is little information on where and when unintentional species introductions fail, assigning mechanisms to these failures has been nearly impossible. Horticulture, forestry, and agriculture each provide examples of endemic pathogens that have decimated introduced plant species so that growing these species is no longer economically viable (Mack 1996, Coutinho *et al.* 1998, Wingfield *et al.* 2001). However, extrapolating from agricultural or silvicultural examples to invasive introduced species requires caution, since the regeneration of hosts is controlled, preventing the host population from evolving resistance or tolerance to the pathogen. Additionally, simplified systems in agriculture and forestry may simply lack moderating effects

of greater biotic complexity in less managed systems (e.g., hyperparasites of the pathogens).

Escape from natural enemies

Introduced plants that become invasive weeds are among our most challenging environmental problems (D'Antonio and Vitousek 1992, Office of Technology Assessment 1993, Vitousek *et al.* 1996, Parker *et al.* 1999, Mack *et al.* 2000), and escape from natural enemies provides a mechanism to explain the increased growth and density of introduced species (Darwin 1859, Crawley 1987, Blossey and Nötzold 1995, Tilman 1999, Maron and Vila 2001, Siemann and Rogers 2001, Keane and Crawley 2002). The "Natural Enemies Hypothesis" posits that introduced species leave behind their natural enemies — herbivores, seed predators, and pathogens — and are thereby released from a key regulating factor, leading to a dramatic increase in plant vigor, population growth, and/or competitive ability. This idea forms the justification for classical biological control, in which natural enemies are brought from the native range to control weedy invaders (Huffaker and Messenger 1976, DeBach and Rosen 1991). Yet despite the importance of the Natural Enemies Hypothesis, rigorous empirical tests are few (Maron and Vila 2001).

A number of studies have tested whether introduced plants tend to grow faster or larger and whether there is evidence of reduced allocation to herbivore defenses in the new range (Crawley 1987, Blossey and Nötzold 1995, Siemann and Rogers 2001) but see (Willis *et al.* 2000, Thebaud and Simberloff 2001, Vila *et al.* 2003). While several of these studies have suggested ecological release and evolution away from defense toward competitive ability, they lack direct evidence of involvement by natural enemies (Blossey and Nötzold 1995, Siemann and Rogers 2001). Other studies have tested the Natural Enemies Hypothesis by comparing the impact of natural enemies on exotic and native species within the introduced range. In a review of 13 studies, Keane and Crawley (2002) found some cases in which generalist herbivores showed higher impacts on native species than non-natives, and others characterized by the reverse pattern. There have been surprisingly few studies with pathogens; in one test Goergen and Daehler (2001) found that smut fungi caused greater reproductive loss in a native grass (*Heteropogon contortus*) than an introduced grass (*Pennisetum setaceum*).

Enemy removal experiments are an important but underutilized tool in comparing the fitness effect of pathogens or herbivores on exotic and native species within the introduced range (Keane and Crawley 2002). Blaney and Kotanen (2001) used a fungicide experiment to remove the effects of soilborne fungi and oomycetes on the survival of seeds of native vs. introduced plants in two habitats. They found no support for a release from fungal pathogens in the seed bank of exotic species compared with native species. More recently, Parker and Gilbert (unpublished data) found no difference in frequency of infection, leaf damage, fitness effects of foliar and damping-off pathogens, or pathogen diversity between

sympatric suites of native and non-native clovers. Although fungal exclusion experiments in this system revealed significant impacts of pathogens, there was no difference in the response of native and non-native species.

Finally, other studies have taken the approach of comparing disease on a single host species in its native and invaded ranges. Wolfe (2002) surveyed for the anther smut fungus *Microbotryum violaceum* in 50 native populations and 36 introduced populations of *Silene latifolium*. He found significantly more populations infected, and at much higher infection frequencies, in the native range than in the introduced range. Mitchell and Power (2003) used published records of pathogen associations with 473 plant hosts in their native and introduced ranges. Plants were infected by 77% fewer pathogen species in their naturalized range. There was also an indication that species leaving behind proportionally more natural enemies were more likely to be categorized as noxious or invasive species by land managers and public agencies. Using an experimental approach, Beckstead and Parker (2003) directly measured the demographic effect of pathogens on an invader in the context of known information from the species' native range. *Ammophila arenaria* in its native Europe is limited to an early-successional role in shifting beach sands by soil-borne pathogens (Van der Putten *et al.* 1993). As an invader on the west coast of the U.S.A., *Ammophila* remains dominant for long periods of time. However, escape from natural enemies does not explain this contrast between its native and invasive ecological roles. By replicating experiments done in the native range, Beckstead and Parker (2003) found the negative effect of soilborne pathogens on early growth in the invaded range was at least as large or larger than their effect in the native range.

To predict whether an introduced plant is likely to benefit from escaping natural enemies, we need to understand the relative importance of host-specialist vs. host-generalist pathogens and pests in the invaded habitat (Maron and Vila 2001, Keane and Crawley 2002). If specialist pathogens predominate and host shifts are rare, native plants may be suppressed more than competing invasive species. On the other hand, if generalist pathogens dominate in a site and do not show a preference for native host species, one would not expect an introduced plant to experience release. Therefore, the wide range of results seen in the above empirical studies may be in part explained by the relative importance of specialist and generalist natural enemies. A greater understanding of the phylogenetic structure of pathogen host ranges, coupled with analysis of the phylogenetic structure of natural plant communities (Webb *et al.* 2002) may help predict the relative importance of specialist vs. generalist pathogens in different kinds of plant communities. For instance, in a high-diversity lowland tropical rainforest (300 + tree species), host generalists dominated the polypore fungal community; all of the more common fungal species were found on multiple families of host trees (Gilbert *et al.* 2002, Ferrer and Gilbert 2003). In contrast, in a nearby low diversity mangrove forest with only three tree species present (each from a different family), 88% of all polypore fungal collections belonged to just three fungal species, and each species was highly specialized on just one mangrove species

(Gilbert and Sousa 2002). Researchers are just now beginning to address the range of host specialization in different plant communities, and the wide range of outcomes suggests that many more studies will be needed before we can formulate predictive generalizations.

INTENTIONALLY INTRODUCED PATHOGENS FOR BIOLOGICAL CONTROL

A direct application of the Natural Enemies Hypothesis is deploying natural enemies from the native range of an invasive plant to control the invader population. Such classical biological control uses plants and pathogens with shared evolutionary histories but a new environmental context. There are several ways in which biological control interactions may be different from native pathogens attacking introduced hosts. First, unlike native pathogens, which are presumably adapted to the local climate, the introduced biological control agent experiences a novel environment, which could have a large impact on disease development. Case studies of failed biological control efforts provide us with many examples of the importance of the disease triangle (Morin *et al.* 1996). Second, both the host and pathogen are likely to be genetically depauperate. However, the pathogen will have been chosen specifically to be virulent on the invasive host, placing the host at a relative disadvantage for evolutionary responses. Third, only fairly host-specific pathogens are selected for biological control releases, meaning that pathogen numerical dynamics should always be closely linked to individual host density. In fact, biological control releases are an excellent opportunity to study factors influencing numerical dynamics. Not only should there be a tight connection between pathogen and host density, but initial conditions of the interaction are well known. That is, the host population is originally free of that pathogen and is usually at high density. A successful epidemic provides an opportunity to quantify both frequency-dependence of transmission of the pathogen and density-dependence as the host density declines.

Predicting the short-term and long-term success of particular biological control introductions is a matter of obvious practical importance. To make such predictions, we need to understand how the numerical dynamics of a host plant following introduction of its biocontrol agent depend on host density, disease incidence, genetic variation, and evolutionary changes in virulence or resistance. Surprisingly, while there are some cases for which we have good information on the dynamics of host numbers after release of a control agent (e.g., Hasan and Ayres 1990, Morris 1997), for many other releases the details of changes are not well documented. In particular, we should ask (i) is control more successful in genetically depauperate weeds? (ii) Do transmission rate and demographic impact of the pathogen attenuate as the host population declines? (iii) Do pathogen and host reach a stable equilibrium or are they dependent on metapopulation dynamics to persist in the landscape? Detailed information on numerical dynamics in biological control systems is scarce, but data are nearly nonexistent for long-term

genetic changes in the host or pathogen. There is great, untapped potential for biological control introductions to be used to understand the factors that drive the ecological end evolutionary dynamics of the plant-pathogen interactions. In fact, the only biocontrol study we found that tracked changes in pathogen virulence or host resistance for a plant-pathogen system was for the rust *Puccinia chondrillina* on *Chondrilla juncea* (see case study below).

Evolutionary ecology of biocontrol with pathogens

The evolutionary dynamics of interactions between weeds and pathogen biocontrol agents have important implications for the long-term success of biological control programs. Generally, we need to know, (i) has virulence of the biological control pathogen changed over time, and has this increased or decreased the success of control? (ii) Has the host developed resistance over time? (iii) Has host specificity changed over time? Recent analysis of emerging diseases has suggested that ecological host shifts (i.e., having a preadapted ability to use a newly encountered host) may predominate as causes of novel epidemics, and that host shifts may only rarely be caused by mutations that allow colonization of a new host (Schrag and Wiener 1995). However, the difficulty of observing such genetic events may distort our perspective. Van Klinken and Edwards (2002) synthesized information on host range from 352 biological control programs using herbivores. They found that host shifts were more quantitative than qualitative, that is, while preference and efficiency on novel hosts evolved, there was no evidence of evolutionary changes in fundamental host-range. Such an analysis should be done for pathogens used as biological control agents. While pathogens are generally thought to offer the opportunity for high host specificity, some have argued that high host specificity may be correlated with evolutionary lability (Brooks and McLennan 1993, Secord and Kareiva 1996). Because they are unable to simply move from an unacceptable host to a more suitable one as an animal might, specialist pathogens may experience even stronger selection for host shifts than herbivores (Roy 2001). Knowing the frequency of evolutionary host shifts in pathogens and understanding the conditions under which they occur are critical to the process of risk assessment in biological control (Secord and Kareiva 1996).

General theory of host-pathogen interactions has played a large role in the choice of biological control agents in the past (McFadyen 1998). For example, it has long been thought that sexually reproducing weeds would be harder to control because their higher levels of genetic variation interfere with pathogen population growth (Burdon *et al.* 1981); however, a more recent analysis has disputed this assertion (Chaboudez and Sheppard 1995). Similarly, the belief that pathogen populations are locally adapted to their host genotypes has had a large influence on the process of selection of control agents, with genetic analysis playing an increasing role in the careful matching of agent genotypes with the populations of origin for the weed (e.g., Holden and Mahlberg 1996). However, the evidence for close local adaptation of pathogens to their host populations is

mixed, and in fact resistance and gene-for-gene virulence should fluctuate in an asynchronous, frequency-dependent way (reviewed in Parker and Gilbert 2004). Biological control practitioners have clearly based their introduction strategies on theoretical considerations, but the simpler rules are now coming under question (McFadyen 1998). There is a need for clear predictions and modern empirical work testing those predictions to help inform the practice of biological control for the future.

An evolutionary ecology case study — *Chondrilla* and *Puccinia* on three continents

No system of classical biological control of a weed by a pathogen demonstrates the importance of interactions between environment, genetics, and numerical dynamics as well as that of *Chondrilla juncea*, rush skeletonweed (Asteraceae). Native to Eurasia, this species was introduced to the eastern United States in the late 1800s, to Australia in the early 1900s, and to the western United states in the 1930s (McVean 1966, Pryor 1967, Schirman and Robocker 1967, Supkoff *et al.* 1988). *Chondrilla* is a significant economic problem in wheat-growing regions (Panetta and Dodd 1995), and is also a widespread rangeland weed. Of three biocontrol agents that were introduced, *Puccinia chondrillina* was the most effective at reducing plant vigor (Supkoff *et al.* 1988), and within two decades of its introduction into Australia and California, *Chondrilla* densities were reduced to those typical in its native range (Wapshere *et al.* 1974, Cullen *et al.* 1982, Supkoff *et al.* 1988).

The extreme host specificity found in the *Chondrilla*/*Puccinia* system makes this example particularly interesting (Hasan 1972). *Chondrilla* is a triploid apomict, and thus reproduction is clonal. In Australia, three clonal types are present, each with a different leaf width; *P. chondrillina* causes disease only on the narrow-leaf type. The original infestation of *Chondrilla* was primarily this narrow-leaf type, but after the successful biocontrol of that clone, the intermediate-leaved clone has spread (Hanley and Groves 2002). Now there is concerted effort to introduce new strains of *Puccinia chondrillina* that are able to attack and control the other clones of *Chondrilla* (Hanley and Groves 2002).

In western North America, three different *Chondrilla* genotypes were found, distinguishable by their multi-locus isozyme phenotypes (Hasan *et al.* 1996); two genotypes are thought to have originated in Yugoslavia (Hasan and Delfosse 1995). The western US genotypes were largely resistant to the rust strain that controlled the narrow-leaf *Chondrilla* in Australia, so additional rust isolates were evaluated for use in biocontrol. Rusts collected from Yugoslavia, the putative site of origin of the US invaders, showed high virulence on some of the US genotypes, but other plants were little affected. In addition, rusts from other regions also showed high virulence. Genetic matching of hosts between the native and introduced range may yield well-adapted biocontrol pathogens when the target weed is genetically uniform, but this approach assumes high local adaptation of

the pathogen to the host. As mentioned above, such local adaptation may not be generally found in natural populations.

Sexual reproduction in both the host and pathogen play a role in this story. Teliospores of *P. chondrillina* only germinate after cold winters (Adams and Line 1984), so sexual recombination of the pathogen occurs readily only in colder regions, such as parts of eastern continental Europe. In these areas of the native range, *Chondrilla* also shows higher clonal diversity and possibly diploid sexual populations (Chaboudez and Sheppard 1995). Interestingly, populations of *Chondrilla* with high vs. low clonal diversity in the native range appear to suffer similar degrees of rust infection (Chaboudez and Sheppard 1995). In northern North America, where cold winters allow for sexual recombination of the pathogen, the evolutionary dynamics of the host-pathogen interaction are potentially more complex (Hasan *et al.* 1996). In Australia, they are particularly concerned about the possibility of introduction of new, sexual types of *Chondrilla*, which could result in a situation where the host reproduces sexually but the pathogen can not (Chaboudez and Sheppard 1995).

This example shows the importance of the "disease triangle" interaction between host, pathogen, and environment in the dynamics of plant-pathogen interactions. The case of *Chondrilla*/*Puccinia chondrillina* is one of the few for which we have such detailed information about the host's and the pathogen's genetic makeup, and more importantly, about how the interaction has played out in a number of different regions. Pathogen biological control cases offer the potential to learn a great deal about the roles of genetics, numerical dynamics, evolutionary dynamics, and environmental factors in determining the long-term outcomes of host-pathogen interactions.

CONCLUSIONS

From the studies described above, it appears that pathogens are sometimes important in the regulation of natural plant populations, may constrain populations introduced to new regions, and may, by their absence, release introduced plants from an important source of regulation. We suggest two key directions that would most advance our understanding of the importance of pathogens in plant population regulation: an integration of numerical and evolutionary dynamics for both the pathogens and plants, and a greater breadth of studies to include more plant-pathogen systems.

Biological control of invasive weeds offers exciting opportunities to evaluate the importance of pathogens in numerical regulation of plant populations, and at the same time to follow genetic changes in plant and pathogen populations. Careful monitoring for changes in host and pathogen genotypes, along with numerical dynamics, should be integral to any introduction of pathogens for biological control of invasive weeds. Equally important, we must move beyond spinning narratives and making general predictions from a handful of examples. We have

a good idea of the role pathogens *should* play in plant population regulation and biological invasions (Table 1); we now need to collect data from a diversity of systems to evaluate our predictions. How often and under what conditions are plant pathogens significant forces in regulating wild plant populations in their native ranges? How often do introduced plants fail to establish because they are attacked by local pathogens? How often is escape from pathogens a key to determining whether an introduced plant invades natural habitats? Through pathogen exclusion and addition experiments, common gardens, phylogenetic analysis of host ranges, analysis of rapid evolutionary changes in plant-pathogen interactions, and careful, creative natural history of plant diseases, we will illuminate the role of plant pathogens in biological invasions.

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