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Disease in Invasive Plant Populations

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Abstract

Non-native invasive plants can establish in natural areas, where they can be ecologically damaging and costly to manage. Like cultivated plants, invasive plants can experience a relatively disease-free period upon introduction and accumulate pathogens over time. Diseases of invasive plant populations are infrequently studied compared to diseases of agriculture, forestry, and even native plant populations. We evaluated similarities and differences in the processes that are likely to affect pathogen accumulation and disease in invasive plants compared to cultivated plants, which are the dominant focus of the field of plant pathology. Invasive plants experience more genetic, biotic, and abiotic variation across space and over time than cultivated plants, which is expected to stabilize the ecological and evolutionary dynamics of interactions with pathogens and possibly weaken the efficacy of infectious disease in their control. Although disease is expected to be context dependent, the widespread distribution of invasive plants makes them important pathogen reservoirs. Research on invasive plant diseases can both protect crops and help manage invasive plant populations.

INTRODUCTION

Invasive plants can alter biodiversity and ecosystem functions, and they are costly to manage (68, 110). The costs of non-native plants that have invaded natural and managed ecosystems in the United States are estimated to be many billions of US dollars per year (107). Invasions often start from the cultivation of plants as food, forage, fiber, or ornamentals (70). Like cultivated plants, invasive plants have been moved from their native range by humans and can experience a relatively disease-free period following introduction (45). Invasive plants can serve as sources of inoculum or reservoirs for pathogens of economically important cultivated plants. However, the pathogens that colonize invasive plants and their subsequent impacts are not often studied (58, 128, 131). Diseases of invasive plants have been studied to a limited extent in the context of biological control agents and bioherbicides (18), sources of inoculum and reservoirs of pathogens of cultivated plants (83, 147), and ecological studies of wild plants (24). Although disease frequently limits crop yields, there are few examples of pathogens regulating invasive plant populations (18, 58). Are the perceived differences in disease impacts on crops versus invasive plants due to differences in biological processes? To explore this question, we contrast ecological and evolutionary processes relevant to pathogen colonization and disease epidemics between invasive and cultivated plants (summarized in **Table 1**). We then consider case studies of disease emergence that illustrate the shared and divergent factors affecting pathogen accumulation on cultivated and invasive plants. Throughout, we highlight opportunities for increased understanding of disease in invasive plant populations.

Invasive plant: non-native, introduced plants that cause harm in a new environment, particularly native ecosystems

Pathogen accumulation: increase in number of pathogen species on a host species or population over time

Weeds: plants that cause harm to the environment, economy, human health, or animal health. Noxious weeds are government-designated species, genotypes, or cultivars that fit this description

DRIVERS OF DISEASE ON INVASIVE PLANTS VERSUS CROPS

Plant Origin, Domestication, and Naturalization

Colonization, trade in live plants, and globalization of production chains have led to the intentional introduction of many species of plants to meet the needs and desires of growing human populations. The vast majority of crop plants are cultivated beyond their center of origin. Escape from cultivation and accidental introduction by seed contamination are primary pathways of introduction for invasive plants (70, 81, 104). In the United States, deliberate introduction of cultivated horticultural plants is responsible for most invasive woody plants, whereas accidental introduction is more common for invasive grasses and forbs (81, 111).

The process of cultivation generally requires some degree of domestication to achieve desired traits, including adaptation to increase performance in a given anthropogenic environment (53, 88). Domestication can cause rapid changes in traits for resource acquisition, seed production, and growth, among others, which change interactions with the biotic community, including pathogens, herbivores, and mutualists (92). Invasive plants have often undergone multiple rounds of artificial or natural selection. Escaped cultivated plants have transitioned from wild to cultivated to invasive, whereas seed contaminants were likely already weeds adapted to agricultural or urban environments that then colonized native ecosystems (wild to weed to invasive). Some invasive plants have more complex histories because of multiple cycles of cultivation and invasion or hybridization with close relatives (143). Hybridization between weeds and crops may increase invasiveness (29). Similarly, interspecific hybridization and recombination among pathogen strains have preceded the emergence of damaging plant pathogens (17, 100, 137).

Resistance to pathogens may or may not be selected for during domestication and subsequent breeding, but economically successful genotypes remain productive in the presence of local pathogens. As cultivation intensifies, pathogens are under strong selection to break existing disease resistance in the crop (10, 132). Resistance breeding uses resistance found in noncommercialized

Table 1 Characteristics of invasive plants that are expected to affect disease impacts

Plant characteristic	Invasive plants	Impacts on plant–pathogen interaction	Contrast to cultivated plants
Origin and naturalization	Primarily cultivated plants or weeds prior to introduction; also crop–weed hybrids	Resistance and tolerance moderate disease and provide population-level resilience to disease	Domesticated; little phenotypic plasticity compared to wild plants; resistance introduced as needed
Population genetic variation	Potentially high because of multiple introductions	Genetic variation in disease resistance; ability to evolve in response to selection from pathogens	Reduced by widespread use of modern varieties; evolution in response to pathogen selection constrained by breeding and grower decisions
Local population size and density	Potentially high density but variable in time and space	Epidemics dependent on local population size and density	Generally high density and large population sizes conducive to disease
Spatial distribution	Metapopulation structure	Pathogen persists across landscape while individual patches may be uninfected	Mostly highly connected; pathogens spread rapidly
Species diversity in surrounding habitat	Potentially high diversity that can be reduced by invasion or pre-invasion disturbance	Dilution of pathogen transmission and lower disease severity	Low species diversity; higher at field margins or on small farms; intercropping and rotations to increase species diversity
Phylogenetic diversity in surrounding habitat	Highly dependent on community invaded and can be reduced by invasion or pre-invasion disturbance	Determines pathogen host range and affects pathogen colonization, persistence, and evolution	Dependent on plant and cropping system
Biotic interactions	Reduced ecological network in invaded range; increases over time	Available niche for pathogens to colonize; beneficial microbes and vectors may be absent	Management practices reduce complexity of interactions; potentially facilitating epidemics
Resource use	Colonize nutrient-rich environments but also heterogeneous	High-nutrient environments can increase disease	Nutrient-rich environments made homogeneous with fertilizers

varieties, landrace varieties, or wild relatives to increase resistance in commercial varieties. Wild relatives used as resources for disease resistance can be invasive. For example, *Aegilops* (goatgrass) and *Triticum* (wheat) are closely related genera with evidence of repeated hybridization (108). *Aegilops* spp. are sources of multiple traits for wheat breeding efforts, including resistance to leaf, stem, and stripe rusts (*Puccinia* spp.), powdery mildew (*Erysiphe graminis*), and eyespot (*Tapesia yallundae*) (121). But, *Aegilops* spp. are also seed contaminants in wheat and have become invasive in the western United States. *Aegilops cylindrica*, which is classified as a noxious weed in several US states, is reported to be an overwintering host of several wheat pathogens (38).

Phenotypic plasticity: ability of a genotype to produce different phenotypes in response to different environments

As invasive plants spread from their point(s) of introduction, heterogeneous environments impose additional selection. Many invasive plants are generalists because they often exhibit high phenotypic plasticity that allows them to thrive across different abiotic environments and biotic communities (67). Pathogens associated with generalist invasive plants may potentially infect a wide range of host species. By contrast, agricultural weeds experience strong selection on the timing of germination, flowering, and seed dispersal, which can cause them to become obligate

RESISTANCE AND TOLERANCE IN CROPS VERSUS INVASIVE PLANTS

When plant genotypes are tolerant, infection can occur and symptoms can be present, but plant fitness is not severely reduced compared with an uninfected individual (120). When plant genotypes are resistant to disease, pathogen growth and reproduction are inhibited (87), leading to fewer or no symptoms and lower direct fitness costs of disease. In agriculture, disease symptoms can make plant products unmarketable even in the absence of mortality or yield losses. Furthermore, disease tolerance can impose a fitness cost in the absence of disease (14). As a result, disease tolerance may not be an acceptable solution and is less likely to be selected for by breeders than disease resistance. In contrast, tolerance can be an advantageous response to disease in wild plants (71). Being a competent host but largely unaffected by disease at the population level may provide a competitive advantage if competing plant species are harmed by the pathogen (69, 148). Infection may prevent damage by other more harmful enemies, as has been shown for endophytic fungi providing defense against herbivores (33). Wild plants with disease tolerance can be pathogen reservoirs. For example, *Xylella fastidiosa* is an emerging plant pathogen that is hosted by species in 63 plant families but does not cause disease in most of these species (2). Specific pathogen genotypes have emerged from diverse reservoir populations to cause major crop diseases.

specialists and noxious weeds of specific crops (143). The phenotypic plasticity of invasive plants can allow escape from infection by reproducing early, before disease reaches epidemic levels. Invasive plants may also tolerate disease (see the sidebar titled Resistance and Tolerance in Crops Versus Invasive Plants) by compensating for disease-induced losses through individual- or population-level growth (**Figure 1**) (1). In contrast, modern crop seasons are timed for the market and crops are bred for determinant growth. These constraints make it more difficult for crops to escape from or compensate in response to disease. In one instance where the tables are turned, a powdery mildew that reduces fitness of invasive garlic mustard in southwest Ohio does not negatively impact susceptible native species because the native species reproduce earlier in the season (32).

Different selective pressures from host plants can cause genetic differentiation among populations of pathogens that affect crops, crop weeds, and invasive plants in natural areas. A study of toxins produced by *Fusarium graminearum* on grasses showed distinct profiles on wheat versus wild grasses, including invasive grasses (52). Alternatively, pathogens may take advantage of phenological phases of susceptibility or other dimensions of host heterogeneity to use multiple host types. The weed barley grass (*Hordeum murinum* species complex) is an important reservoir for the barley (*Hordeum vulgare*) fungal pathogen *Rhynchosporium commune* in Australia and hosts a high diversity of virulence types that could fuel ongoing adaptation to cultivated barley (83). Spatial and temporal variation in selective pressures among host types could help maintain pools of adaptive genetic variation in pathogens.

Population Genetic Variation

When an invasive species colonizes a new location, it may initially have small population sizes and lose genetic diversity during the initial genetic bottleneck. Newly established invasive populations are thus expected to be genetically less diverse than populations in the native range (117). However, multiple introductions of an invasive species can contribute diversity from genetically differentiated source populations. As a result, the genetic diversity of populations within the invaded range can exceed those in the native ranges (79). For example, although the total genetic diversity of *Ambrosia artemisiifolia* (common ragweed) in its native North American range is similar

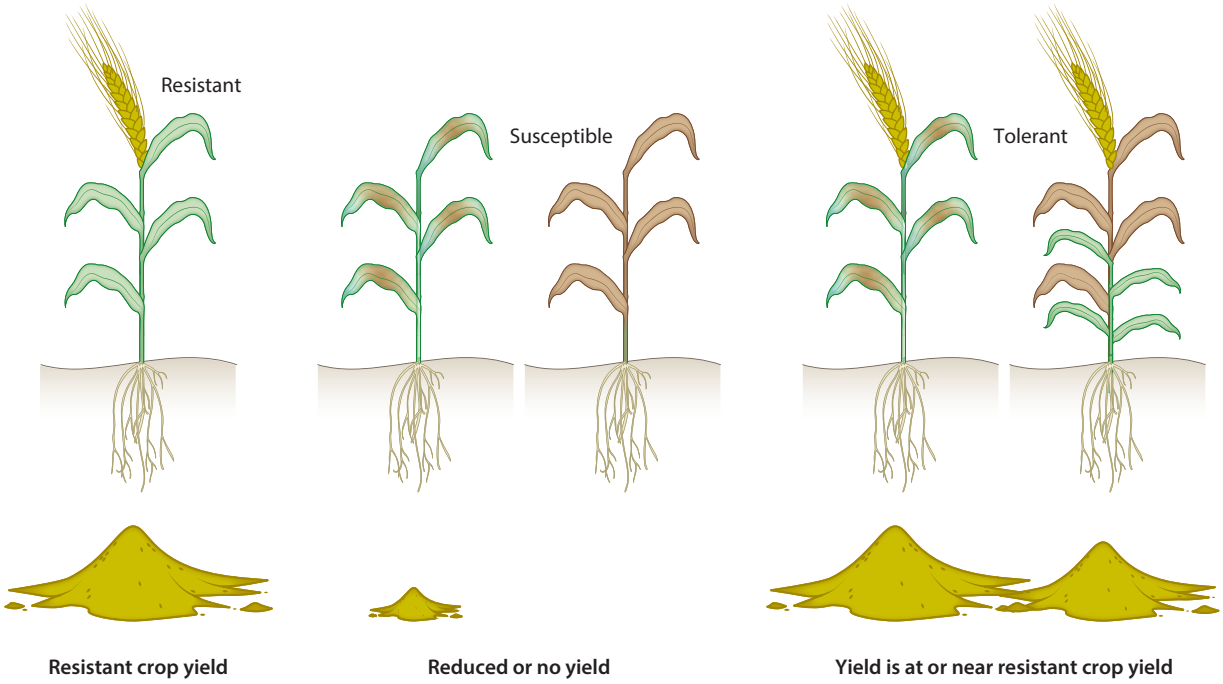

Figure 1

Compensation in response to infection in tolerant invasive plants may lead to seed production nearing that of healthy plants or, in this case, a hypothetical disease-resistant invasive plant. Tolerance may also be observed at the population level via compensation by plants experiencing lower disease severity. In invasive plants, disease tolerance may be as important as disease resistance.

to that in its invaded European range, admixture among introductions from distinct native range populations produced higher local genetic diversity in the invasive range (56). Because coevolution between wild plants and pathogens is expected to lead to genetic variation in resistance within and among host populations, multiple introductions from different source populations should increase variation in plant resistance genes in the invaded range. In the well-studied *Linum marginale–Melampsora lini* flax rust pathosystem in the Kiandra Plain of Australia, wild flax harbors at least 17 alleles for resistance to the rust with variation in resistance within and among populations (136). Genetic variation in disease resistance has been reported in invasive plant populations against fungal pathogens investigated as biocontrols (19, 25, 90).

In comparison, crops undergo a loss of genetic variation during domestication. On average, domesticated annual species retain only 60% of the genetic variation of their wild relatives (93). The majority of this loss in genetic variation is attributed to the use of modern varieties instead of landraces (142). Even when genetic diversity is maintained among varieties, modern crop production is dominated by a limited number of cultivars that have been artificially selected for desired agronomic traits, which may result in the same set of alleles across widely used cultivars. The result is a genetically uniform landscape that is ripe for the outbreak of damaging plant disease epidemics. In the 1960s, seed corn companies began using Texas male sterile cytoplasm to eliminate the need for costly detasseling. However, these hybrids were susceptible to *Cochliobolus heterostrophus*. The unseasonably warm and wet conditions of 1970 allowed this pathogen to proliferate to epidemic proportions, resulting in the loss of 15% of US corn production at a cost of US$1 billion (21). Similarly, wheat cultivars with Yr17 resistance to yellow rust (stripe rust) were

Disease prevalence: the proportion of plants with disease

widely used in Europe in the early 1990s (9). Breakdown of Yr17 resistance was detected in 1994. Although boom-and-bust cycles have been best described on cereals and rusts, many examples exist for the breakdown of resistance following widespread adoption of varieties with major gene resistance (116, 133). The rapid and repeated breakdown of resistance in numerous crop species underscores the risk posed by genetic uniformity for facilitating pathogen evolution. In an effort to reduce the risk of pathogens overcoming resistance, growing attention has been paid to the use of host cultivar mixtures to disrupt directional selection (91). A widespread example of this practice is the use of refuge corn for the maintenance of Bt toxicity. In this case, non-Bt corn is planted among Bt corn, allowing the survival of susceptible organisms and preventing the fixation of resistance alleles within insect populations (124).

In addition to greater genetic variation in resistance within populations, invasive plants should exhibit variation in disease resistance across years. Although crops are bred to quickly emerge after planting, wild populations typically have overlapping generations because of persistent between-year seed banks resulting from seed dormancy. When a disease epidemic in a single year causes high mortality or loss of seed production, the population may be protected by a seed bank, allowing recovery in the following years. This life history attribute also means that not all individuals in the years following an epidemic will be resistant to disease (26). Furthermore, dormant seed banks can facilitate the maintenance of genetic variation for resistance or tolerance of infectious diseases in host species—a within-species analog of the storage effect in community ecology.

Local Population Size and Density

Local density is an important factor in plant disease epidemiology (23) and particularly relevant to invasive plants and crops, which can form dense monocultures (66, 144). Higher local plant densities can directly increase disease prevalence because there are more susceptible individuals crowded together, facilitating transmission (23). Population density can also change host quality (i.e., through intraspecific competition), microenvironment conditions that can affect pathogen reproduction and transmission, vector foraging and movement, and population density and species diversity of nonhost plants (23). For example, cultivation of cacao in plantations promotes epidemics of witches' broom disease in part because of the high density of susceptible host tissue that emerges during synchronous flushes (109). These findings illustrate the concept of a threshold population density required for pathogen establishment in classic epidemiological models of density-dependent transmission (4, 74). Models also indicate that local population density can indirectly affect disease spread through changes in rates of seedling establishment, mortality, and disease recovery, in part through impacts on local communities (73). However, in practice, decreasing host density can also increase disease prevalence by improving the quality of individual hosts from the pathogen's point-of-view. For example, wider spacing between rows of wheat led to higher *Fusarium* head blight damage, potentially because of higher flowering rates and a larger temporal window for infection to occur (118). Plant population size also may alter pathogen dynamics via Allee effects. Garrett & Bowden (55) suggest that the heterothallic Karnal bunt pathogen *Tilletia indica* can experience reduced fecundity at low densities because different mating types must encounter each other to reproduce. A sparse host population necessarily implies low pathogen abundance, making such Allee effects more likely. The relationship of host population size to disease prevalence is likely influenced by the mode of pathogen persistence. Insect vectors of pathogens can behaviorally seek out even rare plants, likely reducing the threshold host abundance required for pathogen persistence (57).

Even when similar in average density, invasive plants and crops differ in patterns of temporal fluctuations in abundance. Crop species are often buffered from poor environmental conditions because of active modification of their environment by resource subsidies, natural enemy

protection, and amelioration of stressful abiotic conditions. In contrast, invasive species are unprotected from biotic and abiotic pressures of the local environment, with populations of the invasive plant and its pathogens fluctuating in response to variable conditions. For example, density-dependent fecundity led to oscillations in *Tripleurospermum perforatum* (scentless chamomile) density (22). Lively et al. (84) showed that the negative effects of infection by *Puccinia recondita* (a rust fungus) on *Impatiens capensis* (jewelweed) growth were lessened at low plant densities, even if infection rates were unchanged by density changes, indicating that periodic thinning may benefit overall productivity. Plant competitive ability, which is most important at high density, can be weakened by disease (50). These effects are strong enough in some cases that infected plants fail to produce seeds when plant density is high (51).

Disease incidence: the proportion of plant populations with disease

In addition to ecological effects, plant population size is likely to have evolutionary consequences. Low host numbers can imply little genetic variation for the evolution of resistance to pathogens. Whereas high host numbers provide a larger potential resource for colonizing pathogens and should sustain greater pathogen population sizes, which can harbor more adaptive pathogen genetic variation fueling adaptive evolution on the new host (87). The net effect of host population size on the likelihood of adaptive colonization by pathogens is thus unclear and likely system dependent.

Spatial Distribution

The spatial distribution of plant hosts has a major role in interactions with pathogens, disease prevalence, and epidemic development. In studies of wild pathosystems, the three general categories of host plant spatial distribution are continuous, patchy, and isolated (31). A plant species with a continuous distribution is widely distributed and highly connected by dispersal. Plants with patchy distributions have populations concentrated in patches, within which there can be considerable dispersal, but among which there is reduced dispersal. Isolated species have spatially separated populations that are not connected by dispersal. The concept of the metapopulation has become important in characterizing the dynamics of species with patchy distributions (24). A metapopulation is a collection of local populations, possibly variable in size and separated in space but with occasional dispersal of individuals among them, resulting in both gene flow across the metapopulation and (re)establishment of populations following local extinction events. Variation in population size, density, genotype frequencies, and connectivity of host and pathogen populations cause patches to experience different combinations of plant–pathogen interactions and different intensities of selection. For example, Carlsson-Granér & Thrall (31) examined anther smut (*Microbotryum violaceum*) on the wild plant *Lychnis* in Sweden and reported disease incidence to be highest in continuously distributed populations and lowest in isolated populations. Disease prevalence exhibited the opposite pattern: Low disease prevalence was found in highly connected populations rather than isolated populations. The authors attributed this pattern to the evolution of resistance in well-connected populations. For pathogen populations that undergo seasonal population crashes, persistence across the metapopulation can depend on the existence of some patches with favorable environments and large susceptible host populations, together with dispersal to other patches that permit the pathogen to recolonize after local extinction (127). Patches with small host populations or high host resistance may remain disease-free despite spatial connectedness (76, 125). The importance of metapopulation processes in both the ecological and evolutionary dynamics of wild plant–pathogen interactions was recently reviewed by Burdon & Laine (24).

Agricultural crops tend to be highly connected because of global production chains, the consolidation of seed companies, and long-distance movement of plants and their products. In

industrialized nations, crops are grown in very large and often uniform patches. Human-mediated connectivity among spatially separated growing regions increases the probabilities of pathogen colonization and spread. Indeed, most of the known pathogens of staple crops, from across their geographic ranges, can be found in highly connected economies (10). Significantly, pathogens are under strong selection to evolve virulence to widely planted genotypes, and connectivity allows for the rapid spread of locally successful pathogen strains (87). Highly isolated patches of crops are an arguably less common spatial pattern for agriculture but may accurately characterize specialty crops that are new to a geographic region and localized therein or found in more traditional agricultural systems. Patchy distributions or metapopulation structure may characterize agronomic crops grown by smallholder farmers.

Competent host: hosts that transmit a given pathogen when infected. Hosts may be competent without exhibiting disease symptoms

Disease dynamics across patchily distributed invasive plants may resemble well-studied wild plant metapopulations. *Silene latifolia* (white campion) is an invasive perennial plant found across much of North America. Anther smut of *S. latifolia* was tracked along nearly 500 roadside populations in the mountains of western Virginia from 1989 to 1993 (5). Over this period, disease incidence across populations ranged from 16% to 19%. Although the pathogen appeared homogeneous, variation in plant resistance was a key driver of disease dynamics. One area warranting more investigation is how plant–pathogen interactions shift along environmental gradients, including near geographical range margins. Pathogens with frequency-dependent transmission, such as anther-smut pathogens vectored by insect pollinators, could create host range edges. In the Italian Alps, anther-smut disease prevalence was substantially greater near the altitudinal range limit (20). The authors noted plants near the range margins could be more susceptible to infection because of either abiotic conditions or reduced genetic variation for disease resistance.

Invasive plants that are actively managed may experience higher population extinction than unmanaged wild populations. Conversely, invasiveness indicates high dispersal rates and hence higher colonization rates and connectivity among patches, which may increase infection rates because of inadvertent movement of pathogens by managers. A metapopulation perspective may help identify key patches or clusters of patches that have a disproportionate impact on disease persistence and spread. The metapopulation impacts of higher patch extinction coupled with increased pathogen dispersal are unclear.

Plant Community Diversity

Community diversity is likely to differ dramatically between environments experienced by crops and those by invasive plants. With the exceptions of small farms and eco-farms that may grow multiple crops in small plots or use intercropping, most crops are grown in monoculture, where species diversity remains low. Invasive plants may originate in relatively species-poor disturbed areas, ornamental gardens, or crop fields but then spread to undisturbed natural areas. In general, increases in species diversity, above some minimum threshold, can decrease disease prevalence, particularly at small spatial scales (62). When community diversity is high, the abundance of a particular plant host species is relatively low, so a pathogen that is a host specialist is more likely to encounter nonhosts than when species diversity is low and host abundance is high. Nonhosts are sinks for pathogen propagules and vectors, potentially reducing transmission (73). For example, increasing diversity in a grassland can reduce foliar fungal infection severity by decreasing the percent cover of competent hosts (61, 96). The strength of this dilution effect depends on the abundance of host species and their frequency in the community (60, 96). Its relevance also depends on the host range of the pathogen; a broad host range pathogen may encounter more hosts than a narrow host range pathogen, and indeed one might see amplification rather than dilution (see the sidebar titled Host Range). In agriculture, the removal of alternative hosts and

HOST RANGE

Pathogen host range is critically important in the ecological and evolutionary dynamics of wild plant–pathogen interactions, which take place in the context of diverse biotic communities. For invasive plants, pathogen host range determines whether local pathogens colonize an invasive species and whether non-native pathogens that arrived with an invasive species spread to resident species. Plant pathogens are sometimes described as generalists or specialists to indicate the range of interactions with host species. However, there are many axes of specificity (7), and host range can be difficult to define (98). Some authors have recently advocated taking an evolutionary perspective to better understand pathogen host range (98, 119). Invasive plants can escape their native pathogens, but their evolutionary history may influence their responses to new pathogens. Invasive plants may have robust defenses to new pathogens that are similar to native range pathogens with which they coevolved, such as a different member of a species complex. In contrast, invasive plants may not be evolutionarily prepared to mount a defense against pathogens with novel infection strategies.

addition of nonhosts has been employed to control pests and pathogens. Mixtures of crop species and genotypes in space and time can effectively reduce disease pressure (82, 149). Practices such as crop rotations and sequencing also increase plant diversity over time and can thereby hamper the spread of pathogens (141).

Beyond species diversity, the details of species composition in local communities can affect invasive plant–pathogen interactions. Phylogenetically closely related hosts are more likely to share pathogens (59). Phylogenetic relatedness of invasive plants to resident plants can determine whether the invasive plants are colonized by local pathogens or native plants are colonized by pathogens brought into natural areas by invasive plants. In wild plant communities, pathogens are more likely to colonize invading species that are phylogenetically closely related to resident plant species (105). In agriculture, weedy relatives of crop plants are known to serve as pathogen reservoirs and sources for crop-pathogen emergence (132, 147). Furthermore, invasion may be more likely in plant communities that are phylogenetically distant from the invader (130), in part because invasive plants that are functionally distinct from resident plants are often more successful. However, ecological traits that are key to invading particular habitats can be phylogenetically correlated (103). Thus, phylogenetic distance among plant species in a community is likely to mediate plant invasion and the opportunity for pathogen colonization. Indeed, a study of native and non-native tree and fungal interactions in France showed that interactions were driven by the phylogenetic history of the tree and the life history strategy of the fungus (140).

Biotic Interactions

Plant–pathogen interactions occur within complex ecological networks (72). Plants and their pathogens interact with other microbes, plant species, insects, and other natural enemies, leading to important impacts on disease susceptibility and transmission (48). Coinfection by multiple pathogens is relatively common in wild plant communities and agricultural systems, with antagonistic and facilitative effects on disease (43, 77, 138). Nonpathogenic microbes in the phyllosphere and rhizosphere also alter infection and disease severity (28, 75). Insects serving as vectors of pathogens and herbivores interact with pathogens through behavioral or demographic responses to changes in plant performance, immunity, or volatile compounds (13, 113).

Invasive plants are likely to host fewer pathogen species in the introduced range than the native range, at least initially (45, 64, 94). Invasive plants accumulate herbivores, pathogens, and

mutualists over time (37, 64, 78, 131), suggesting that biotic interactions likely become more complex over time. Similarly, crops introduced to new regions tend to acquire pathogens with time and intensification of cultivation because of host shifts by local pathogens and repeated imports from other production regions (10, 34). However, agricultural practices aim to restore lost mutualists and limit pathogens, insect vectors, and herbivores. Consequently, invasive plants may be most similar to crops in their suite of interactions with microbes and insects early in invasion rather than at later stages.

Recently introduced invasive plants and crops likely have reduced networks of biotic interactions compared to native wild plants. Depending on the pathogen, subsequent disease emergence may be limited by initially low diversity pathogen and insect vector assemblages associated with the plant. For example, insect-vectored viruses are unlikely to become established in the absence of their vector species (10). Diseases that require coinfection by multiple pathogens may take longer to become established if the pathogens are not introduced together, e.g., helper-dependent viruses (35). The absence of herbivores that induce plant defenses may allow more targeted immune suppression of pathogens (145). However, reduced biotic networks may also facilitate establishment and spread of pathogens. For example, direct and indirect antagonistic interactions from microbes can limit pathogen establishment, and antagonistic interactions may be weaker in sparser ecological networks (11, 75, but see 6). Reduced diversity in the plant phytobiome, together with large or dense invasive plant populations, provides an abundant resource for pathogens to exploit, making pathogen colonization and establishment potentially more likely. Indeed, Anderson et al. (3) identified agricultural intensification as one of the main causative factors for the emergence of crop diseases.

Resource Availability

Resource availability can influence the severity and prevalence of disease by promoting host fitness, pathogen fitness, and host defenses (106, 126). A combination of nutrient inputs and pest management strategies should provide a net benefit to crops, despite the advantages pathogens gain from the enriched resources available within individual hosts. Invasive plants also may inadvertently benefit from an interaction between pest reduction and nutrient availability. The combination of enemy release and access to nutrient-rich environments is one possible mechanism for successful invasion (15, 65). Resources essential for both plants and their pathogens include nitrogen (N), phosphorus (P), potassium (K), and other nutrients. Fertilizers, N deposition, and N fixation by legumes can be sources of nutrients for both crops and invasive plants. Crops are more likely to receive high nutrient inputs through fertilizers than invasive plants, and crops can have higher leaf N and/or P content than their wild plant counterparts even in the absence of fertilizers (36, 47, 86), suggesting that pathogens may experience a more nutrient-rich environment in agricultural systems. However, fertilizer use is highly variable at a global scale and greater fertilizer use does not necessarily lead to more plant nutrient uptake (54, 86).

Higher nutrient content of plants can either increase or decrease disease severity and prevalence depending on whether the resource provides a larger benefit to the host or the pathogen (39). The addition of N and P to grasslands has increased the prevalence of viruses that infect invasive grasses (16, 123) and foliar fungal infection severity on C-4 grasses (95). Similarly, N fertilizers enhanced the performance of aphids (virus vectors) (134) and leaf rust severity on wheat (118). Increases in pathogen fitness on hosts may be due to direct increases in resource availability or indirect increases through host growth (146). The addition of N and P can also increase the relative abundance of non-native plants in grasslands (122), which may also increase disease prevalence (61). In contrast, the addition of K has a more consistent negative effect on disease severity, suggesting that plant defenses benefit from the nutrient more than pathogens do (39).

CASE STUDIES OF PATHOGEN EMERGENCE ON CULTIVATED AND INVASIVE PLANTS

The factors discussed above are expected to affect the ecological and evolutionary dynamics of disease in invasive plant populations (**Table 1**). Here, we examine case studies of cultivated and invasive and plants, focusing on pathogen emergence on both invasive and cultivated plants.

Pathogen Emergence on Non-Native Plants

Many crops have been cultivated outside of their native range for hundreds of years and the process of pathogen colonization and establishment on these crops can only be reconstructed in retrospect (132). Pandemic pathogens, by definition, have spread from their geographic and evolutionary origin. Long-distance movement often precedes ecologically and economically devastating plant disease epidemics (3). Pathogens also emerge locally via ecological and evolutionary mechanisms, including host shifts facilitated by gene acquisition via horizontal gene transfer, hybridization, or recombination. Here, we use cultivated *Eucalyptus* to illustrate the potential processes of pathogen emergence on invasive plants but note that pathogen accumulation may be accelerated in intensively cultivated plants compared with accidentally introduced invasive plants.

Ornamental cultivation of *Eucalyptus* spp. outside of their native range of Australasia began hundreds of years ago with the result that some are considered invasive species (112). Commercial cultivation of *Eucalyptus* has dramatically increased in the past 30 to 40 years (27). The majority of pathogens that affect *Eucalyptus* were discovered first in the introduced range, with some later discovered to be emerging in managed *Eucalyptus* forests in Australia (27). The Mycosphaerella and Teratosphaeria leaf diseases that affect plantations outside the native range were confirmed to have originated in Australia and moved with *Eucalyptus* germplasm in the form of seed or vegetative material (27). Destructive eucalypt pathogens have colonized *Eucalyptus* where it is cultivated. For example, *Puccinia psidii* is endemic to Myrtales in South America, where it colonized *Eucalyptus* and then spread from *Eucalyptus* in South America to plantations in North America, Asia, Africa, and Australia. South Africa has non-native plantations of *Eucalyptus*, acacias, and pines that have all accumulated pathogens (34). Of 26 pathogens recorded on *Eucalyptus* in South Africa, 23 were non-native and only three had shifted from native trees. Of these non-native pathogens, the majority infect plants across multiple families, which may have aided their global movement. In contrast, Crous et al. (34) found that non-native pines in South Africa accumulated only eight pathogens despite being present over a longer period of time, possibly because pine pathogens tend to be conifer specific, which reduces the opportunity for pathogen colonization coincident with the introduction of nonconifer host species. In addition, there are no pines and few conifers native to South Africa to serve as local pathogen sources.

Novel Diseases of Southern Highbush Blueberry

The blueberry industry in Florida represents a case of a specialty crop recently established in a region with native close relatives and rapid accumulation of local pathogens. The primary production of blueberry in Florida is the variety referred to as southern highbush blueberry (SHB), an interspecific hybrid of several diverse, outcrossing wild blueberry species (8, 85). The first SHB variety 'Sharpblue' was released in 1984, resulting in approximately 1,500 acres planted in Florida by 2002 and an estimated 8,000 acres in production in 2019 (139). Plant disease resistance was not a primary selection factor in the breeding program. The 1990s and early 2000s could be considered a brief honeymoon phase for the industry. In the first 20 years of production, disease problems were minor in well-tended plantings and included stem blight and canker diseases and Phytophthora

root rot (85). Numerous foliar fungal diseases also occurred and caused defoliation in summer or fall of some years and on some genotypes, but it was hypothesized that most native blueberries exhibited high levels of horizontal resistance to all major blueberry diseases in Florida. However, in the past decade, several new endemic pathogens that are difficult to manage have accumulated on SHB and had not previously been reported on SHB or any related native species. These pathogens include bacterial wilt caused by *Ralstonia solanacearum* and bacterial scorch caused by *Xylella fastidiosa* (30, 63, 99, 102).

The bacterial scorch pathogen *X. fastidiosa* that has recently colonized cultivated blueberry is an emerging global threat to agriculture because it infects a diverse array of woody plants, is adaptable via recombination among strains, and causes asymptomatic infection of many hosts that facilitate long-distance movement without phytosanitary detection (2). In Florida, populations of *X. fastidiosa* on SHB appear to be diverse and changing, with the initial host shift to blueberry attributed to homologous recombination events among subspecies (100). Because both *X. fastidiosa* subsp. *multiplex* and subsp. *fastidiosa* can cause bacterial wilt in SHB, it is possible that recombination occurred in SHB (101). It remains unclear where the *X. fastidiosa* populations on SHB came from or if wild *Vaccinium* populations are infected.

The most recent and problematic disease to emerge is bacterial wilt, which has been diagnosed throughout the production range of SHB in Florida and has been primarily associated with the susceptible variety 'Arcadia' (99). On farms where 'Arcadia' is grown, other less susceptible varieties have developed symptoms in rows adjacent to 'Arcadia.' Genome sequences from *R. solanacearum* strains collected across the state have revealed three distinct phylotypes: IIA sequevar 38, IIA sequevar 7, and phylotype I sequevar 13 (99). The discovery of multiple endemic *R. solanacearum* phylotypes on SHB indicates that local pathogens shifted to SHB from other plant hosts.

As SHB production area increased in Florida, pathogens have increasingly limited industry growth and profitability despite significant resource inputs. It appears that pathogen emergence has occurred not because of a static transfer of existing pathogen communities onto the new host but rather through the interplay of several complex mechanisms involving pathogen colonization, pathogen evolution, and expansion of the industry into new climates conducive to disease.

Crop-Facilitated Pathogen Accumulation on Invasive *Sorghum*

Sorghum species are cultivated for grain, forage, biomass, and sugar syrup, and some are invasive. *Sorghum halepense*, commonly known as Johnsongrass, has naturalized in many areas outside its native range and is a problematic agricultural weed and invader of natural areas in the United States (115). Mitchell & Power (94), in their analysis of enemy release of North American invaders native to Europe, identified *S. halepense* as an outlier because it is an invasive plant and noxious weed that has not escaped its native range pathogens and is also accumulating new pathogens. In contrast, other plants with similar degrees of pathogen escape had a lower noxiousness score and, of the non-native plants examined, none had comparable pathogen accumulation. In addition to *S. halepense*, conspecifics of cultivated sorghum (*S. bicolor*) are also noxious agricultural weeds.

Invasive *Sorghum* spp. are illustrative of the potential synergism among invasive plants, agricultural weeds, and agricultural crops in regard to pathogens and disease. Because phylogenetic similarity is often the most important predictor of host range, pathogens of *Sorghum* spp. are expected to move among crops and weeds. In fact, pathogens are shared among *S. halepense* and corn (129), indicating broader movement of pathogens within the Poaceae. Multiple invasive plants and crops within the same taxonomic group likely create opportunities for pathogen host shifts to *Sorghum* spp. from other grasses by providing a large well-connected host community for pathogen colonization and spread. Furthermore, imports of crop breeding material, grains, and forage grasses

from Eurasia probably brought pathogens from the native range to the invasive range. The shared phylogenetic and geographic history of cultivated and weedy *Sorghum* spp. helps explain the lack of enemy escape and the gain of native pathogens on *S. halepense*. In addition, gene flow from crops to wild relatives is documented for several systems, including sorghum (41, 97). It is possible that traits selected for in cultivated varieties (114) could spread to invasive *Sorghum* spp. by crop–weed hybridization. We do not have evidence that disease resistance has moved from crops to invasive wild relatives, but the possibility is intriguing. If pathogens accumulate on invasive plants in part via cultivated relatives but cultivated relatives also provide genetic resistance that introgresses into the invasive population, then the cultivated–invasive relationship could accelerate the plant–pathogen arms race in invasive populations.

Disease Emergence on the Invasive Grass *Microstegium vimineum*

The annual C4 grass *Microstegium vimineum* (Trin. A. Camus, stiltgrass) is an invasive species in the United States that experienced an extended release from pathogens. It was introduced accidentally from Asia to the eastern United States in the early 1900s (42). Invasive populations became widespread and abundant starting in the late 1990s and early 2000s. It is now distributed in more than 25 midwestern and eastern US states where it can create large, dense populations that suppress native trees and understory species (44) and alter soil properties (40, 80).

The invasive success of *Microstegium* was thought to be due, in part, to escape from enemies because no pathogens and few herbivores were known to infect or attack the species. In 2010, however, foliar fungal pathogens from the *Bipolaris* genus were documented infecting *Microstegium* and experiments demonstrated infection of co-occurring native species (46). A broad field survey of more than 80 sites in 18 states revealed genetically diverse *Bipolaris* species widely infecting invasive *Microstegium* populations (131). The primary predictor of population infection was time since invasion, suggesting that pathogens were accumulating on invasive populations over time. Furthermore, experimental suppression of pathogens using fungicides at multiple sites in Indiana and West Virginia resulted in significantly greater *Microstegium* biomass and seed production (46, 131), indicating that the pathogens could influence invader population dynamics. However, the consequences of pathogen emergence on *Microstegium* may be more complicated because pathogens may also spillover to native species where they could have greater population-level effects on native species than on the invader (45). Key questions remain, such as whether emerging and accumulating pathogens will suppress invasive *Microstegium* and alter competitive effects on native species, whether pathogens will reduce the competitive ability of native co-occurring species, and how accumulating pathogens will affect invaded communities. It is also unknown how the accumulation of fungal pathogens affects the massive amounts of litter that are produced by *Microstegium* each year. Fungal pathogens such as *Bipolaris* contain toxins that could directly alter litter decomposition or indirectly affect decomposition by changing the succession of microbial decomposers (49).

CONCLUSIONS

The ecological plasticity and evolutionary adaptability that are key to the success of invasive plants are analogous to traits of damaging and globally invasive plant pathogens, such as *X. fastidiosa* and *Phytophthora cinnamomi*. Invasive plants have spatial, genetic, and historic connections with agricultural crops but often inhabit similar ecological and evolutionary contexts as native wild plants (58). Conditions that cause crop failure, mainly high pathogen loads in a conducive and homogeneous environment containing an abundant susceptible host, are not typically replicated in invasive plant populations. Even high-density invasive plants are in heterogeneous environments that may vary

in conduciveness to pathogen transmission, and they experience more genetic and ecological variation than do crops. This variation stabilizes disease dynamics by modifying the interactions between invasive plants and pathogens over space and time and may weaken the efficacy of pathogens as agents that could keep invasive populations in check. The metapopulation-like spatial structure of invasive plants and their pathogens also challenges the use of biocontrol pathogens for population management. Biocontrol in natural areas may be most useful when occasional epidemics causing high levels of invasive plant mortality allow unaffected native species to regain a foothold. In actively managed natural areas, suppression of invasive plants by pathogens may provide an opportunity for native vegetation restoration (89). Pathogens may also be effective biocontrols when inoculum can be artificially increased, as has been proposed for *Puccinia punctiformis* against *Cirsium arvense* (Canada thistle) (12).

Invasive plant species are unique in providing bridges among urban, agricultural, and natural ecosystems. Their colonization of roadsides, cultivated or fallow land, urban greenspaces, and disturbed and undisturbed natural areas may provide green and brown bridges for pathogens to move among hosts and environments (135). This coupling of habitats may contribute to pathogen accumulation on invasive plants but also increases the likelihood that novel pathogen species or genotypes spread from invasive plants to economically important crop plants or threatened native plants (**Figure 2**). Altogether, greater study of invasive plant diseases can have dual benefits of protecting economically important crops from diseases and safeguarding natural areas.

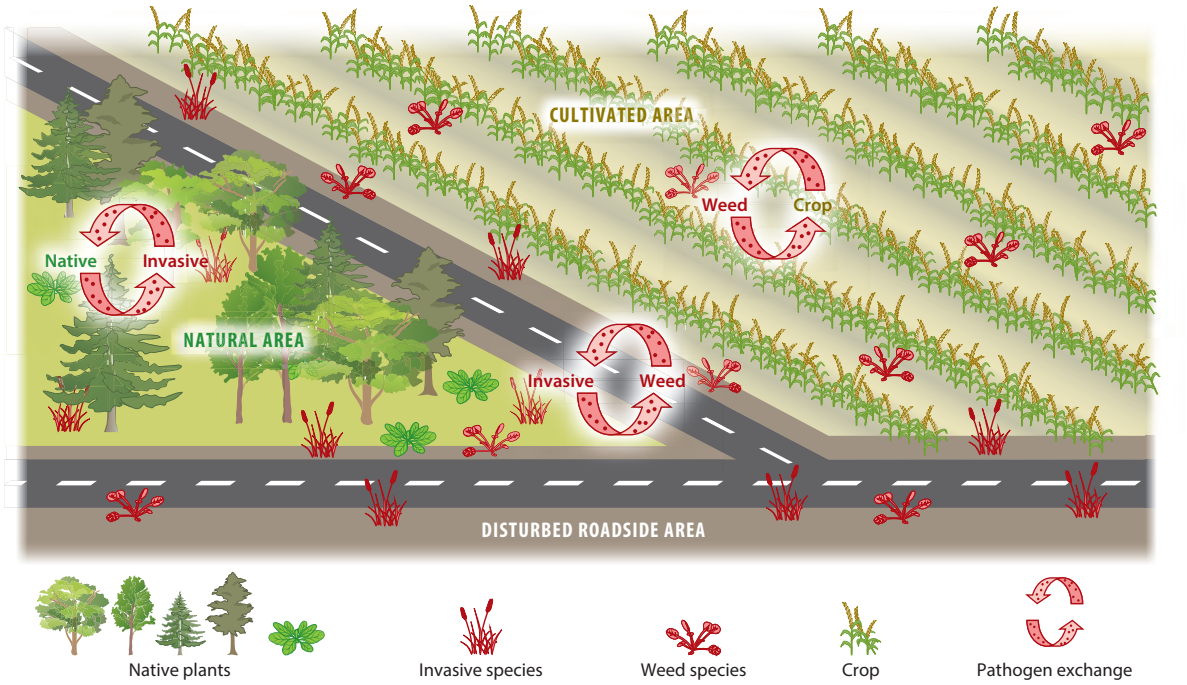

Figure 2

Invasive plants colonize natural areas but also inhabit disturbed areas such as roadsides and, along with agricultural weeds, fallow fields. Non-native invasive plants and weeds may facilitate the movement of pathogens between crops and native plants. Pathogens that emerge first on invasive plants can shift to cultivated plants. In the other direction, invasive pathogens may be transported on cultivated plants or seed, spread from crop to agricultural weeds, from weeds to invasive plants, and from invasive plants in natural areas to native plants.

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