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 as 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 as compared to cultivated plants, the dominant focus of the field of plant pathology. Invasive plants experience more spatial, genetic, biotic, and abiotic variation than cultivated plants, which is expected to stabilize the ecological and evolutionary dynamics of interactions with pathogens. While disease is expected to be context dependent, the ubiquity 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, 111). The costs of non-native plants that have invaded natural and managed ecosystems in the United States are estimated to be many US$ billions per year (108). Invasions often start from 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, 130, 133). 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 (84, 148), and ecological studies of wild plants (24). While 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 common and divergence factors affecting pathogen accumulation on cultivated and invasive plants. Throughout, we highlight opportunities for increased understanding of disease in invasive plant populations.

DRIVERS OF DISEASE ON INVASIVE PLANTS VERSUS CROPS

Plant origin, domestication and naturalization

Colonization, demand for and 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, 82, 105). In the United States, deliberate introduction of cultivated horticultural plants is responsible for most invasive woody plants, while accidental introduction is more common for invasive grasses and forbs (82, 112).

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, 89). 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 (93). Invasive plants have often undergone multiple rounds of artificial or natural selection. Escaped cultivated plants have transitioned from wild to cultivated to invasive, while seed contaminants were likely already weeds adapted to agricultural or urban environments (wild to weed to invasive). Some invasive plants have more complex histories due to multiple cycles of cultivation and invasion, or hybridization with close relatives (144). Hybridization between weeds and crops may increase invasiveness (29). Similarly, interspecific hybridization and recombination among pathogen strains has preceded the emergence of damaging plant pathogens (17, 101, 139).

Resistance to pathogens may or may not be selected for during domestication and subsequent breeding, but the selected genotypes must have survived and continued to be

productive in the presence of local pathogens. As cultivation intensifies, pathogens are under strong selection to break existing disease resistance in the crop (10, 134). Resistance breeding uses resistance found in non-commercialized or 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) is a genus closely related to *Triticum*, with evidence of repeated hybridization between the genera in their evolutionary history (109). *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*) (122). 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 pathogens (38).

As invasive plants spread from their point(s) of introduction, heterogeneous environments impose additional selection. Invasive plants can contain generalist and specialist phenotypes. Generalist types are fit across different habitats, whether natural or agricultural, while specialist types have adapted to particular habitats. 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), and if they carry pathogens those pathogens 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 specialists and noxious weeds of specific crops (144). The phenotypic plasticity of invasive species can allow for escape or tolerance to pathogen infection (see Sidebar: **Resistance and tolerance in crops versus invasive plants**). Annual invasive plants may escape fitness effects of disease by reproducing early, before disease reaches epidemic levels, or they may be resilient to disease due to individual or population level compensatory responses (Figure 1; (1)). In contrast, modern crop seasons are timed for the market and crops are bred for determinant growth. These constraints prevent escape from or compensation 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 between pathogens that affect crops, crop weeds, and invasive plants. 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 distinct periods of susceptibility to use multiple host types. Weedy barley grass is an important reservoir for the barley fungal pathogen *Rhynchosporium commune* in Australia hosting a high diversity of virulence types that could fuel ongoing adaptation to barley (84).

Population genetic variation

When an invasive species colonizes a new location, it may initially have small population sizes and lose genetic diversity through the resulting genetic bottleneck. Newly established invasive populations are expected to be genetically less diverse than populations in the native range (118). 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 (80). For example, while the total genetic diversity of *Ambrosia artemisiifolia* (common ragweed) in its native North American range is similar 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 (138). Genetic variation in disease resistance has been reported in invasive plant populations against fungal pathogens investigated as biocontrols (19, 25, 91).

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 (94). The majority of this loss in genetic variation is attributed to the use of modern varieties instead of landraces (143). 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 1960's, seed corn companies began using Texas male sterile cytoplasm to eliminate the need for costly detassling. However, these hybrids were susceptible to *Cochliobolous 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 (21). Similarly, wheat cultivars with Yr17 resistance to yellow rust (stripe rust) were 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 (117, 135). The rapid and repeated breakdown of resistance in numerous hosts 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 (92). 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 (125).

In addition to greater genetic variation in resistance within populations, invasive plants should exhibit variation in disease resistance across years. While crops are bred to quickly emerge after planting, wild populations typically have overlapping generations due to 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 following years. This life history attribute also means that not all individuals in the years following an epidemic will be resistant to disease (26).

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, 145). Higher local plant densities can directly increase disease incidence 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 the population

density and species diversity of non-host plants (23). For example, cultivation of cacao in plantations promotes epidemics of witches' broom disease in part due to the high density of susceptible host tissue that emerges during synchronous flushes (110). 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 incidence 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 due to higher flowering rates and a larger temporal window for infection to occur (119). Plant population size also may alter pathogen dynamics via Allee effects. Garrett and 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, leading to frequency-dependent rather than density-dependent transmission, which in turn implies there is no 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 often are buffered from poor environmental conditions because of active modification of their environment by resource subsidies and natural enemy protection. In contrast, invasive species are largely subject to the full extent of the 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. (85) showed that the negative effects of *Puccinia recondita* (a rust) infection 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).

In addition to ecological effects, plant population size is likely to have evolutionary consequences. Low host numbers can imply little genetic variation for evolution of resistance to pathogens. On the other hand, high host numbers provide a larger target for pathogen spillover from other hosts and should sustain larger pathogen population sizes, and thus pathogen genetic variation for adaptive evolution on the new host (88).

Spatial distribution

The spatial distribution of plant hosts has a major role in interactions with pathogens, disease incidence and epidemic development. In studies of wild pathosystems, three general categories of host plant spatial distribution are continuous, patchy and isolated (31). A continuous distribution is when a plant species is widely distributed and highly connected. Plants with patchy distributions have populations concentrated in patches connected by dispersal. Similarly, isolated refers to 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 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 and 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. Results for disease prevalence were opposite to this pattern: low disease prevalence in highly connected populations, compared to 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 with dispersal to other patches such that the pathogen can recolonize after local extinction (129). Patches with small host populations or high host resistance may remain disease-free despite spatial connectedness (77, 127). The importance of metapopulation processes in both the ecological and evolution dynamics of wild plant-pathogen interactions was recently reviewed by Burdon and Laine (24).

Agricultural crops tend to be highly connected, due to consolidation of seed companies, global production chains, and long-distance movement of plants and their products. In industrialized nations, crops are grown in very large patches. Human-mediated connectivity among spatially separated growing regions increases the probabilities of pathogen colonization and spread. Indeed, staple crops in highly connected economics are nearing pathogen saturation, meaning that all known pathogens of a given crop are or have been reported across its cultivated range (10). Significantly, pathogens are under strong selection to evolve virulence to widely planted genotypes and connectivity allows for rapid spread of locally successful pathogen strains (88). Highly isolated patches of crops are less common but may accurately characterize specialty crops that are new to a geographic region or in more traditional agricultural systems. Patchy distributions or metapopulation structure may characterize agronomic crops grown by small holder farmers.

Disease dynamics across patchily distributed invasive plants may resemble well-studied wild plant metapopulations. *Silene alba* (white campion) is a non-native, weedy perennial plant found across much of North America that was the focus of influential studies of plant disease ecology. Anther smut of *S. alba* was tracked along nearly 500 roadside populations in the mountains of western Virginia from 1989 to 1993 (5). Over this period, disease incidence ranged from 16 to 19%. While the pathogen appeared homogeneous, variation in plant resistance was a key driver of disease dynamics. An 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, could create host range edges. In the Italian Alps, disease prevalence was substantially greater near the altitudinal range limit (20). The authors noted plants could be more susceptible to infection due to abiotic conditions or due to reduced genetic variation for disease resistance.

Invasive plants that are actively managed may experience higher population extinction than unmanaged wild populations. Conversely, their invasiveness itself indicates high dispersal rates, and hence higher colonization rates and connectivity among patches, which may increase

infection rates due to inadvertent movement of the pathogen by managers. A metapopulation perspective may help identify key areas 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. Most crops are grown in monoculture, with the exceptions of small farms and eco-farms that may grow multiple crops in small plots or use intercropping, where species diversity remains low. Invasive plants may originate in relatively species poor disturbed areas, ornamental gardens, or crop fields, but 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 host species is relatively low, so a pathogen is more likely to encounter non-hosts than when species diversity is low and host abundance is high. Non-hosts 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 less competent hosts (61, 97). The strength of this dilution effect depends on the abundance of host species and their frequency in the community (60, 97). 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 sidebar: **Generalist and specialist pathogens**). In agriculture, the removal of alternative hosts and addition of non-hosts has been employed to control pests and pathogens. Mixtures of crop species and genotypes in space and time can effectively reduce disease pressure (83, 150). Practices such as crop rotations and sequencing also increase plant diversity over time (142).

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 (106). In agriculture, weedy relatives of crop plants are known to serve as pathogen reservoirs and sources for crop pathogen emergence (134, 148). Furthermore, invasion may be more likely in plant communities that are phylogenetically distant from the invader (132), in part because invasive plants that are functionally distinct from resident plants are more successful, except that ecological traits that are key to invading particular habitats can be phylogenetically correlated (104). Thus, phylogenetic distance among plants 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 fungus interactions in France showed that interactions were driven by the phylogenetic history of the tree and the life history strategy of the fungus (141).

Biotic interactions

Plant-pathogen interactions occur within complex ecological networks (72). Plants and their pathogens interact with other microbes, plant species, and insects, 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, 78, 140). Non-pathogenic 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 changes in plant performance, immunity, or volatile compounds (13, 114).

Invasive plants are likely to host fewer pathogen species in the introduced range than the native range, at least initially (45, 64, 95). Invasive plants accumulate herbivores, pathogens, and mutualists over time (37, 64, 79, 133), 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, due 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 and insect vectors and herbivores. Consequently, invasive species 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 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 (146). However, reduced biotic networks may also facilitate establishment and spread of pathogens. 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, provide 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 (107, 128). A combination of nutrient inputs and pest management strategies should provide a net benefit to crops, despite 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, 87), 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, 87). In addition, N and P addition can increase the relative abundance of non-native plants in grasslands (123).

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). N and P addition to grasslands has increased the prevalence of viruses that infect invasive grasses (16, 124) and foliar fungal infection severity on C-4 grasses (96). Similarly, N fertilizers enhanced the performance of aphids (virus vectors) (136) and leaf rust severity on wheat (119). Increases in pathogen fitness on hosts may be due to direct increases in resource availability or indirect increases through host growth (147). Nutrient addition may also increase disease prevalence through increases in invasive species abundance (61). In contrast, K addition has a more consistent negative effect on disease severity, suggesting that plant defenses benefit from the nutrient more than pathogens (39).

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 following the "honeymoon" period typically experienced by 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 (134). 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, with the stipulation that pathogen accumulation is accelerated in intensively cultivated plants.

Ornamental cultivation of *Eucalyptus* spp. outside of their native range of Australia began hundreds of years ago with the result that some are considered invasive species (113). Commercial cultivation of *Eucalyptus* has dramatically increased in the last 30 to 40 years (27). The majority of pathogens that affect *Eucalyptus* were discovered in the introduced range, with some emerging in managed *Eucalyptus* forests in Australia (27). The Mycosphaerella and Teratosphaereia leaf diseases that affect plantations outside the native range were confirmed to have originated from Australia and moved with *Eucalyptus* germplasm seed or vegetative material (27). Destructive eucalypt pathogens have also host-shifted where *Eucalyptus* 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 accumulated pathogens (34). Of 26 pathogens recorded on *Eucalyptus*, 23 were non-native pathogens and only three shifted from native trees. Of these non-native pathogens, the majority infect plants across families, which may have aided their global movement. In contrast, Crous et al. (34) found that pines accumulated only eight pathogens over a longer period of time, possibly because pine pathogens tend to be conifer-specific and thus face a narrower bottleneck to introduction from other regions, and 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. Wild populations of eight *Vaccinium* spp. have been studied in Florida and the diverse outcrossing species *V. darrowii*, *V. elliottii*, and *V. fuscatum* were used for the interspecific hybrid variety releases referred to as southern highbush blueberry (SHB) (8, 86). The native rabbiteye blueberry (*Vaccinium virgatum*) has been cultivated thoughout Florida's history with relatively low inputs on small acreages but is not particularly well represented in the genetic background of SHB. While SHB are not invasive in Florida, North American highbush blueberries and their various hybrids have invaded some bogs, tree plantations, roadsides and other non-agricultural lands of Lower Saxony in northern Germany where they escaped cultivation (76).

The first SHB variety 'Sharpblue' was released in 1984 resulting in about 1500 acres planted in Florida by 2002, and an estimated 8000 acres in production in 2019 (2017 Florida Census of Agriculture). The production system involves intense inputs of resources estimated to cost as much as $20,000 per acre (126). Plant disease resistance was not a primary selection factor in the breeding program, except that genotypes had to survive long enough to be selected, or about 4 years. 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 (86). Numerous foliar fungal diseases also occurred and could cause 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. In the last decade, several new difficult to manage endemic pathogens have accumulated on SHB that 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, 100, 103).

The bacterial scorch pathogen, *X. fastidiosa*, is an emerging global threat to agricultural because it infects a diverse array of woody plants, is adaptable via recombination among strains, and it 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 (101). Because both *X. fastidiosa* subsp. *multiplex* and subsp. *fastidiosa* can cause bacterial wilt in SHB, it is possible that recombination occurred in SHB (102). Different varieties of SHB are susceptible and tolerant in the field. Artificial greenhouse inoculations yield severe symptoms when varieties currently considered tolerant and without symptoms in the field are injected with *Xylella*. 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' (100). On farms where Arcadia are grown, other less susceptible varieties have developed symptoms in rows adjacent to Arcadia. Greenhouse inoculations of all current commercially grown varieties of SHB quickly die when inoculated with high concentration suspensions of *R. solanacearum* at or above 30°C (Harmon, unpublished data). Genome sequences from *R. solanacearum* strains collected across the state have revealed

three distinct phyllotypes: IIA sequevar 38, IIA sequevar 7, and phylotype I sequevar 13 (100). Finding of multiple endemic *R. solanacearum* phylotypes on SHB indicate that local pathogens shifted to SHB from other plant hosts.

As production area increased in Florida, pathogens have limited growth and profitability of the industry, despite significant resource inputs. It appears that pathogen emergence has not occurred due to a static transfer of existing pathogen communities onto the new host, but rather through several complex mechanisms involving pathogen introduction, 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 also 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 U.S. (116). Shattercane is a conspecific of cultivated sorghum (both *S. bicolor*) and a noxious agricultural weed. Mitchell and Power (95), 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 while 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 they examined, none had comparable pathogen accumulation.

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 the most important predictor of host range, pathogens of *Sorghum* spp. are expected to move among crop and weeds. In fact, pathogens are shared among *S. halepense* and corn (131), 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*. But, why is *S. halepense* successful as a weed even in the presence of disease? Gene flow from crops to wild relatives is documented for several systems, including sorghum (41, 98). It is possible that other traits introduced to cultivated varieties to provide resistance to common pathogens (115), 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 are also providing 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. Introduced accidentally from Asia to the eastern US in the early 1900s, it was first documented in 1919 in Tennessee (42). Invasive populations became widespread and abundant starting in the early

2000s. It is now distributed in more than 25 midwestern and eastern US states where it can grow to large biomass, suppressing native trees and understory species (44), and altering soil properties (40, 81).

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 over 80 sites across 18 states revealed genetically diverse *Bipolaris* species widely infecting invasive *Microstegium* populations (133). The primary predictor of population infection was time since invasion, suggesting that pathogens were accumulating on invasive populations over time. Moreover, experimental suppression of pathogens using fungicides at multiple sites in Indiana and West Virginia resulted in significantly greater *Microstegium* biomass and seed production (46, 133), 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: Will emerging and accumulating pathogens will suppress invasive *Microstegium* and alter competitive effects on native species, reduce the competitive ability of native co-occurring species, or have no long-term consequences for 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 *Xylella fastidiosa* and *Phytophthora cinnamomi*. Invasive plants have spatial, genetic, and historic connections with agricultural crops, but inhabit similar ecological and evolutionary contexts as native wild plants (58). Conditions that cause crop failure, mainly high pathogen loads in a conducive and homogenous environment containing an abundant susceptible host, are not typically replicated in invasive plant populations. Even high-density invasive plants are in heterogenous environments that may vary in conduciveness to pathogen transmission, and they experience more spatial, genetic, biotic, and abiotic variation than do crops. This variation stabilizes disease dynamics by modifying the interactions between invasive plant and pathogens over space and time, and may weaken the efficacy of pathogens as agents keeping 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 wildlands may be most useful when occasional epidemics causing high levels of invasive plant mortality allow unaffected native species to regain a foothold. On actively managed lands, suppression of invasive plants by pathogens may provide an opportunity for native vegetation restoration (90). 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 wildlands may provide "green and brown bridges" for pathogens to move among hosts and environments (137). This coupling of habitats may contribute to pathogen accumulation on invasive plants, but also increases the likelihood that invasive plants introduce new pathogens or genetic variation to pathogens on 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 the ecology of natural areas.

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TERMS AND DEFINITIONS

Invasive species: Non-native, introduced species that causes harm in a new environment, particularly to native ecosystems.

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

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

Competent host: A host that transmits a given pathogen when infected. A host may be competent without exhibiting disease symptoms.

SIDEBARS

Resistance and tolerance in crops versus invasive plants

Tolerance is when infection occurs and symptoms are present, but plant fitness is not severely reduced as compared to a less infected individual (121). 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, 149). 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). From diverse reservoir populations, specific genotypes have emerged to cause major crop diseases.

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 if local pathogens colonize an invasive species and if 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 host species interactions. But, there are many axes of specificity (7) and host range can difficult to define (99). Authors have recently advocated taking an evolutionary perspective to better understand pathogen host range (99, 120). Invasive plants can escape their native pathogens, but their evolutionary history is expected to influence their responses to new pathogens. Invasive plants may have robust defenses to species that are new but similar to native range pathogens that they coevolved with. For example, 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.

FIGURE CAPTIONS

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. [Author note: Figure is mock-up. Would be preferable to have seed piles instead of seed bags. And plants with clear buds/seed heads on resistant and tolerant plants but not on susceptible.]

Figure 2. Invasive plants colonize natural areas but also inhabit disturbed areas such as roadsides and fallow fields. Agricultural weeds can spread to roadsides and also be invasive in natural areas. 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 and then spillover to agricultural weeds, and then spread to invasive and native plants. [Author note: Figure is mock-up. Please add plant diversity to natural area and make crop into actual corn or wheat. There could also be more than one type of invasive/weed, for example, different plants in crop versus natural but then one plant that spans the two habitats.]

Figure 1

Figure. Compensation for infection in tolerant invasive plants may lead to seed production nearing that of healthy plants, or in this case a hypothetical resistant invasive plant. Tolerance may also be observed at the population level via compensation by plants with lower disease severity. While some diseases cause little loss in crop yield, they may be economically harmful due to loss of quality.

Figure 2

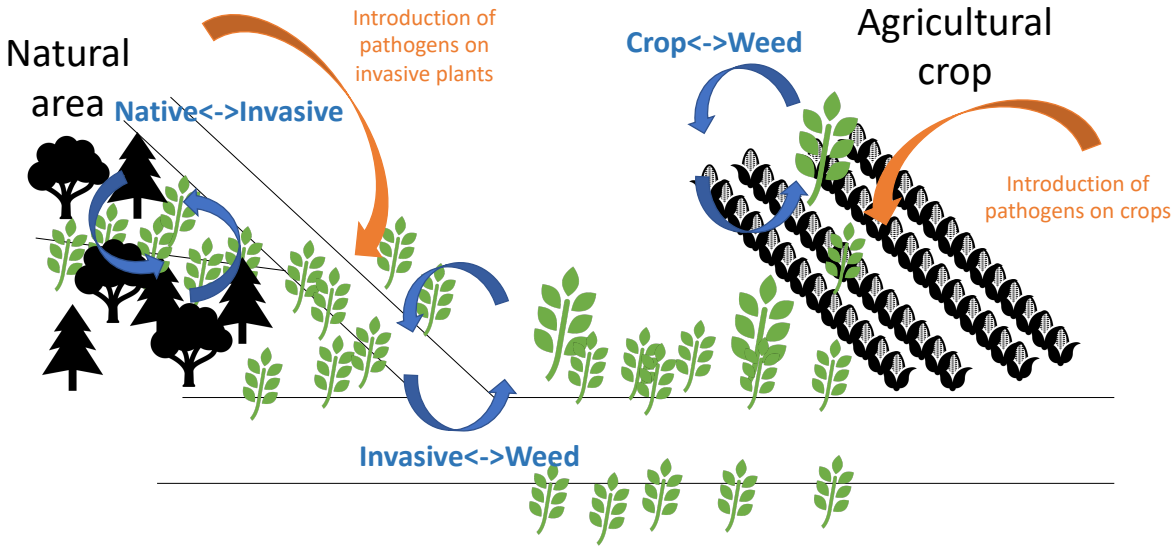
Natural area
Native<->Invasive
Introduction of pathogens on invasive plants
Crop<->Weed
Agricultural crop
Introduction of pathogens on crops
Invasive<->Weed

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 due to multiple introductions	Genetic variation in disease resistance; ability to evolve in response to selection from pathogens	Reduced by widespread use of modern varieties; no evolution in response to pathogen selection
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 by disturbance pre-invasion	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	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