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THE EVOLUTIONARY ECOLOGY OF NOVEL PLANT-PATHOGEN INTERACTIONS

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■ **Abstract** Novel plant-pathogen combinations occur whenever pathogen or plant species are introduced to regions outside their native range. Whether a pathogen is able to acquire a new host depends on the genetic compatibility between the two, through either preadaptation of the pathogen or subsequent evolutionary change. The ecological outcome of the novel interaction—for example, a spreading disease epidemic or the extinction of an incipient plant invasion—depends on the life history of the pathogen, opportunities for rapid evolution of virulence or resistance, and the presence of a suitable environment. We review recent work on the biology of pathogen virulence and host resistance, their mechanisms, and their costs. We then explore factors influencing the ecological and evolutionary dynamics of novel plant-pathogen interactions, using that evolutionary ecology framework to provide insight into three important practical applications: emerging diseases, biological invasions, and biological control.

INTRODUCTION

Advances in transportation technology and expanding global trade have greatly accelerated the rate of biological invasions (Perrings et al. 2002), which has in turn driven an increase in the number of novel encounters between plants and pathogens. In the past century, plant pathogens introduced from distant continents have caused the large-scale transformation of native ecosystems around the world, either by attacking a broad range of host species and altering the landscape through diffuse impacts on many plant species [e.g., *Phytophthora ramorum* in California oak woodlands (Rizzo & Garbelotto 2003)] or by dramatically reducing the populations of single species that played crucial roles in ecosystem structure or function [e.g., *Cryphonectria parastica* in North America and Europe (Anagnostakis 1987)]. At the same time, invasions by weedy plant species are ever more common, and plant pathogens native to their new ranges may play a role in regulating their spread (Mack 1996, Duncan & Williams 2002, Beckstead & Parker 2003). Conversely, under the assumption that escape from native pathogens may

help explain invasion success (Blaney & Kotanen 2001, Mitchell & Power 2003), pathogens are sometimes introduced to control weedy invaders (Charudattan & Dinoor 2000). Whether a novel pathogen-plant combination will lead to disease development and major outbreaks is of key concern for understanding the consequences of novel epidemics in conservation biology, for the control of invasive weeds and the safe introduction of biological control agents, and for the development of robust policy for international trade that protects plant and ecosystem health. Outcomes and dynamics of the interactions between hosts and pathogens are shaped by a complex set of ecological and evolutionary influences (Alexander et al. 1996, Simms 1996). Here we review the biological context, theory, empirical studies, and applications of the evolutionary ecology of novel plant-pathogen interactions following from the introduction of plants and/or pathogens into new environments.

We examine the initial interactions and potential for rapid evolutionary changes in novel plant-pathogen interactions for four scenarios, depending on whether the plant, pathogen, or both are novel to the environment (Table 1). Native plant-pathogen interactions (Table 1A) provide a reference for "normal" ecological and coevolutionary dynamics, with fluctuating selection on both hosts and pathogens. Costs of resistance genes should maintain variation in wild populations of the host (Roy & Kirchner 2000), and costs of virulence genes, as well as tradeoffs between transmission and damage to the host, should maintain variation for virulence in populations of the pathogen (Jarosz & Davelos 1995, Jenner et al. 2002). Because coevolutionary interactions are temporally dynamic, pathogens and hosts at a site may or may not be closely coadapted (Kaltz & Shykoff 1998).

Newly introduced plant pathogens (Table 1B) face both a novel environment and novel host species. The first hurdle for these microbes is to arrive in the new range by exploiting introduction pathways (Goodell et al. 2000, Brown & Hovmøller 2002), but environmental conditions and host availability also place strong limits on survival and spread (Aylor 2003). Pathogens must first be able to survive the abiotic conditions of the new location, including extremes of temperature, moisture, and UV irradiation (Gilbert 2002). Those that survive must encounter and infect suitable susceptible hosts, then reproduce and disperse. Each of these factors presents a strong selection filter, which may lead to rapid adaptation to new abiotic conditions and rapid evolution of novel host use. Particulars of the pathogen's life

TABLE 1 Four scenarios for the emergence of novel plant-pathogen interactions

	Pathogen	
Plant	**Stay**	**Move**
Stay	Native [A]	Novel Epidemic [B]
Move	Invasive Plant [C]	Biological control [D]

history and degree of host specialization are important determinants of the invasion process.

Newly introduced plant species (Table 1C) face a similar set of challenges, including surviving, growing, and reproducing in the new habitat. Tolerance of local abiotic conditions is most likely the dominant factor influencing initial survival of most species. However, if the naive host plant is highly susceptible to locally abundant plant pathogens, these pathogens could play an important role in limiting incipient invasions (Mack 1996) or preventing cultivation of agronomic species (Coutinho et al. 1998, Wingfield et al. 2001). Local pathogens have the potential to be particularly damaging if they are generalists or preadapted to the new host because they would already be well suited to local environmental conditions and would have a ready source of inoculum on their native host (Watson 1970). However, until an invading plant becomes dominant in the new habitat, there should be only weak selection on local pathogens to increase their ability to infect the new species. Therefore, rapid evolution probably plays only a small role for the pathogen. In contrast, introduced plants, if susceptible to local generalist pathogens, should experience strong selection pressure to develop resistance mechanisms. If not susceptible to local generalist pathogens, introduced plants may experience a demographic release associated with escape from their pathogens from the home range (Blaney & Kotanen 2001, Beckstead & Parker 2003, Mitchell & Power 2003).

Finally, both host and pathogen may find themselves in a novel environment (Table 1D). The two players may be introduced independently to a new area, or the pathogen may hitchhike on the introduced host, or the pathogen may be a biological control agent brought from the native range to control an invasive plant. Because both the pathogen and host are experiencing new conditions, there may be selection on both to adapt to the local environment. Although conditions in the new range may be less optimal for disease development, in some cases the reverse may also occur; a pathogen considered insignificant in its native range has sometimes caused severe outbreaks on the same host under new climatic conditions (Zwolinski et al. 1990, Wingfield et al. 2001). If resistance is costly, there may be initial selection on the invading plant against resistance to pathogens left behind, but then strong selection for resistance after the interaction is re-established.

Our goal is to present a synthetic context for explaining and predicting patterns in novel plant-pathogen interactions. Therefore, we begin by reviewing the biology of plant-pathogen interactions relevant to understanding ecological and evolutionary host shifts and dynamics. We then explore in greater depth predictions for the initial interactions, subsequent changes, and final outcomes of the four scenarios presented in Table 1. Throughout, we consider both numerical (ecological) dynamics and rapid evolutionary dynamics. Finally, we consider fundamental questions underlying three practical applications of scenarios B, C, and D of Table 1: unintended novel epidemics, introduced and invasive plants, and pathogens introduced as classical biological control agents for invasive plants.

CONCEPTS AND MECHANISMS OF DISEASE INTERACTIONS

Definitions

Terminology used in the plant pathology literature has a conflicted history, and meanings may differ from those in studies of animal diseases. In this review we follow the conventions adopted by the American Phytopathological Society (D'Arcy et al. 2001). Pathogenicity is the ability of a pathogen to cause disease on a particular host—it is a qualitative term. Virulence is then a quantitative measure of pathogenicity denoting the degree of damage caused on that host, usually assumed to correlate negatively with host fitness. Aggressiveness (as used by Vanderplank 1968) is then a synonym of virulence. Virulence is not necessarily correlated with factors that determine pathogen fitness, such as the efficiency with which a pathogen uses the host plant as a substrate or its ability to colonize new plants (Statler & Jones 1981, Welz & Leonard 1994). Mechanistically, the ability of many plant pathogens to cause disease on a particular host is regulated at least in part by gene-for-gene interactions between resistance (*R*) genes in the host and specific avirulence (*Avr*) genes in the pathogen (Flor 1956). In that context, *Avr* genes code for products (effectors) recognized by the corresponding *R* genes and lead to an incompatible interaction (no disease development); pathogens lacking those *Avr* genes (i.e., genotype *avr*) are termed virulent on that host. The combination of *Avr* genes in a pathogen strain determines the race of the pathogen. When we use the terms resistance and virulence in the context of gene-for-gene interactions, we hope to avoid confusion by indicating the special use.

We use the term host shift to refer to the acquisition of a new host, which may or may not involve evolutionary change in the pathogen. When their implications differ, we distinguish between evolutionary host shifts and ecological host shifts, which are often referred to in the biological control literature as host switches.

The Disease Triangle

A central principle of plant pathology is the disease triangle. That is, development of plant disease requires the junction of three equally important components: (*a*) a susceptible host, (*b*) a virulent pathogen, and (*c*) suitable environmental conditions. Even if a virulent pathogen is introduced into an environment with a genetically susceptible host, suboptimal moisture, temperature, or soil conditions may prevent disease development (e.g., Weste & Marks 1987). The environment can also affect the rate of reproduction of a pathogen (Garbelotto et al. 2003), or determine whether it can reproduce sexually (Adams & Line 1984). For novel plant-pathogen interactions, changes in pathogen virulence, host resistance, and pathogen tolerance of environmental conditions can dramatically affect disease impacts and evolutionary dynamics.

Host-Pathogen Recognition and Infection

Agricultural disease management relies primarily on deploying cultivars with *R* genes that provide resistance to pathogens with corresponding *Avr* genes. Such gene-for-gene interactions determine specificity for particular cultivars (genotypes) or species of crop plants and are important in many wild plant-pathogen interactions (Burdon 1991, Holub 2001, Takabayashi et al. 2002, Thrall & Burdon 2003). Characterization of numerous *R* and *Avr* genes shows that most *R* genes encode proteins thought to act as receptors that recognize specific *Avr* gene products in the pathogen (reviews in Leach & White 1996, Laugé & De Wit 1998, Martin et al. 2003, Nimchuk et al. 2003). Such recognition genes are diverse and common in plants—as much as 1% of the total genome (Ellis et al. 2000, Mondragón-Palomino et al. 2002, Nimchuk et al. 2003). Unlike plant *R* genes, the primary function of avirulence effectors in pathogens is not (needless to say) to trigger defense responses in plants. *Avr* gene products include a diversity of extracellular proteins, as well as viral coat proteins (Laugé & De Wit 1998). The recognition of an *Avr* gene product by the corresponding *R* gene product elicits plant defense mechanisms that produce an incompatible reaction (Jia et al. 2000), often through production of programmed host cell death called a hypersensitive response. Modification of an *Avr* (avirulent) allele to an *avr* (virulent) allele allows the pathogen to defeat host resistance by preventing recognition by the host.

Cost of Overcoming *R* Genes

Of particular interest for novel plant-pathogen interactions is genetic variation for virulence and resistance. One mechanism thought to maintain polymorphisms in *Avr* genes and *R* genes in natural populations is the cost of virulence (in the gene-for-gene sense) and resistance. Because recognition by the host is detrimental to the pathogen, we would expect avirulent *Avr* alleles to be quickly lost through selection, unless they have important functions that contribute to other aspects of pathogen fitness (Simms 1996). In the absence of a cost, pathogens would be expected to accumulate and retain the ability to circumvent many different host resistance genotypes. However, as demonstrated in early survey work, pathogen populations are often dominated by races that carry no *avr* alleles beyond those for the corresponding *R* genes present in the crop cultivar from which they were isolated (termed "unnecessary *avr* alleles") (reviewed in Vanderplank 1968, but see Parlevliet 1981 for counter examples). In addition, time series show a loss of *avr* (virulent) alleles in pathogens 6–10 years after a crop line with the corresponding *R* genes is removed from cultivation (e.g., Grant & Archer 1983). Similar loss might be expected in introduced pathogens after they leave their prior hosts behind. Vanderplank (1968) argued that such observations and *R* gene removal studies implied a fitness cost to the pathogen of carrying unnecessary *avr* alleles. However, such studies cannot rule out the possibility that the original *avr* mutation occurred in a pathogen lineage that was otherwise less fit; under this scenario, without the benefit associated with the presence of the corresponding *R* gene, this lineage would

then decline (Parlevliet 1981). As a more direct test of the cost of carrying extra *avr* alleles, known mixtures of pathogen spores with different virulence phenotypes have been passed through susceptible hosts. In some studies, races with more *avr* diversity were rapidly selected against in susceptible hosts (Watson & Singh 1952, Leonard 1969, Chin & Wolfe 1984, Thrall & Burdon 2003), whereas others showed no effect or the opposite pattern (review in Vanderplank 1968, Parlevliet 1981). Recent molecular genetic studies on pathogens have begun to elucidate some important functions performed by *Avr* gene products (review in Laugé & De Wit 1998). For instance, all *Phytophthora* species produce extracellular 98–amino acid proteins (elicitins) that are important in sterol scavenging, and they also induce a range of defense responses in plants (reviewed in Tyler 2002). Other *Avr* gene products play key roles in inhibiting the hypersensitive response (Abramovitch et al. 2003), promoting growth or reproduction within the host plant (Leach & White 1996), acting as plant toxins (Wevelsiep et al. 1993), or masking other *Avr* genes (Tsiamis et al. 2000). In plant breeding, crop resistance is more durable when the targeted *R* genes recognize pathogen effectors with particularly important housekeeping functions because changes to these genes inflict a large fitness cost on the pathogen (Vera Cruz et al. 2000).

Cost of Resistance

Polymorphism in resistance within and among host populations affects the dynamics of novel interactions. Pathogens can impose strong selection for particular *R* alleles in a host population. Unless there is a cost to maintaining resistance alleles in the absence of corresponding pathogen races, plant lineages should accumulate *R* alleles and become universally resistant. Although the many examples of polymorphisms for resistance in plant populations imply a significant cost to resistance (e.g., Parker 1988, Burdon 1991), the effects of genetic background, environment, and genetic linkage have made measuring a cost of resistance to plant pathogens difficult and controversial (Parker 1990, Schmid 1994, Brown 2002). In a review of the literature, Bergelson & Purrington (1996) found that 56% of 55 comparisons of resistant and susceptible host genotypes showed significant costs of resistance. For 11 pathogen species and 7 host species, they found the mean cost to fitness to be about 4% in the absence of disease. Tian et al. (2003), using isogenic pairs of transgenic host lines, estimated a 9% cost of specific *R* genes.

The reasons for such a cost of resistance are unclear because there is little evidence that *R* genes have important pleiotropic functions outside of recognizing corresponding pathogens. No doubt there is a significant energetic and fitness cost to mounting a defense response (Smedegaard-Petersen & Stølen 1981), but in the absence of elicitors from the specific pathogens, why should *R* genes be costly? Basal levels of expression or accidental induction by environmental factors or conserved *Avr* gene products from nonpathogenic microbes may be responsible for the costs (Tyler 2002, Brown 2003). There are numerous examples of *R* gene products recognizing multiple elicitors (Parker et al. 1991, Tyler 2002). Individual *R* genes can recognize different *Avr* gene products in a single pathogen species

[*RPM1*, (Grant et al. 1995)], recognize *Avr* gene products from different, closely related species [*RPW8*, (Xiao et al. 2001)], or even provide resistance to two pathogens as different as a bacterium and an Oomycete [*NPR1*, (Cao et al. 1998)]. Plants must face tradeoffs between being too selective—and potentially incurring costs from disease when pathogens are not detected—and the costs of unnecessarily expressing defense responses when induced by nonpathogens. These tradeoffs may be crucial in determining preadaptation in novel plant-pathogen encounters.

Interestingly, costs of resistance (as well as virulence) may decline over time, as modifier genes are selected to mask the disadvantageous traits that are genetically correlated with the virulence or resistance factor. In bacteria, this compensatory evolution has been shown to influence the maintenance of antibiotic resistance after removal of the antibiotic (Levin et al. 2000) and maintenance of resistance to viral infection (Lenski 1988).

Quantitative Resistance and Virulence/Aggressiveness

Whereas *R* genes and *Avr* genes determine the compatibility of a plant-pathogen interaction and regulate host shifts, numerous quantitative resistance and virulence factors determine the amount of damage a pathogen causes on a compatible host. Such genes are also likely to be extremely important in the evolutionary ecology of novel interactions. Pathogens produce toxins against host cells (Wolpert et al. 2002) and enzymes to detoxify host defenses (George & VanEtten 2001) as well as many factors important in colonization (e.g., Saile et al. 1997). Such factors have quantitative effects on disease development, disease severity, and pathogen fitness, and although they are not involved in gene-for-gene associations between plant and pathogen, they are under similar selection for rapid coevolutionary adaptation (Bishop et al. 2000).

Similarly, quantitative traits in plants are involved in many defense-related responses (Holub 1997), including the production of chitinases and endoglucanases that degrade fungal cell walls (Bishop et al. 2000), phytoalexins involved in the hypersensitive response (Bennett et al. 1994), enzymes that detoxify pathogen toxins (George & VanEtten 2001), and morphological traits conferring resistance (Bradley et al. 2003). Although all plant-pathogen interactions are likely to involve defense strategies that combine qualitative and quantitative resistance mechanisms (e.g., Bevan et al. 1993), the specific genetic basis of resistance can vary among populations of a single host species (Parker 1988, 1991a). Plants may also respond to selection from a pathogen by evolving tolerance, that is, the ability to maintain fitness at high levels of pathogen infection (Roy & Kirchner 2000). Tolerance evolves differently from resistance and may influence selection on resistance (Roy & Kirchner 2000, Mauricio 2001).

Transmission and the Evolution of Virulence

Until the emergence of Darwinian medicine in the early 1990s (Ewald 1993), there was a widely held view that host-pathogen interactions should evolve

toward a lower level of virulence because causing harm to the host should ultimately prove destructive to the pathogen as well (e.g., Alexander 1981). Therefore, novel, emergent epidemics were thought to be caused by maladapted pathogens whose impacts should decrease over time. Models of the evolution of virulence in animal pathogens, such as classic work by Anderson & May (1982), relied on the assumption that the evolution of increased virulence would be limited by opportunities for transmission to a new susceptible host. Although virulence should decrease if that enhances the probability of vertical transmission (Kover & Clay 1998), evolutionary theory suggests that complex tradeoffs between transmission modes, reproductive rates, and other life history traits determine whether natural selection should lead to increased or decreased virulence (Williams & Hesse 1991, Ewald 1993, Bull 1994, Lenski & May 1994). Caution should also be used in applying the predictions of the classic, animal-based models too strictly in the case of novel plant pathogens, as several assumptions of the models may not hold for plant diseases (Jarosz & Davelos 1995). In fact, in a review of the evidence using a series of different pathogen types (local versus systemic, soil-borne, foliar, floral, etc.), Jarosz & Davelos (1995) found little support for trends toward decreased virulence in natural plant-pathogen systems.

One key to predicting the trajectory of virulence in novel host-pathogen interactions is the relationship between virulence and pathogen fitness. Within a host, high virulence may be a necessary consequence of having higher pathogen titer (Chang et al. 1995), or it may directly increase pathogen fecundity (Fox & Williams 1984). However, several studies indicate that the fecundity of pathogens is sometimes uncorrelated, or even negatively correlated, with virulence (Johnson 1947, Imhoff et al. 1982, Robert et al. 2002, Zhan et al. 2002). Within-host fitness must in turn be linked to transmission, or between-host fitness (Bull 1994). Greater virulence may increase horizontal transmission if higher fecundity also means greater spore dispersal (Fox & Williams 1984). In contrast, serial passage studies of RNA viruses have found strong tradeoffs between virulence within the host (here tightly linked with pathogen fitness) and transmission by either insect or fungal vectors (Tamada & Kusume 1991, Hernandez et al. 1996). In one of the few empirical demonstrations of reduction of virulence in field populations, Escriu et al. (2000, 2003) investigated cucumber mosaic virus (CMV) and the replacement of virulent (necrosis causing) satellite RNA (satRNA) by nonvirulent satRNA. The more virulent satRNA replaced nonvirulent satRNA in mixtures, showing higher within-host fitness. However, virulent satRNA led to a depression in the accumulation of CMV, which led to a reduction in aphid transmission of the virus. This case demonstrates a tradeoff between virulence and transmission, suggesting that a pathogen may experience fitness tradeoffs associated with being highly virulent on a host. On the other hand, Zhan et al. (2002) used DNA fingerprinting to track the relative fitness of 10 strains of the fungus *Mycosphaerella graminicola* in the field and found no significant correlation between fitness and virulence. The relationship between fitness, virulence, and transmission appears to be complex.

PREDICTIONS FOR NOVEL PLANT-PATHOGEN INTERACTIONS

Conservation biologists, land managers, trade officials, and policy makers would like to be able to predict the trajectory of novel plant-pathogen interactions. The course of novel interactions is determined primarily by (*a*) the likelihood of an initial host shift, (*b*) the expected numerical dynamics of both the host and the pathogen, (*c*) the probability of evolution of resistance in the host, and (*d*) the probability of evolution of virulence in the pathogen. Here we examine how the factors described above may influence each of these steps.

What Is the Likelihood of an Initial Host Shift?

Estimating the probability that a pathogen will successfully shift to infect a novel host is a major challenge. Because most plants do not act as hosts for most plant pathogens, we expect that most encounters between novel combinations of plant and pathogen species never result in a compatible, disease-causing interaction. But host shifts do happen, and the likelihood of a shift occurring depends on the particular pathogen, host, and environmental conditions. Host shifts may be purely ecological, when a pathogen is preadapted to attack a newly encountered host species (Anagnostakis 1987). Alternatively, host shifts may involve an evolutionary change to permit infection of a host. For example, plant pathogens have acquired new hosts through hybridization (Brasier 2001) or by adapting to environmental conditions that allow access to new hosts (McDonald & Hoff 2001). Some have argued that ecological host shifts play the dominant role in novel host-pathogen interactions (Schrag & Wiener 1995, Altizer et al. 2003), although this area needs further study (Secord & Kareiva 1996).

Four factors are particularly important in determining the chance that a host shift occurs: (*a*) the degree of dependence of the pathogen on live hosts (i.e., pathogen survival and saprotrophic abilities), (*b*) the degree of specialization of the pathogen, (*c*) the phylogenetic distance between the novel potential host and hosts with which the pathogen is familiar, and (*d*) the degree of ecological association between the pathogen and the potential host.

SURVIVAL/SAPROTROPHIC ABILITIES OF PATHOGEN The life history of the pathogen is an important consideration in predicting which pathogens are most likely to infect invading plants or invade themselves. Some pathogens are obligate biotrophs, which require a living host to complete their life cycle (e.g., rusts, smuts, and powdery mildews). However, many pathogens are facultative saprobes and can persist, grow, and sporulate on dead tissue. Most published work on plant-pathogen interactions in natural systems excludes this important factor because researchers have chosen to focus on tightly linked, two-species interactions that are exclusively biotrophic (e.g., Burdon 1991, Alexander et al. 1996, Roy 2001). However, simply because modes of long-distance transportation should give an advantage

to pathogens that survive well without a living host (as resting spores or as saprotrophs), one would expect that many successful invasive microbes are facultative saprobes. Long-lived resting stages or saprotrophic abilities in the new habitat should increase the opportunities for and likelihood of host shifts.

DEGREE OF SPECIALIZATION OF PATHOGEN Some pathogen species are highly specialized on one or a few closely related host species, and individual genotypes (races) may be even more specialized on particular host genotypes. When such highly specialized pathogens are introduced to new regions (Table 1B), they are unlikely to survive the initial introduction unless their host is broadly distributed across both the native and introduced range. Biological control pathogens (Table 1D) are highly specialized by design, implying a low probability of host shift. Interestingly, some have argued that high host specificity may be correlated with evolutionary lability (Brooks & McLennan 1993), an idea that raises concerns about the long-term environmental safety of biological control agents (Secord & Kareiva 1996). For a newly introduced plant (Table 1C), host-generalist pathogens should be more likely to colonize and have the largest negative effect. For instance, the idea of biotic resistance to invasion requires that invaders are repelled by aggressive local pathogens (Elton 1958, Mack 1996, Blaney & Kotanen 2001); this idea relies on the ability of pathogens with broad host ranges to attack novel, naive hosts. In a comparison of sympatric suites of 18 native and non-native clovers, we found no difference in pathogen diversity, infection, leaf damage, or fitness effects of foliar and damping-off fungi (I.M. Parker & G.S. Gilbert, unpublished data). Fungicide experiments revealed significant effects of pathogens on plants in the field, but no difference between native and non-native species. In this system, host-generalist fungi dominate the relationship between plants and their pathogens, leveling the playing field between native and non-native hosts.

The life history of the pathogen plays an important role in its host range. We examined published records of host distribution for necrotrophs (pathogens that kill host tissue and live off the dead material) and biotrophic rusts and smuts (Farr et al. 2004) and found that species in all three groups can attack dozens or hundreds of plant species. However, rusts and smuts are generally limited to hosts from just one order or family (two, in the case of macrocyclic heteroecious rusts), whereas necrotrophs like *Alternaria alternata* or *Verticillium dahliae* have been found attacking plants from 29 or more plant orders (G.S. Gilbert & I.M. Parker, unpublished data). These broad patterns are intriguing, but a more detailed understanding of patterns of association between fungi and hosts is currently limited by unequal effort in different taxonomic groups and a lack of experimental cross-inoculations.

The degree of specialization affects not only whether the pathogen and host interact at all, but also how tightly the numerical dynamics of the pathogen are linked to the host. Invading pathogens that have a broad host range are less dependent on stochastic events, such as dispersing to the right host at the right time. They also have the ability to build up inoculum on a common host and then disperse onto

a second host in high numbers, increasing the chances that a pathogen genotype adapted to that second host may successfully colonize it.

PHYLOGENETIC DISTANCE AMONG HOSTS Along the gradient from highly specialized to highly generalized pathogens, host ranges are not a random selection of taxa. Host ranges often have predictable phylogenetic structure, with closest relatives being more likely to share pathogens. For example, of the 70 recorded fungal species from the common California coastal woodland tree *Quercus agrifolia* (Farr et al. 2004), 51% were recorded only on *Q. agrifolia*, and an additional 13% were restricted to the genus *Quercus*. Eighty percent were restricted just to the Fagales. This suggests that the success of an invading pathogen depends in part on the phylogenetic distance between its host(s) in the native range and available potential hosts in the new range. Similarly, whether local pathogens are capable of attacking a new introduced host depends in part on how closely related this plant species is to the resident native species. Mack (1996) tested this idea by looking at patterns of invasiveness in several floras, finding that invaders were more likely to be in families or genera that contain no native species, and attributed this to pressure from native pests and pathogens. However, taking a similar approach but controlling for introduction opportunities, Duncan & Williams (2002) found that introduced species in genera that already had resident natives were more likely, not less likely, to naturalize successfully in New Zealand. To understand how phylogeny may influence patterns of host specialization and host shift, we need detailed studies using experimental cross-inoculations among hosts. Such linkages of ecological and phylogenetic information would also help in assessing risk in phytosanitary policy decisions and biological control cases.

ECOLOGICAL ASSOCIATION Upon careful inspection of the phylogenetic patterns of host use in pathogens, one notices that despite marked structure, seemingly unpredictable host shifts also occur onto widely divergent taxa (Weste & Marks 1987, Eckenwalder & Heath 2001). Recently, researchers have emphasized the importance of ecological association as a driver of host shifts. For instance, Roy (2001) constructed phylogenies for flower-mimic rusts (genus *Puccinia*) and their hosts in the Brassicaceae. Major jumps occurred between distant clades, and overall there was no tight phylogenetic congruence between pathogen and host. Instead, patterns of host use showed strong geographic clustering, such that physical proximity appeared to play an important role in host shifts. Similarly, published records (Farr et al. 2004) demonstrate that despite the strong phylogenetic signal in host range for the fungi that attack *Quercus agrifolia* (Fagaceae), a number of fungi are shared with the phylogenetically distant, but commonly co-occurring Ericaceae.

Factors that increase the likelihood of contact between current and potential hosts should also increase the probability of a host shift onto an introduced plant. In addition to geographic range overlap, other factors include ecological requirements, phenology, and environmental drivers such as fog-drip or the seasonality of rainfall, which could make disease development synchronous within regions and

asynchronous among regions (or habitats). Insect vectors that use multiple host species increase the ecological association of these species beyond that caused by physical distribution alone, thereby increasing the opportunity for a host shift. In the case of introduced pathogens (Table 1B), the geographic range of potential hosts relative to the main ports of entry for importation of microbes should have a large influence on whether host shift and invasion will occur.

In addition to factors that increase the probability that new hosts will be in spatial and temporal proximity to current hosts, density of the new host also plays an important role. As an invading plant (Table 1C) increases in density and becomes locally dominant, both ecological opportunity and selection pressure for host switching should also increase. The subsequent dynamics of the plant-pathogen interaction also depend critically on this increase in host density.

What Are the Expected Numerical Dynamics of Pathogen and Plant?

After the initial shift of a pathogen to utilize a novel host, a number of factors influence the epidemiology of the pathogen over time. Here we consider the potential roles of (*a*) pathogen response to host density and (*b*) genetic diversity in the host population.

DENSITY DEPENDENCE In most plant pathosystems that have been studied, disease development is dependent on host density (reviews in Burdon & Chilvers 1982, Gilbert 2002). Density-dependent disease development may arise from increased transmission rates mediated by decreasing distance between hosts, or indirectly through intraspecific competition effects on host vigor (reviewed in Gilbert 2002). The importance of density dependence is strongly influenced by the degree of host specialization of the pathogen; the dynamics of most strict host specialists are strongly tied to the density of their hosts, whereas populations of generalist pathogens are decoupled from the density of any single host. In more complex systems, pathogens with multiple hosts may respond to the joint population densities of several host species (Garbelotto et al. 2003), they may be influenced by competition between alternative host species (reviews in Alexander & Holt 1998, Gilbert 2002), or, for heteroecious rusts with two obligate alternate and competing hosts, they could show negative density-dependent patterns (Burdon & Chilvers 1982). For the sake of argument, however, here we follow the case of a single host and a single pathogen.

The case of density-dependent transmission has special implications for novel host-pathogen interactions because of the interplay between invasion dynamics and disease dynamics. For an invading pathogen (Table 1B) with density-dependent transmission, the relative density of different prospective native hosts influences both the probability that an epidemic will be initiated and the rate at which it spreads. For an invading plant (Table 1C), the rate of pathogen transmission should be low at the early stages of invasion, making epidemics unlikely for

density-dependent pathogens. As the invasion proceeds, the plant reaches higher densities; in the case of weedy, high-impact invaders, plant densities may be very high relative to the co-occurring native species. At these high host densities, a pathogen that gains the ability to infect the host is able to reach maximum transmission rates, and epidemics should proceed extremely rapidly. The same is true for introduced biological control agents (Table 1D), which by definition are usually released onto invasive plants that have already reached high density. In the case of intentionally introduced plant species, native pathogens have produced dramatic effects on plants important in agriculture or forestry (Coutinho et al. 1998, McDonald & Hoff 2001). We could find no explicit examples of widespread invasive weeds experiencing substantial attack by a native pathogen, which perhaps supports the contention that delayed, evolutionary host shifts are rare. However, some invaders have been known to grow exponentially to high densities and then crash mysteriously, and pathogens have often been suggested (although with little empirical support) as a possible mechanism (Simberloff & Gibbons 2003).

Clearly, we need more studies on the timing of host shifts relative to the density and spread of novel invasive hosts. If pathogens play a role in the precipitous crash of certain established introduced species, then investigators should make a concerted effort to identify these pathogens and understand their origin. That is, are these pathogens persisting at low numbers when hosts are rare and becoming more prevalent in a host-density-dependent fashion, or do they undergo host shifts only when plants reach high density? These questions are interesting not only where pathogens have caused population crashes, but for all introduced species. Studies should be initiated to track the accumulation of pathogens in relation to the timing of host arrival and host density. Note that highly virulent, generalist pathogens may *not* show density-dependent responses, and as discussed above, they may prevent introduced plants from spreading. Such events are extremely difficult to observe.

EFFECTS OF HOST VARIATION ON EPIDEMIOLOGY One of the most common generalizations in the field of plant-pathogen interactions is that disease should be lower in more genetically diverse host populations (Adams et al. 1971, Harlan 1976, Barrett 1988, Mundt 2002). Highly inbred agricultural species planted in large monocultures have been vulnerable to devastating disease epidemics; one response has been to plant multilines of different resistance genotypes together (Mundt 2002). Outside of agriculture, however, the evidence for a direct link between genetic variation and disease pressure has been mixed (Kranz 1990, Roy 1993, Thrall & Burdon 2000). Roy (1993) observed that correlations have been found primarily in systems with very low host genetic diversity—that is, just a handful of clones or selfed lines—whereas studies with slightly higher diversity tend not to show a relationship. In her study of three populations with a range of 6–27 genotypes, the population with lowest disease incidence was unexpectedly the one with lowest clone diversity and highest density. However, most such studies focus on snapshots in time, and temporal fluctuations in disease pressure could obscure the relationship between disease and host genetic diversity.

Introduced plants could provide an interesting context in which to test ideas about how genetic variation should influence disease incidence and dynamics. The introduction bottleneck should lead to reduced genetic variation, at least for Mendelian traits such as molecular markers (Barrett & Husband 1990, Amsellem et al. 2000, Lee 2002); *R* genes also fall into this category and should be genetically depauperate in invading plants. The analogous low diversity of *R* genes in crop plants leads breeders to return to wild crop progenitors in the habitat of origin to access a diversity of *R* genes (Hoisington et al. 1999). As an introduced species spreads, the colonization process results in different subsets of the original diversity in different sites. Pathogens, either native (Table 1C) or introduced (Table 1D), on these introduced hosts could provide a simplified system for looking at the ability of a pathogen to colonize and spread in populations of differing genetic diversity. Similarly, the impact of successful pathogens should be greater in populations that are less diverse. This idea has important implications for strategies in biological control. For example, researchers have long thought that sexually reproducing weeds are harder to control because their higher levels of genetic variation confer greater resistance (Burdon & Marshall 1981, but see Chaboudez & Sheppard 1995).

The best-studied case of classical biological control by a pathogen is that of the rust *Puccinia chondrillina* on *Chondrilla juncea* L. (rush skeletonweed, Asteraceae), a perennial weed of cereal crops native in Europe and introduced to Australia and North America (Burdon et al. 1981, Panetta & Dodd 1995). An interesting aspect of the *Chondrilla*/*Puccinia* system is the extreme level of host specificity found in the pathogen (Hasan 1972). *Chondrilla* is a triploid apomict in Australia, where only three clones are known and where the pathogen is also exclusively asexual. The initial control program introduced a pathogen isolate highly virulent to the dominant and most widespread clone (McVean 1966), which resulted in replacement by a different clone rather than elimination of the weed (Burdon et al. 1981, Chaboudez & Sheppard 1995). In light of the way host genetic diversity foiled the success of *Chondrilla* biological control in Australia, it is interesting that the level of disease pressure experienced by populations of *Chondrilla* is not strongly influenced by the degree of clonal diversity in its native range (Chaboudez & Sheppard 1995).

What Is the Probability of Evolution of Resistance in the Host?

In addition to host shifts and numerical dynamics, coevolutionary dynamics of the host and pathogen also influence the trajectories of novel plant-pathogen interactions. These coevolutionary dynamics involve both resistance of the host and virulence of the pathogen. For both players, the rate of evolution of a trait is proportional to (*a*) the strength of selection and (*b*) the amount of genetic variation for the trait (Fisher 1930, Crow 2002).

STRENGTH OF SELECTION If selection on the host by the pathogen is very weak, evolution of resistance will be imperceptibly slow or swamped by other factors. Different types of plant pathogens provide different strengths of selection (reviewed in Gilbert 2002). Pathogens that cause damping-off of seedlings, and many root rots, wilts, and canker diseases of mature plants can cause a high rate of mortality in host populations. Many smuts and some systemic pathogens can castrate hosts. In contrast, although most foliar diseases reduce host survival and reproduction, their effect may vary from insignificant to very strong.

For a native pathogen on a relative of an invading plant (Table 1C) that does overcome resistance in the invader, it is likely to sweep quickly through the susceptible host population and could provide strong selection pressure on the host to evolve resistance. For a native plant battling an introduced pathogen (Table 1B), evolving resistance to the new pathogen may come at a cost, either in terms of energy allocated to defense, or by modifying *R* genes that might otherwise be committed to conferring resistance to local native pathogens. Thus, plants may also experience fluctuating or stabilizing selection on resistance traits.

GENETIC VARIANCE FOR RESISTANCE Even if selection is strong, evolution will occur only if there is genetic variation in resistance for selection to act upon. The many examples of rapid evolution of herbicide tolerance in crop weeds (Heap 1997) suggest that there is often enough genetic variation to respond to strong selection. However, in contrast to herbicides, pathogens and herbivores themselves evolve in response to the host, which may exhaust genetic variation for resistance more rapidly. In several cases of novel forest epidemics, local variation for resistance has been quickly exhausted, and forest pathologists have resorted to bringing in resistance genes from distant regions or related species (Anagnostakis 1992, Smalley & Guries 1993). Mutation will, over long periods, be expected to generate new resistant forms; however, the great difference in generation times and population size between the pathogen and host puts the plant at a disadvantage (Schafer & Roelfs 1985, Hafner et al. 1994). In fact, if the impact of the pathogen on the host is extreme, then resistance may never evolve because the host is eliminated first. For example, the rapid sweep of the chestnut blight *Cryphonectria parasitica* through the distribution of the American chestnut eliminated every large adult and, in effect, keeps the species from reproducing (Anagnostakis 1987); therefore it is not clear if resistance will ever evolve in that system. In novel epidemics (Table 1B), we may expect to see resistance evolving in the host most readily when the pathogen's effect on the host is strong, but not exceedingly strong.

For an invading plant (Table 1C), we would predict that little specific resistance would be present in the population at the start, unless it happened to express an *R* gene receptor that recognized a corresponding *Avr* gene product in a local pathogen. Because individual *R* genes may code for resistance to multiple pathogens, the responses to different pathogens can be genetically correlated. If the same allele recognizes multiple pathogens, a positive correlation will result,

whereas if alternate alleles at the same locus recognize different pathogens, a negative correlation will result. Novel hosts therefore may be preadapted to resist a pathogen, or may rapidly lose resistance to previous pathogens when faced with selection by a new suite of pathogens. Such tradeoffs could make invaders more susceptible to introduced biological control pathogens (Table 1D).

What Is the Probability of Evolution of Virulence in the Pathogen?

As in the host, evolution of the pathogen will depend on genetic variance and the strength of selection. However, predicting how selection should act is not so straightforward in the case of pathogen virulence. As discussed above, early conventional wisdom suggested that virulence should start high on a naive host and evolve to a lower, intermediate level (reviewed in Bull 1994, Lenski & May 1994). The classic case of the myxamatosis virus introduced to control invasive rabbits in Australia (Fenner & Fantini 1999) suggests that invading host species might be the best place to look for examples of loss of virulence over time. However, currently we have few examples, or even indirect evidence, of reduction in virulence in plant pathogens through natural selection (Jarosz & Davelos 1995).

Key factors that influence the evolution of virulence in plant pathogens are (*a*) linkage between virulence and pathogen fitness, (*b*) availability of alternative hosts, and (c) ability to reproduce on dead plant material or produce long-term survival structures. Evolution of increased virulence will be limited if it decreases the probability of transmission to a new host either by reducing pathogen fecundity or by reducing access to suitable hosts. However, if a pathogen can reproduce abundantly on an alternative host on which it has low virulence, the pathogen may reach very high virulence on some hosts while maintaining population size on the sympatric host. This may be the case in the Sudden Oak Death epidemic, where *Phytophthora ramorum* reproduces prolifically on the reasonably tolerant *Arbutus menziesii* but is often lethal on *Lithocarpus* and *Quercus*, where it has low fecundity (Garbelotto et al. 2003, Rizzo & Garbelotto 2003). Similarly, if the pathogen is able to grow and reproduce as a facultative saprobe, or if it produces durable resting structures (e.g., oospores and chlamydospores produced by many species of *Phytophthora*), the pathogen may be able to wait for years to encounter a suitable host and favorable environmental conditions. Such saprotrophic/long-term survival strategies could allow evolution of very high virulence without a fitness cost to the pathogen.

What Constrains Evolution?

There are constraints on the degree of local adaptation that occurs in plant-pathogen interactions, which can affect pathogen virulence or host resistance and complicate associations between *R* genes and recessive alleles at *Avr* loci. Gene flow may swamp local adaptation, and gene flow in fungal pathogens has the potential to be very large, as many of these organisms travel long distances by wind dispersal (Fitt

et al. 1987, Brown & Hovmøller 2002, Aylor 2003). Studies that have looked for evidence of local adaptation in pathogens have provided a mixed picture. Using experimental inoculations of hosts collected over a range of scales from meters to kilometers apart, some studies have found that incidence or virulence of pathogens is higher on their original hosts (Parker 1985, 1991a), whereas others have found no significant "home-host advantage" (Parker 1989, Davelos et al. 1996, Zhan et al. 2002). This lack of consistent local adaptation may obscure our ability to validate predictions about the evolution of virulence (Zhan et al. 2002).

Because genotypes of hosts and pathogens should fluctuate asynchronously, host-pathogen coevolution is difficult to infer from a single sample in time (Dybdahl & Lively 1995, Kaltz & Shykoff 1998). In only a few cases do we have data on changing genetic structure of host and pathogen in natural systems, making it difficult to assess how often these change in a way consistent with local adaptation. So far, studies that have followed the dynamics of evolutionary changes in resistance have not provided clear evidence that these changes are easily predictable (Burdon & Jarosz 1992, Burdon & Thompson 1995, Parker 1991b). As populations of both pathogen and host are linked by gene flow, a metapopulation perspective may be necessary to make sense of long-term dynamics (Thompson 1994). What this implies for novel host-pathogen interactions is that, even if these interactions are governed by predictable evolutionary processes, it may be difficult to discern how selection operates from invasion studies unless they are conceived in a broad geographic framework.

The lack of observed local adaptation in pathogens also has important implications for the practice of classical biological control. Biocontrol strategies are increasingly employing genetic analysis in the careful matching of agent genotypes with the population of origin for the weed (e.g., Hasan et al. 1996, Holden & Mahlberg 1996). This practice is based on the assumption that pathogens from the population of origin are better adapted to exploit the invasive host, which may not always be true. For example, Yugoslavia is the putative site of origin for invasive *Chondrilla juncea* in the western United States (Hasan et al. 1996). *Puccinia chondrillina* genotypes collected from Yugoslavia showed high virulence on some U.S. genotypes, but other genotypes were little affected. In addition, some *P. chondrillina* isolates from other regions also showed high virulence. Our review of the evidence for local adaptation in natural plant pathosystems suggests that close genetic matching of hosts between the native and introduced range may not be an efficient biological control strategy.

CONCLUSIONS: UNRESOLVED APPLIED QUESTIONS

Ecological and coevolutionary interactions between pathogens and their plant hosts help shape the structure and dynamics of natural plant populations and communities. Novel plant-pathogen interactions are a consequence of long-distance movement of pathogens, plants, or both into regions outside of their historical

distributions. Most often, these movements are driven by global trade, transportation technology, and changing land-use patterns. In this review we have attempted to create an evolutionary ecology framework for thinking about novel plant-pathogen interactions that will be useful for understanding, predicting, and managing novel disease epidemics and invasions by introduced plants.

We have outlined the key factors affecting novel plant-pathogen interactions and offered predictions for how they should influence the three forms of novel interaction: novel epidemics, invasive hosts, and biological control (Table 1B,C,D). However, these predictions are conjecture based largely on application of principles learned from studies in agriculture or a few native wild systems, with few studies from wild novel interactions themselves. Currently underutilized, studies of novel epidemics and novel host introductions should be used to illuminate these unresolved questions about the evolutionary ecology of plant-pathogen interactions.

In this final section we focus on the practical side of the three forms of novel interactions, outlining some ways in which an evolutionary ecology perspective is necessary for improving policy and management.

Novel Epidemics

Novel disease epidemics are best controlled through prevention. Effective trade regulations, quarantine policy, and land-use planning are essential tools in the prevention of novel epidemics. An increasingly globalized economy ensures a continued increase in the movement of plants and other materials between otherwise biologically isolated regions. Changing land-use patterns, including increasingly fragmented landscapes, often with close juxtaposition of remaining wildlands with low-diversity agricultural and forestry systems, may increase opportunities for the development of novel epidemics. We suggest that the answers to several questions are key to effectively mitigating the effects of novel epidemics. (*a*) Can we design more effective quarantine procedures by understanding which pathogen life history traits are most likely to lead to epidemic development? (*b*) Are considerations of ecological adaptations to novel environmental conditions as important as pathological attributes? (*c*) In creating lists of pathogens for quarantine exclusion, what is the relative importance of preadaptation to particular hosts versus the likelihood of evolutionary host shifts with ecological or economic consequence? (*d*) Could an understanding of the phylogenetic structure of plant communities and the importance of host phylogeny to host shifts be incorporated into land-use planning, in order to minimize the probability of novel epidemics arising at the borders between agricultural and wildlands?

Invasive Hosts

Although most introduced plants do not invade, the few that do represent one of our most challenging environmental problems (D'Antonio & Vitousek 1992, Office of Technology Assessment 1993, Vitousek et al. 1996, Parker et al. 1999, Mack et al.

2000). The central questions for plant invasion biologists are why most plants fail to establish viable populations when introduced into a new habitat, and why a few become noxious invasive weeds. The Biotic Resistance Hypothesis suggests that in natural habitats, native pests colonize the naive exotic hosts and eliminate them before they can become established (Elton 1958, Simberloff 1986, Mack 1996, Duncan & Williams 2002). In contrast, the Natural Enemies Hypothesis proposes that successful invaders leave behind their regulating insect pests and pathogens (Darwin 1859, Crawley 1987, Blossey & Nötzold 1995, Maron & Vila 2001, Siemann & Rogers 2001, Keane & Crawley 2002, Beckstead & Parker 2003). Because empirical studies testing both of these central ideas are surprisingly limited, there are significant unresolved issues for those trying to understand, manage, and prevent plant invasions. In particular: (*a*) How often do native pathogens contribute to biotic resistance in reducing the success of potential invaders, and what does this imply for debates about the value of biodiversity for ecosystem invasibility? (*b*) Is genetic variation for pathogen resistance particularly low for invasive plants relative to native species? (*c*) If so, why do we not see more dramatic epidemics emerging on high-density, invasive plants? (*d*) Is "escape from natural enemies" a common phenomenon in introduced plants, and does it contribute significantly to invasiveness? (*e*) Can we expect aggressive plant invader populations to eventually accumulate enough pathogens to reduce their ecological impacts on the invaded ecosystem?

Biological Control

Biological control of invasive weeds is unusual in that disease epidemics are the desired outcome. The short-term and long-term success of particular biological control introductions depends on host and pathogen numerical dynamics and the way these dynamics are influenced by density, frequency of diseased hosts, and genetic variation. In particular, (*a*) What is the effect of novel environmental conditions on the dynamics of the host-pathogen interaction? (*b*) Is control more successful in genetically depauperate weeds? (*c*) Do transmission rate and demographic impact of the pathogen decline as the host population declines? (*d*) Do pathogen and host reach a stable equilibrium or do they depend on metapopulation dynamics to persist in the landscape?

When the host is genetically depauperate and the pathogen has been chosen for its virulence, the host should be at a relative disadvantage. It is then somewhat surprising that we do not see more cases of spectacularly successful biological control with pathogens. Evolutionary change after introduction may help explain the varied effectiveness of biological controls, but such changes are almost unexplored; we could find no studies that have tracked changes in pathogen virulence or host resistance for a biological control system outside of *Chondrilla juncea* (Chaboudez & Sheppard 1995, Hanley & Groves 2002). Modeling efforts suggest that virulence of the pathogen, and whether it influences host survival or fecundity, will affect the evolution of resistance in the weed population and the long-term effectiveness of biological control agents (Thrall & Burdon 2004).

Because of the implications for policy and risk assessment, we also need to understand the ecological and evolutionary factors influencing shifts to nontarget hosts. Specifically, we need to ask (*a*) Does virulence of biological control pathogens change over time, and has this increased or decreased the success of control? (*b*) Has the host developed resistance over time? And finally, (*c*) Has host specificity changed over time, and are the shifts predictable based on phylogenetic distance from known hosts?

Novel plant-pathogen interactions pose significant threats to natural and managed ecosystems. A better understanding of the evolutionary ecology of such interactions in different contexts may improve management options. Simultaneously, novel epidemics, invasive hosts, and biological control efforts provide numerous underexploited opportunities to increase our understanding of the basic biology of novel plant-pathogen interactions.

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