



Aerial Dispersal of Pathogens on the Global and Continental Scales and Its Impact on Plant Disease

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Some of the most striking and extreme consequences of rapid, long-distance aerial dispersal involve pathogens of crop plants. Long-distance dispersal of fungal spores by the wind can spread plant diseases across and even between continents and reestablish diseases in areas where host plants are seasonally absent. For such epidemics to occur, hosts that are susceptible to the same pathogen genotypes must be grown over wide areas, as is the case with many modern crops. The strongly stochastic nature of long-distance dispersal causes founder effects in pathogen populations, such that the genotypes that cause epidemics in new territories or on cultivars with previously effective resistance genes may be atypical. Similar but less extreme population dynamics may arise from long-distance aerial dispersal of other organisms, including plants, viruses, and fungal pathogens of humans.

Long-distance dispersal (LDD) in the air is an important survival strategy for many organisms, enabling them to colonize new territory rapidly or to migrate between summer and winter habitats (1). It is especially relevant for fungi pathogenic on crop plants, because wind dispersal of their spores for hundreds or thousands of kilometers has caused the spread of several important diseases on a continental or global scale and allows the regular reestablishment of diseases in regions where the climate is seasonally unfavorable (Fig. 1). For obligately biotrophic fungi, including those that cause rust, powdery mildew, and downy mildew diseases, the production of huge numbers of spores, which are wind dispersed from one susceptible host to another, is essential for reproduction and survival because the pathogens are completely dependent on living host tissue for survival (2). The need to control these diseases has been a major stimulus for recent research on the theory of LDD (3–8), and the fungi themselves illustrate some of the best characterized and most striking consequences of aerial LDD. Research on these organisms may help in understanding the less extreme population dynamics of other aerially dispersed organisms, including plants and pathogens of humans and other animals.

The world's agriculture depends largely on a small fraction of the many thousands of plant species grown worldwide, so intercon-

tinental dispersal of pathogens may cause diseases of crops on a global scale. As plants have been redistributed from their centers of origin, fungi have followed, resulting in devastating outbreaks of such diseases as potato late blight (*Phytophthora infestans*) (9) and chestnut blight (*Cryphonectria parasitica*) (10). In the past half-century, improved plant quarantine has restricted the movement of many pathogens, but it has not halted those that cause such destructive diseases as coffee leaf rust (*Hemileia vastatrix*) (11) or sugarcane rust (*Puccinia melanocephala*) (12) because the dispersal of airborne spores cannot be constrained.

The risk of global spread of disease is increased by the limited genetic diversity of many modern crops, as compared to that of related wild species or landrace crops. Extreme examples are coffee (13) and banana (14), of which single clones, propagated throughout the tropics, are susceptible to leaf rust and black Sigatoka disease [black leaf streak (*Mycosphaerella fijiensis*)], respectively. More generally, the international exchange of germplasm has been a feature of plant breeding since its origin in the late 19th century and has accelerated in recent decades, so several resistance genes are now used on a global scale. Pathogens able to overcome those resistances may cause disease wherever their spores are dispersed. An important class of genes for resistance to obligate biotrophs has specific “gene-for-gene” interactions with pathogen genotypes (15), such that just one mutation may cause a pathogen to become virulent (16) on a host with the matching resistance gene.

Here, we discuss the role of aerial dispersal over distances of 500 km or more in the spread of plant diseases. We consider new invasions and periodic reestablishment of dis-

eases and discuss the effects of limited host species and genetic diversity on disease spread, bringing together new ideas from epidemiology and genetics to discuss the relation between pathogen dispersal and adaptation to host crops. We also consider the implications of research on biotrophic fungal pathogens of plants for the study of other organisms.

Invasion of New Territory

Two forms of aerial dispersal spread diseases to places that are distant from the original source of inoculum. The more spectacular kind involves the transport of spores over very long distances, even between continents, in a single step, and the other is a gradual, though possibly rapid, expansion of the range of a pathogen population within a continent. Single-step invasions are rare, inherently unlikely, and therefore unpredictable, so they are usually thought to be unique events requiring special explanations. Range expansions, by contrast, are generally treated as part of the normal dispersal process of pathogens. Recent research, however, suggests that single-step invasions and range expansions may be caused by the same dispersal processes.

It is often difficult to determine the original cause of a single-step pathogen invasion, but in a few cases, there is good evidence that the inoculum was airborne rather than carried by people or on plants or plant products. Most of these involve rusts, which may be more likely than other diseases to be aerially dispersed over very long distances because their spores are comparatively robust against environmental damage (17). Detailed meteorological data indicate that sugarcane rust was almost certainly introduced into America (the Dominican Republic) from Cameroon in West Africa through the transport of *P. melanocephala* uredospores by cyclonic winds in early June 1978 (12) (Fig. 1, dispersal I). A general analysis of transatlantic winds showed that *H. vastatrix* uredospores may have been carried from Angola, where coffee leaf rust was first reported in 1966, to Bahia in Brazil in 1970 (11) (Fig. 1, dispersal II), although transport on diseased plants could not be excluded (18).

In a wider range of diseases, inoculum has been transported from one continent to another by means other than the wind, but the subsequent expansion of the pathogen's range

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through the new area has been by airborne spores. Intercontinental dispersal of black Sigatoka disease, which, in the past 40 years, has devastated banana and plantain crops worldwide (Fig. 1, area IX), probably occurred through the transport of infected plants, but thereafter, dispersal within continents has been by airborne ascospores (19). Potato late blight was introduced into Europe at least twice in infected tubers imported from Mexico (9). The original introduction was in the 1840s (20), when it destroyed crops in Ireland and elsewhere (Fig. 1, transport V), then in the 1970s, when the A2 mating type first appeared outside Mexico (21) (Fig. 1, transport VI). A disease invasion that was probably caused by spores carried on clothing occurred when wheat yellow rust [or stripe rust (*Puccinia striiformis* f. sp. *tritici*)] appeared in Australia in 1979, probably from a source in Europe (22) (Fig. 1, transport VII). It subsequently spread to New Zealand in 1980, probably by wind dispersal of uredospores from eastern Australia (23) (Fig. 1, dispersal III).

Population Dynamics of Rare Events

Single-step pathogen invasions are remarkable because they are rare. An emerging view is that unusual, extreme events may have greater influence on the large-scale distribution of populations than do typical or normal events (7). Biological invasions have usually been modeled by dispersal functions in which

the tail of the probability distribution of propagules is exponentially bounded, so that the rate of population expansion is constant (24). New insights into invasions, particularly those of plant pathogens, are coming from models involving non-exponentially bounded ("fat-tailed") dispersal, in which a relatively high proportion of propagules are dispersed over long distances and population expansion accelerates with increasing distance from the source (3, 5, 6). It is often assumed that when observed dispersal data cannot be modeled by a single, exponentially bounded function, as is often the case (5), there must be two or more dispersal processes at different length scales (24). Turbulent diffusion, however, may give rise to fat-tailed dispersal (3, 6, 8), so a single process may account for disease spread at all scales (5). Examples of accelerating range expansion include the spread of potato late blight through Europe and North America in the 1840s, which was more than three orders of magnitude faster than the expansion of a single focus (6), and radial expansion of wheat yellow rust in Europe, which was five orders of magnitude faster (on scales of hundreds of kilometers) than in primary foci (24).

Few spores are dispersed over very long distances, even when dispersal is fat-tailed, so rare dispersal events create secondary foci at unpredictable sites far from the source.

Patchy demography and population genetic structure may arise from leptokurtic exponentially bounded dispersal (25), but they are especially likely when dispersal is fat-tailed, because expanding primary foci may never catch up with secondary and subsequent foci (4). This is illustrated by *C. parasitica*, which was introduced to North America from east Asia, probably Japan (10) (Fig. 1, transport VIII), then wind dispersed by ascospores throughout the range of the American chestnut, *Castanea dentata*. There was no correlation between genetic distance and geographic distance of North American *C. parasitica* populations, which is consistent with stochastic LDD rather than steady expansion of an epidemic front (26).

LDD of pathogens can only occur if there is a susceptible host in the target area. A striking example of a genetically uniform, globally distributed crop is arabica coffee, as the original plantations in the Americas can be traced back to a single bush taken from Java in 1706 (13). Hence, a genotype of *H. vastatrix* capable of attacking any one bush could cause leaf rust in coffee plantations throughout the two continents. What is important here is not genetic uniformity of a general kind, but uniform susceptibility to the disease. In Australia, though many wheat cultivars were grown in 1979, the original epidemic of yellow rust was caused by a pathotype (27) of *P. striiformis* f. sp. *tritici* (22) to which few cultivars had useful resis-

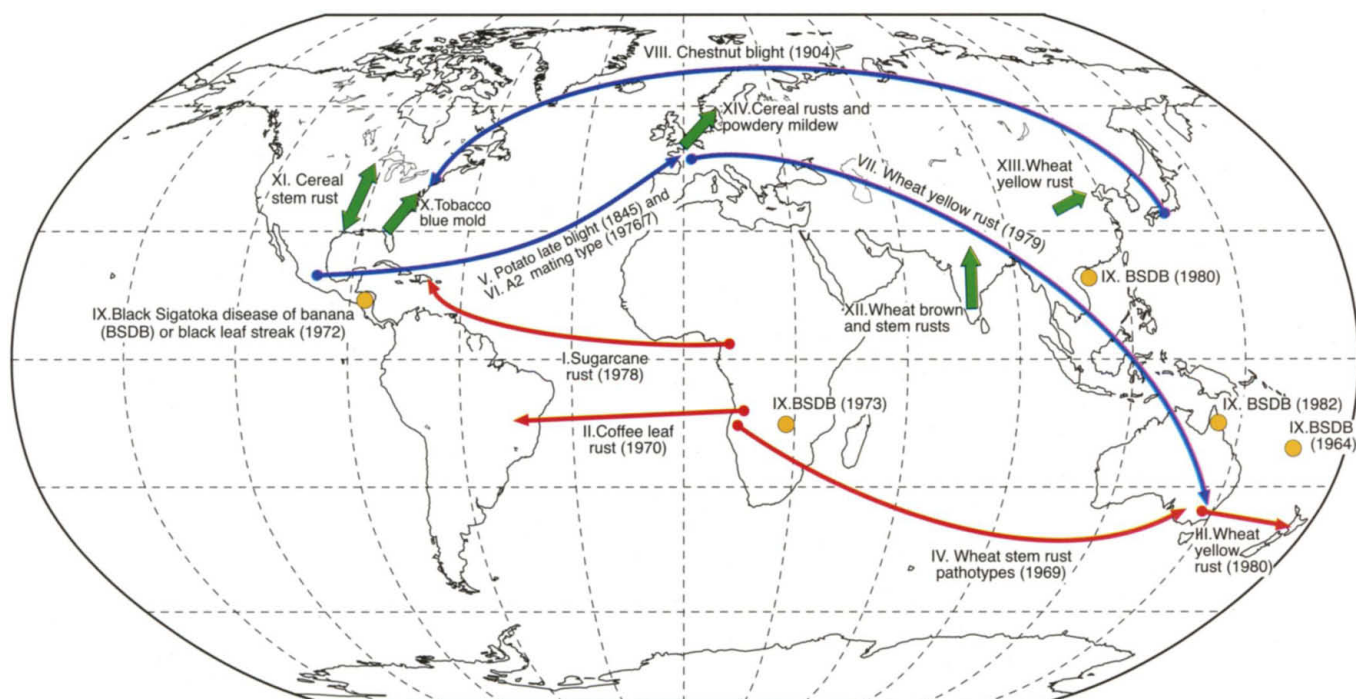


Fig. 1. Selected dispersal events of fungal pathogens. Red and blue arrows indicate invasions of new territories (first year recorded in brackets). Red arrows indicate dispersal that probably occurred by direct movement of airborne spores [I (12), II (11), III (23), and IV (52)]. Blue arrows indicate pathogens that were probably transported to the new territory in infected plant material or by people and spread thereafter as airborne spores [V (9),

VI (21), VII (22), and VIII (10)]. Orange circles indicate the worldwide spread of black Sigatoka disease of banana; the first outbreak recorded on each continent is marked [IX (19)]. Green arrows indicate periodic migrations of airborne spores in extinction-recolonization cycles [X (32), XI (33), XII (34), XIII (35–38), and XIV (41)]. [Background world map © C. Lukinbeal, Southern Connecticut State University, New Haven, Connecticut]

tance (23). In an important model of pandemics of plant diseases, the velocity of disease spread depended only to a limited extent on the density of the host population in the target area (28). The model used deterministic, exponentially bounded dispersal, so one might expect this conclusion to be even more strongly supported in models that involve stochastic or fat-tailed dispersal.

Establishment of a new population by LDD of very few individuals may cause an extreme form of genetic drift. If secondary populations remain distinct from their primary source (4, 25), they may contain only a small subset of the original variation, and this founder effect may persist, particularly in clonal organisms such as *P. striiformis* f. sp. *tritici* (23) or the pre-1976 global population of *Phytophthora infestans* (29). This difference was less marked in *M. fijiensis*, which had genetic diversity in dispersed populations in Africa, America, and the Pacific Islands that was 51% less than the genetic diversity in the source area of southeast Asia (30), or in *C. parasitica*, which had similar diversity in North America, Europe, and Japan (10), suggesting that several genotypes of these two fungi may have been dispersed between continents.

Fat-tailed dispersal is an interesting model for very long distance disease spread, because it might account for rare, apparently unpredictable events. As in other organisms, however, including plant seeds (31), critical data on plant pathogens are lacking on empirical dispersal functions within fields, let alone on the global or continental scales. This is a major challenge for both experimental and theoretical epidemiology.

Local Extinction and Recolonization

Obligate biotrophic fungi cannot survive on plant debris or in the soil between crops, so either they must survive in a dormant stage, usually the product of sex, or their populations must be reestablished from external sources when new host plants are available. Such extinction-recolonization cycles may involve dispersal on a scale of 500 to 2000 km. As a survival strategy, this is akin to the annual migration of birds and insects whose summer range is inhospitable in winter, or vice versa. For all of these organisms, the atmosphere is a pathway for rapid movement between their summer and winter ranges (1).

LDD has an important role in recoloniza-

tion, but the population dynamics tend to be more predictable than for invasions of new territory because larger numbers of spores are dispersed and the distances involved are generally shorter. Recolonization depends critically on the availability of susceptible host tissue, and thus the composition of pathogen populations differs substantially from year to year, sometimes causing host resistance to become ineffective.

Extinction-recolonization cycles occur for all three major groups of obligate biotrophs, but most of the well-studied cases involve rusts; however, a notable exception is the downy mildew *Peronospora tabacina* (tobac-

wheat plants during summer (Fig. 2). Data on disease distribution (35), environmental conditions (36), winds (37), and fungal pathotypes (38) indicate that the principal source area of the recolonizing populations includes the southern Gansu and northern Sichuan provinces, where the pathogen survives year-round (Fig. 2, solid blue arrows). Recently, additional pathways of interprovincial dispersal have been proposed (Fig. 2, dashed arrows) (39). With a multicopy restriction fragment length polymorphism system, 97 DNA fingerprints were found in 160 isolates, 7 of them in more than one province (40). The distribution of six of these fingerprints was consistent with southern Gansu and northern Sichuan being a source for dispersal of diverse genotypes of *P. striiformis* f. sp. *tritici* into the main wheat belt, and the presence of fingerprint 39 (Fig. 2, brown circles) in Yunnan and Sichuan supports one of the new hypotheses about spore dispersal (39). In both cases, uredospores are dispersed hundreds or thousands of kilometers from western China to the main wheat belt.

LDD of biotrophic fungi also occurs in Europe [e.g., 650 km across the North Sea from Great Britain to Scandinavia when winds are favorable (41) (Fig. 1, migration XIV)] and presumably elsewhere. This rarely contributes to recolonization of crops by pathogens, however, because the cultivation of both autumn- and spring-sown crops usually allows pathogens to persist through theyear. It is relevant, however, when cultivars, resistant in one country, become diseased by virulent fungi from another.

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Adaptation to Host Resistance

Spores dispersed over long distances may overcome previously effective resistance in a target region.

As in pathogen invasions, dispersal of virulent genotypes may be successful even when the density of susceptible hosts is low (28); neither *Mla13* nor *Yr17*, two genes discussed below, was deployed over a large area when cultivars carrying them first became susceptible (42). When a resistance gene is used widely, mutation to virulence in a single pathogen genotype may mean that previously resistant cultivars become susceptible across huge areas. In accordance with the prediction that unusual events may have a disproportionate effect on population dynamics (7), virulent clones may initiate local epidemics whether they are dispersed with the prevail-



Fig. 2. Dispersal of the wheat yellow rust pathogen, *Puccinia striiformis* f. sp. *tritici*, in China. The main area of winter wheat cultivation includes Shaanxi, Henan, Hebei, and Shandong provinces. Solid blue arrows show dispersal of uredospores from southern Gansu province to the main wheat-growing area in autumn (35–38). Dashed arrows indicate additional proposed pathways of uredospore dispersal in autumn (blue) and spring (green) (39). Colored circles indicate sites where genotypes of *P. striiformis* f. sp. *tritici* with indistinguishable genetic fingerprints were found (40) (green, genotype 6; red, 10; purple, 34; blue, 35; orange, 36; brown, 39; and yellow, 46).

co blue mold) in the eastern United States (32) (Fig. 1, migration X). Recolonization of rust pathogens of wheat in North America (33) and India (34) generally follows “*Puccinia* pathways” that are predictable on the basis of host availability and seasonal prevailing winds (Fig. 1, migrations XI and XII).

Research on wheat yellow rust in China has combined molecular markers with other information to investigate extinction and recolonization (Fig. 1, migration XIII; Fig. 2). *Puccinia striiformis* f. sp. *tritici*, which has no sexual phase, must be reestablished each autumn in the main wheat-growing provinces of northern China because there are no host

ing wind (generally west to east in Europe) or against it. Virulent clones may then diversify by further mutation or by recombination with other genotypes (43, 44).

When *Yr17* was introduced widely into European wheat breeding in the early 1990s, it controlled yellow rust completely until 1994, when the first outbreak on wheat with *Yr17* was reported in Great Britain, involving one pathogen genotype (45) (Fig. 3, type K). Type K isolates were then found in 1997 in the first outbreak of yellow rust on *Yr17* wheat in Denmark, then in France and Germany in 1997 and 1998 (46), respectively. Limited diversification of type K has occurred since (Fig. 3, type K*). In 1995, a different *Yr17*-virulent genotype (Fig. 3, type O) was found in England, then in 1997, it was found elsewhere in northwestern Europe at sites up to 1700 km apart (46). The most likely explanation for the widespread distribution of types K and O in northwest Europe was wind dispersal of uredospores from a common source, presumably Britain. In Denmark, the establishment of these types was in part the result of a recolonization after the local extinction of *P. striiformis* f. sp. *tritici* for 1 year (45, 47), whereas dispersal of type O to western France was against the prevailing wind.

The evolution of virulence of *Blumeria graminis* (synonym *Erysiphe graminis*) f. sp. *hordei*, the powdery mildew pathogen, to *Mla13* resistance in barley followed a similar pattern. *Mla13* was first used in the former Czechoslovakia around 1980, then elsewhere in continental Europe, and finally in Great Britain in 1986. In 1988, when *Mla13* cultivars in Britain first had substantial levels of mildew, three clones of the fungus were found in widespread locations (43, 48). One of these was indistinguishable from older isolates from Czechoslovakia and was mostly likely dispersed to Britain from continental Europe (49). Diverse genotypes of *B. graminis* f. sp. *hordei* now have *Mla13* virulence (50), through further mutation and recombination.

Occasionally, single-step intercontinental dispersal overcomes an effective resistance. In 1981, a genotype of the wheat brown rust (or leaf rust) fungus, *Puccinia triticina*, of unknown origin but clearly from outside Australasia, appeared in New Zealand, where it overcame the brown rust resistance of cv. Karamu,

which was >90% of the wheat grown in the North Island at the time (51). By contrast, two genotypes of the wheat stem rust fungus, *Puccinia graminis* f. sp. *tritici*, which were found in Australia in 1969 and had almost certainly been wind dispersed from southern Africa, possibly Angola (52) (Fig. 1, dispersal IV), did not cause disease on any previously resistant cultivar because they lacked the necessary virulences.

How Unusual Are Crop Pathogens?

Aerial LDD is not unique to fungal pathogens of crop plants, but these species show its consequences for population distribution and genetics at their most extreme. Four critical points, common to both invasions of new territory and adaptation to new resistances, are that (i) these species are adapted for LDD; (ii) they can reproduce clonally; (iii) they are highly specific to particular host species or genotypes; and (iv) uniquely, humans frequently create new niches for them by global trade in host species or by the widespread release of effective resistances in new cultivars (2).

The last point means that similar dynamics are rare in nature. New islands and volcanoes, for example, may be colonized by LDD, but conditions are rarely specifically suitable for just a few genotypes of just one species. An exception is the moss *Campylopus pyriformis*, of which a single clone, presumably dispersed by airborne spores, has

colonized snow-free ground near the summits of two very remote volcanoes in Antarctica, Mount Melbourne and Mount Erebus, and diversified thereafter (53). The extreme conditions involved in this example emphasize the unusual nature of the population dynamics of crop pathogens. LDD of seeds, by wind as well as other agents, was important in the colonization of new territory in the form of previously glaciated regions in the Holocene (31) and led to spatial clustering of genotypes that persists to the present (54). The role of farmers in creating vacant niches for pathogens is emphasized by the current pandemic of foot-and-mouth disease (FMD), in which herds in countries kept free of disease through quarantine, culling, or vaccination are potential hosts for the PanAsia strain of the FMD virus (FMDV) serotype O, which is now found worldwide (55). The transmission kernel of FMD, which approximates to an inverse power function (56), is consistent with rare, wind-borne LDD of FMDV particles (57), although wind dispersal seems to have been less important in the 2001 epidemic in Great Britain than in previous epidemics (56).

The limited data available for pathogenic fungi in natural populations generally show no evidence of dispersal on the scale of hundreds of kilometers, except where human intervention has brought together aggressive pathogens and uniformly susceptible hosts, as in chestnut blight (10, 26). This is because of the patchiness and diversity of host

populations, since spores of fungal pathogens of wild plants presumably have the same dispersal characteristics as those of crops. The patchiness of populations following postglacial expansion should be as fascinating a topic with fungi as it is with plants and animals (54), but to our knowledge, only one case has been studied so far. *Coccidioides immitis*, an aggressive pathogen of mammals, is endemic to North America but appears to have codispersed from Texas to South America, which has comparatively little genetic diversity, with one of its hosts, probably humans, in the late Pleistocene. Although *Coccidioides immitis* undergoes LDD by windblown arthroconidia, it is more likely that intercontinental dispersal was by cysts in its human host (58).

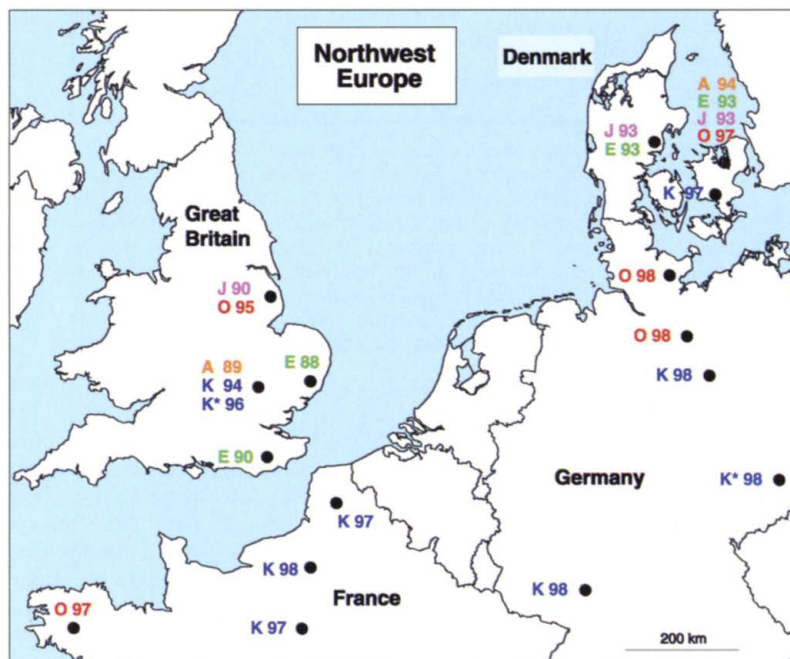


Fig. 3. Distribution of *Puccinia striiformis* f. sp. *tritici* (wheat yellow rust) genotypes found in more than one country in northwest Europe between 1988 and 1998 (45, 46). Letters indicate genotypes of indistinguishable amplified fragment length polymorphism phenotype (45) and pathotype (27); numbers refer to the year of sampling. Genotypes virulent on *Yr17* were O, K, and K* (which differed from K only in being virulent to *Yr6*). Phylogenetic analysis indicated that each type shown on this map had a single clonal origin within the European population of the fungus (46).

The unique combination of biological factors and human intervention in the life histories of wind-dispersed plant pathogens can result in such exceptional events as invasions of new territory on global or continental scales or the rapid spread of virulent genotypes on previously resistant cultivars. It is a considerable challenge to develop a predictive model that would describe such rare events. The combination of separate lines of research in epidemiology and population genetics has provided qualitative insights into the causes and consequences of these processes, and a goal for the next few years is to define with greater precision the limits of predictability of the occurrence of such unusual events.

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