Resurgence of the Irish Potato Famine Fungus

After 150 years, the late blight fungus is again menacing farmers

William E. Fry and Stephen B. Goodwin

More than 150 years after it first devastated potato crops in the United States and Europe and led to the Irish potato famine, the plant pathogenic fungus *Phytophthora infestans* is again creating a major plant health problem. Migrations of virulent and fungicide-resistant strains in the past two decades have caused a worldwide resurgence of the potato (and tomato) late blight disease. Epidemics in parts of the United States and Canada during the early 1990s were locally devastating, sometimes causing total crop loss and severe economic hardship for many potato and tomato growers. This resurgence supports the view that introduced pathogens and new variants of old ones present a real and immediate threat for plants as well as for animals and humans.

The resurgence of previously controlled infectious diseases and the emergence of new ones are of increasing concern to scientists and the general public (Berkelman et al. 1994, Cassell 1994, CDC 1994, 1995, Garrett 1994). Optimism fostered by the discovery and successful application of potent antibiotics has been dampened by the appearance of drug-resistant pathogens. An increasing number of human pathogens are becoming resistant to several previously effective drugs. For example, multiple drug–resistant tuberculosis pathogens have become especially noticeable and worrisome (Cassell 1994). Some strains of staphylococci are also resistant to multiple drugs, making effective drug treatment, at times, impossible (Cassell 1994). Furthermore, introductions of exotic pathogens to new locations or novel hosts have created new diseases and worsened old ones. For example, cholera had all but disappeared from the New World during the nineteenth century, but in the early 1990s nearly 1 million cases were reported in Central and South America (Cassell 1994). In addition, a deadly new strain of the pathogen was detected recently in India and Bangladesh—killing some adults in only nine hours (Cassell 1994). One case of the new cholera was detected in Los Angeles in a recently returned traveler, but this case was contained by vigilant health care professionals (Cassell 1994). During the past ten years, epidemics caused by new pathogens that threaten human health (e.g., HIV, Ebola virus, hantavirus) have raised concern throughout the world.

Migrations of virulent and fungicide-resistant strains in the past two decades have caused a worldwide resurgence of the potato late blight disease

Although most attention has been directed at human illnesses, there are also serious new problems with diseases of animals and plants. Mad cow disease (bovine spongiform encephalopathy) of cattle in the United Kingdom has been of particular concern because of some suggestions that it could be transmitted to humans who consumed infected beef (Shadduck et al. 1996). A symptomatically similar spongiform encephalopathy has appeared recently in mule deer, elk, and white-tailed deer reared in captivity on the great plains of North America (Nettles 1996). Many plant diseases that are caused by geminiviruses (transmitted by whiteflies) have wreaked havoc in Africa and now in the Caribbean. Geminiviruses are now accompanying their whitefly vectors to the continental United States (Fry 1996). Dogwood anthracnose recently has destroyed flowering dogwoods throughout much of eastern United States, although the origin of the fungal pathogen that causes the disease remains unknown (Daughtrey et al. 1996). Head scab of wheat and

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Figure 1. Potato late blight (caused by the oomycete *Phytophthora infestans*) is one of the most devastating of all plant diseases (Hooker 1981). All parts of the plant, including leaves (**a**), stems (**b**), and tubers (**c**), are susceptible to infection. Tomato foliage and fruit (**d**) also are highly susceptible to some genotypes of the pathogen (Legard et al. 1995). The disease is strongly influenced by environmental conditions, being favored by high moisture and moderate temperatures (15–25 °C). Photos: (**a**) R. V. James; (**d**) T. A. Zitter.

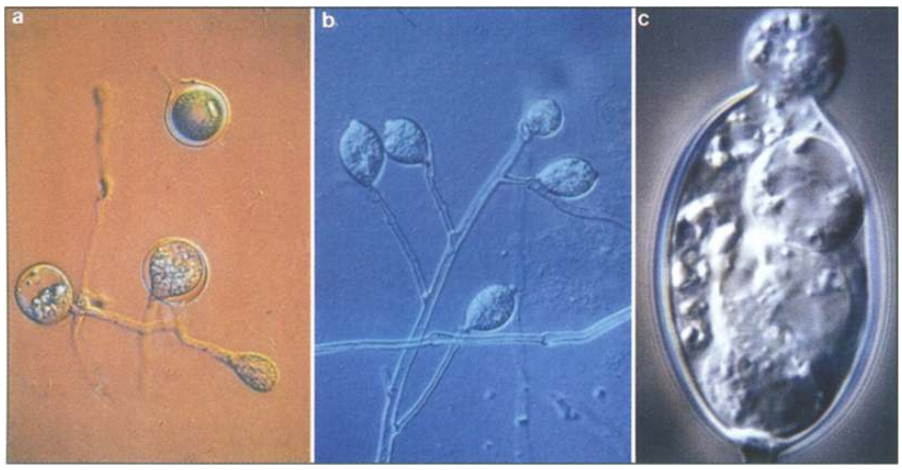

Figure 2. Reproduction of the oomycete *Phytophthora infestans*. The product of sexual reproduction is an oospore (**a**), which is the only form of the fungus that can survive for months or years in the absence of a living host. Oospores are typically 20–30 µm in diameter. Asexual reproduction occurs via multinucleate spores called sporangia (**b**), which are typically 20–25 × 25–35 µm. These sporangia are produced under moist conditions (relative humidity at or near saturation or with free moisture) and can be dispersed by wind currents or in splashing water droplets. Sporangia germinate directly via germ tubes or indirectly (**c**) by the release of uninucleate, biflagellate swimming zoospores. Zoospores can remain motile for nearly one hour before encysting. The zoospores subsequently form germ tubes that can penetrate host tissue. Epidemics are caused by rapid asexual reproduction. Although the fungus can survive indefinitely as an asexual organism, as such it is essentially an obligate parasite. There are two mating types (A1 and A2), and sexual structures are typically produced only in the presence of an individual of opposite mating type. Each individual is capable of producing both male (antheridia) and female (oogonia) structures, so although the organism is bisexual, it is almost exclusively outbreeding. Photos: (**b**) R. C. Shattock; (**c**) B. G. Turgeon.

barley, caused by a ubiquitous fungal pathogen, has recently caused losses to midwestern farmers of more than $1 billion (McMullen et al. 1997).

The plant disease known as potato (and tomato) late blight is a particularly well understood example of how resistance to chemical controls and migrations have worsened a disease that had been effectively managed for decades. Late blight (Figure 1) is caused by the filamentous fungal-like oomycete, *P. infestans* (Figure 2). Oomycetes have close affinities with golden-brown algae, sharing attributes such as motile zoospores (Figure 2), diploid vegetative cells, and cellulose cell walls—none of which are possessed by the true fungi. *Phytophthora* species are sometimes called "water molds" because their growth (and disease development) is favored by wet conditions. Each individual is bisexual, capable of producing both female (oogonia) and male (antheridia) structures. Sexual reproduction requires the interaction of two mating types (A1 and A2). Fertilized oogonia develop into oospores (Figure 2) that can survive adverse conditions, such as freezing or drying, for as long as months or even years. When only one mating type is present, the organism reproduces asexually through the production of sporangia (Figure 2). Late blight epidemics are essentially population explosions resulting from rapid asexual reproduction of *P. infestans* in susceptible host tissue (foliage, stems, and tubers of potatoes or foliage, stems, and fruits of tomatoes). During its asexual phase, *P. infestans* is essentially an obligate parasite, requiring living host tissue for its continued survival.

Late blight can be a remarkably rapid and destructive disease: Fields that appear healthy, but contain low incidence of disease, can be devastated within days (Figure 3). It is difficult to detect low levels of disease, and the pathogen can reproduce rapidly. The disease cycle (penetration, colonization, sporulation, and dispersal) can occur in less than five days; each individual late blight lesion (three lesions are seen in the leaflet in Figure 1a) can produce as many as 300,000 sporangia per day (Legard et al. 1995). Sporangia pro-

Figure 3. Effects of late blight on potatoes. When severe, the disease converts fields of healthy potatoes (a) into fields of partially (b) or completely (c) destroyed plants. When infected tubers are stored in warm, wet conditions, soft-rot bacteria can become especially troublesome and can destroy thousands of pounds of potato tubers. Sometimes, rotting tubers are discarded in huge piles (d). This is a serious problem because infected tubers that survive the winter in large piles can be sources of the fungus to infect the crop in the succeeding year. Thus, an important component of late blight management is the destruction of piles of cull potatoes.

duced in the foliage can be washed from leaves, to infect the tubers. Some infected tubers may be destroyed before harvest, but others become visibly diseased in storage. Bacteria that cause soft-rot diseases often invade potato tubers infected with *P. infestans*, resulting in the "meltdown" of stored tubers. Under severe infection, entire storages must be discarded, sometimes producing huge piles of cull (unusable) potatoes (Figure 3d). Thus, late blight can continue to cause problems even after potatoes are harvested.

Late blight was first noticed in the 1840s and became of historic significance in Europe when it caused the Irish potato famine. In Ireland, 1.5 million people died and a similar number emigrated (Bourke 1993, Large 1940, Woodham-Smith 1962). These epidemics stimulated intense investigation about the nature of plant disease and are generally regarded as initiating the development of plant pathology as a discrete discipline. They also stimulated thought about the nature of disease in general. DeBary's demonstration that inoculation of potatoes with *P. infestans* could induce late blight contributed to the acceptance, during the 1870s, of the germ theory of disease. By the mid-twentieth century, late blight was kept to generally tolerable levels by agricultural practices that included planting healthy seed tubers, eliminating other sources of the fungus, treating potato with fungicides, and using moderately resistant potato varieties. However, at the end of the twentieth century, late blight has once again become a widely feared disease.

In this article, we describe the resurgence of late blight, document how contemporary and historical migrations of the pathogen have contributed to disease outbreaks, and suggest actions to minimize future resurgences.

Table 1. The impact of late blight on one farm in the northeastern United States in 1994.

Farm characteristic	1992	1993	1994
Potato acreage	202 ha	202 ha	196 ha
Cost of production per hectare	$3777	$3871	$4265
Cost of pesticide spray per hectare	$312	$406	$694
Marketable yield (tons) per hectare	32	32	6
Estimated value after harvest[a]	$1,010,000	$1,215,000	$212,000

[a]The value of the harvested crop is influenced by fluctuation in the price of potatoes. The cost inflicted by late blight on this individual grower was devastating. Marketable yield decreased by 80%, despite a doubling of pesticide costs. The grower defaulted on three contracts to provide potatoes, had to dispose of 4090 metric tons of rotting potatoes in an environmentally benign manner, and was forced out of business.

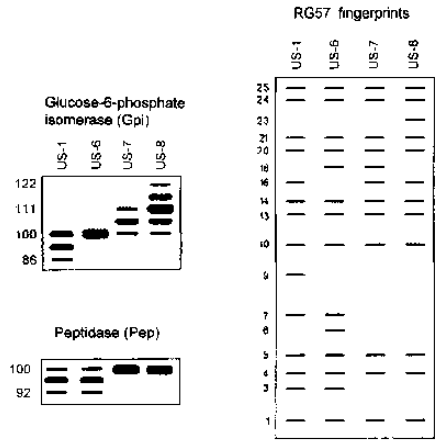

Figure 4. Schematic representations of dimeric allozyme banding patterns for the enzymes glucose-6-phosphate isomerase (GPI) and peptidase (PEP) and DNA fingerprints for the four major clonal lineages of *Phytophthora infestans* detected in the United States since 1980 (Goodwin et al. 1994a, b, 1995b). Allozyme banding patterns (left) were determined from homogenates of mycelia using potato starch gels or cellulose acetate membranes as described in Goodwin et al. (1992a, 1995b). The numbers along the left side of the allozyme patterns represent the relative migration rates of dimeric enzymes; 100 refers to the migration of the most commonly occurring enzyme species. Alleles are named for the migration rates of their proteins relative to the migration rate of the protein produced by the 100 allele. Because *P. infestans* is diploid, each individual has at least two alleles at each locus. Thus, the genotype for US-1 at the locus for GPI is 86/100. DNA fingerprint patterns (right) were determined from Southern analysis of nuclear DNA fragments digested with the restriction enzyme EcoRI and probed with RG57, a moderately repetitive nuclear DNA, as described by Goodwin et al. (1992a). Numbers on the left side of the DNA fingerprint patterns identify band numbers detected with RG57. The bands range in size from 1.1 kb (band 1) to approximately 18 kb (band 25). US-1 and US-6 are of the A1 mating type, and US-7 and US-8 are of the A2 mating type. US-1 is thought to have been in the United States since the nineteenth century (Goodwin et al. 1994b), whereas US-6, US-7, and US-8 have been introduced recently (Goodwin et al. 1994a, 1995b). Strains US-6 and US-7 infect both potatoes and tomatoes but seem especially virulent on tomatoes. The association of GPI patterns with genotype has enabled plant pathologists to use a rapid-allozyme technique (Goodwin et al. 1995a) to predict the biological characteristics of isolates from affected fields.

Resurgence of potato late blight

Crop losses due to potato late blight disease increased dramatically in the early 1980s in Europe, and subsequently in the Middle East and Far East (Dowley et al. 1995, Fry et al. 1993, Leary 1993, Lucas et al. 1991). By the late 1980s and early 1990s, potato late blight was causing severe problems in North America (Faber 1994). Late blight is now so important internationally that in March 1996 the International Potato Center in Lima, Peru, called for a global initiative to combat late blight (CIP 1996). This disease has become the subject of meetings and newsletters involving scientists and growers worldwide.

Initially, the sudden intensification of problems with late blight disease in Europe was attributed to the emergence of a drug-resistant strain. Resistance to metalaxyl, the only effective therapeutic fungicide (i.e., that can slow an established infection) was found in European populations of the pathogen beginning in 1980 (Davidse et al. 1981, 1991, Dowley and O'Sullivan 1981, 1985). (All other available fungicides were prophylactic, that is, they were effective only when applied before the pathogen caused infection.) However, retrospective analysis of *P. infestans* populations using phenotypic, biochemical, and DNA fingerprint markers showed that increased levels of disease in Europe also coincided with the appearance of exotic pathogen strains (Drenth et al. 1993, Goodwin et al. 1994b, Hohl and Iselin 1984, Mosa et al. 1989, Spielman et al. 1991, Sujkowski et al. 1994; see below). Moreover, the exotic European strains consisted of individuals of both mating types, which has now enabled the pathogen population in Europe to reproduce sexually (Drenth et al. 1995, Sujkowski et al. 1994) rather than only asexually, as it had previously (Goodwin et al. 1994b). The exotic strains probably came from Mexico (Niederhauer 1991).

The United States and Canada were at first spared Europe's severe late blight problems, but in the late 1980s and early 1990s, occurrences of severe late blight epidemics and of metalaxyl resistance began to be noticed in North America. Late blight resurgence was observed first in the Pacific Northwest and subsequently in almost all major potato (and tomato) production areas in the United States and Canada. The disease has destroyed huge acreages of potatoes (Figure 3) and caused economic ruin for many farmers (Dao 1995, Faber 1994, Gillis 1993). The total economic impact is still being investigated. These recent epidemics were frequently fearsome in their sudden appearance and severity. For example, the pathogen's impact on one potato farm in the Northeast in 1994 is illustrated in Table 1.

Contemporary migrations of late blight

Because migrations of exotic strains had been associated with intensified disease problems in Europe and elsewhere, plant pathologists quickly investigated whether exotic strains were associated with increased dis-

ease in the United States. Not only were exotic strains detected in most US and Canadian locations where disease had been especially severe, but such exotic strains were the only ones detected (Goodwin et al. 1994a, b). Surprisingly, genetic marker analysis showed that most disease was attributable to only a few asexually reproducing strains of the pathogen (Figure 4). In many cases, only a single genotype (clone) has been detected in any given field and, in some cases, even in an entire region (Goodwin et al. 1994a, 1995b, 1996). Three of the four most common strains had been introduced recently from northwestern Mexico (Goodwin et al. 1994a, 1995b); the fourth strain was a relic of the indigenous population.

Although other plant pathogen migrations have occurred, in most cases their impacts are not well known. Therefore, plant pathologists were not prepared for the rate at which the new strains spread (Figure 5) or for the severity of the current late blight epidemic. In most potato and tomato production regions in the United States and Canada, late blight had not been a serious problem for decades, and when the disease had occurred it had been suppressed easily with effective chemical and cultural techniques. Growers who were unaccustomed to the fungicide resistance and aggressiveness of the exotic strains and who believed that they could avoid the new strains (by good luck) were the least prepared and incurred the greatest losses. Growers who were more prepared took more extensive precaution to prevent the initiation of an epidemic by planting only healthy seed tubers and by using fungicides prophylactically. The rapid "colonization" by one of these strains, US-8, has been especially impressive. After being detected only in New York in 1992 and in Maine in 1993 (Goodwin et al. 1995b), US-8 was reported from 23 states during 1994 and 1995. As happened in the 1980s in Europe (Spielman et al. 1991, Sujkowski et al. 1994), the exotic strains have largely replaced the resident strains (Figure 6).

The magnitude and severity of the recent epidemics have galvanized US and Canadian scientists to intensify a multifaceted approach for late blight control (Lehnert 1996, Powelson et al. 1996). The short-term goal has been to keep potato and tomato growers in business until more could be learned about how to manage the exotic strains. The foundation of this short-term strategy is integrated management. Cultural techniques (e.g., planting disease-free seed tubers and eliminating overwintering sites of the fungus, such as unharvested or cull potatoes) have been used to lessen the probability of pathogen occurrence. Procedures that slow *P. infestans* growth rates (the use of less-susceptible cultivars and the application of appropriate prophylactic fungicides) have also been emphasized. Certain fungicides registered for use in Europe received emergency permission for use in the United States during the 1995 and 1996 growing seasons. These fungicides, which had proven effective in Europe and in Central and South America, provided some therapeutic activity. None of the other available fungicides had therapeutic activity against the exotic strains. Monitoring for first indications of disease and for disease-favorable weather (i.e., moderate temperatures with rainfall or high relative humidity and abundant leaf wetness) was intensified. This management program was combined with an intensive education effort to inform growers of the much greater threats that the exotic strains posed in comparison to the previous strains.

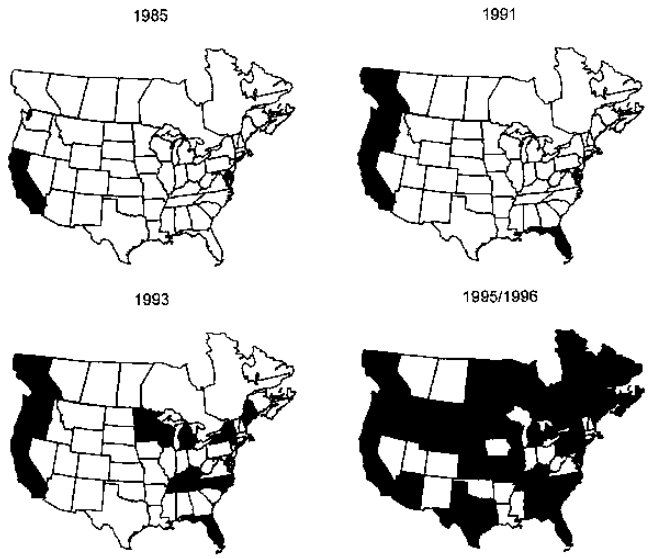

Figure 5. States and provinces (cumulative from 1985 to 1995/1996) in which exotic strains of *Phytophthora infestans* were detected in the United States and Canada. Data are from many sources (W. E. Fry and S. B. Goodwin, unpublished results; Deahl et al. 1991, Goodwin et al. 1994a, 1995b, 1996). Shading indicates states and provinces with confirmed occurrences.

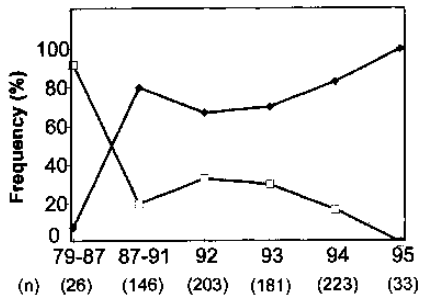

Figure 6. Relative frequencies of exotic (black diamonds) and resident (open squares) strains of *Phytophthora infestans* in the United States and Canada from the 1980s to 1995. Samples were sent to Cornell University for identification from regions reporting late blight by colleagues throughout the United States and Canada. The aggregate sample was not comprehensive, but it was probably representative of known occurrences. Sample sizes for each year are indicated in parentheses (S. B. Goodwin and W. E. Fry, unpublished results; Goodwin et al. 1994a, 1995b, 1996).

Despite these precautions, which were implemented during spring 1995, the exotic strains appeared in new locations during the summer and fall of that year. Disease was particularly severe in parts of the Pacific Northwest, where the weather is conducive to disease development. For example, in the Columbia Basin of Washington and Oregon, late blight affected 66,000 ha, and the increased costs associated with combating the problem are estimated to be $30 million (Johnson et al. 1997). In Idaho, where late

blight had previously been rare, exotic strains caused significant problems on at least 8000 ha. The alarming events of the last several years have caused farmers and scientists to view future growing seasons with apprehension.

Although the reasons for the resurgence are not yet fully understood, some factors contributing to the severity of the recent epidemics are known. Resistance of the exotic strains to the fungicide metalaxyl is one (Deahl et al. 1993, Goodwin et al. 1996). This fungicide, in combination with prophylactic fungicides, previously provided almost complete protection against late blight (Fry et al. 1979). The appearance of metalaxyl-resistant exotic strains (Deahl et al. 1993, Goodwin et al. 1996) created a serious problem because no other available fungicide could halt an established epidemic. Due to the speed of late blight epidemics, there were sometimes only a few days between disease detection and total foliage destruction (Figure 3). In many cases, by the time it was clear that the pathogen was resistant to metalaxyl, the disease was so well established that there was insufficient time for effective use of prophylactic fungicides.

Another factor contributing to the severity of the most recent epidemics is the increased virulence of exotic strains compared with those that were present previously. The US-8 strain, which had not been detected in the United States before 1992, is especially virulent on potato foliage and tubers (Kato and Fry 1995, Lambert and Currier 1997). This strain destroyed whole fields in the eastern United States in 1994 and was distributed even more broadly in 1995. Computer simulations predict that because of increased aggressiveness, adequate control of exotic strains may require large increases (15%–25%) in the use of prophylactic fungicides in the United States and Canada.[1]

One possible explanation for the greater virulence of the exotic strains than that of the strains they have replaced is "Muller's ratchet" (Muller 1964), a term that refers to the gradual diminution of fitness in an asexual lineage due to the accumulation of deleterious mutations. Before the 1980s, populations of *P. infestans* worldwide consisted primarily of only a single mating type (A1) and therefore had been limited to asexual reproduction for more than 150 years. During this time, deleterious mutations could have accumulated in the asexual population. Because sexual reproduction allows recombination to occur, it can yield individuals with fewer deleterious mutations and, thus, with greater fitness than the parents. The exotic strains were introduced recently from sexually reproducing populations in Mexico, and they may have contained fewer deleterious mutations than individuals of the resident populations in Europe and in the United States and Canada.

Now that sexual reproduction is part of the *P. infestans* life history outside Mexico, the effect of Muller's ratchet will be diminished. Sexual reproduction has been detected in the Netherlands and Poland (Drenth et al. 1995, Sujkowski et al. 1994). The complex genetic structure of *P. infestans* populations in southwestern British Columbia (Goodwin et al. 1995b) is consistent with the occurrence of sexual reproduction, and this population differs from most other populations in the United States and Canada. If Muller's ratchet ceases to operate, populations in Europe and in the United States and Canada may remain very aggressive.

Migrations in the history of late blight

Movements of both host and pathogen have played a pivotal role in the history of late blight. The highlands of central Mexico is a secondary center of diversity for a number of tuber-bearing *Solanum* species; this region also contains the center of diversity for *P. infestans* (Goodwin et al. 1992b, Tooley et al. 1985). By contrast, cultivated potatoes (*Solanum tuberosum*) are derived from plants that are native to the Andes of South America (Hawkes 1945). Most plant pathologists believe that until recently, populations of *P. infestans* outside of central Mexico were asexual (Goodwin et al. 1992b, 1994b). These populations contained only one (A1) of the two mating types required for sexual reproduction. Populations in central Mexico, by contrast, were sexual (Niederhauser 1991, Tooley et al. 1985). In central Mexico, therefore, *P. infestans* presumably coevolved with a number of wild *Solanum* species (Niederhauser 1991). Therefore, late blight of cultivated potato (and tomato) probably did not exist until host and pathogen were brought together by humans. The agricultural problem of late blight therefore may be considered almost entirely related to human activity.

Because *P. infestans* evolved in a small geographical area, it has been possible to trace the probable paths and timing of each major migration. Late blight was unknown before 1843, when it first affected potato crops in the northeastern United States (Stevens 1933). Thus, the first known migration of *P. infestans* from Mexico probably occurred around 1840. Europe was spared until June 1845, when the new disease appeared in Belgium. The mechanism of introduction to either location is not known with certainty. We can speculate that a scientist or amateur botanist might have brought infected plant materials from Mexico to northeastern United States; subsequent transmission from North America to Europe in an infected tuber is certainly plausible. Although other maladies had affected potatoes in previous years (Bourke 1993, Large 1940), this disease was different because it completely destroyed both the foliage and tubers. By August 1845, the disease occurred throughout northwestern Europe and southern England (Bourke 1964). John Lindley, editor of the *Gardener's Chronicle and Agricultural Gazette*, sounded the alarm in a 23 August 1845 editorial: "A fatal malady has broken out amongst the potato crop. On all sides we hear of the destruction. In Belgium the fields are said to have been totally desolated" (Large 1940). By September, the disease reached Ireland.

The high productivity of potatoes in the absence of late blight set the stage for the misery that ensued in Ireland (Bourke 1993). An Irish "potato economy" had fueled a popula-

[1] M. Kato, E. S. Mizubuti, and W. E. Fry, unpublished results.

tion explosion (Bourke 1993); the Irish population had nearly tripled, to almost 8.5 million, during the 60 years before the outbreak (Woodham-Smith 1962). Although cereal crops were also grown in nineteenth-century Ireland, these crops were used to pay rents; it was the potato that sustained the population, most of whom were destitute. After the appearance of late blight, the population fell precipitously, until it approached 4 million by the turn of the century (Cox and Large 1960).

Retrospective analysis has led to the hypothesis that the Irish potato famine was caused by a migrating pathogen population of extremely limited genetic diversity. Thus, migrations of even one or a few clones of the late blight pathogen can have devastating consequences. DNA fingerprint analyses of extant populations worldwide suggest that the initial migration in the 1840s contained little genetic diversity (Goodwin et al. 1994b). In fact, the Irish potato famine was probably caused by a single *P. infestans* clone that caused the pan-European epidemic in 1845. Subsequently, this pathogen strain was introduced to Asia, Africa, and South America (Goodwin et al. 1994b).

Why have migrations from Mexico not occurred more frequently throughout history? Mexico, historically, has not been a large producer of potatoes, so opportunities for migrations associated with the movement of potatoes have been extremely limited until recently. After the 1840s, there may not have been another significant migration of the fungus out of Mexico until the 1970s. In 1976, a drought resulted in the underproduction of potatoes in Europe, and large quantities of potatoes (approximately 25,000 tons) were shipped to Europe from Mexico during the winter of 1976–1977 (Niederhauser 1991). These potatoes appear to have provided the vehicle for the second migration because new genotypes of the fungus were detected in isolates collected in Europe at least as early as 1981 (Hohl and Iselin 1984). Some potato cultivars with moderate resistance to the old clonal lineage of *P. infestans* succumbed to the new genotypes (Sujkowski et al. 1996). Development of potato and tomato production areas in northern Mexico within the last 20–25 years has provided increased opportunities for migrations into the United States. Mexico exports tomatoes to the United States, and infected tomato fruits can carry *P. infestans*.

Consequences and next steps

The presence of highly virulent, fungicide-resistant genotypes of *P. infestans* in major potato- and tomato-growing regions of the world, combined with the low levels of genetic resistance to these strains in commercial cultivars of these crops, is alarming. Although control of the exotic strains is possible with significantly increased application dosage and frequency of available fungicides, this strategy is not only expensive but also inconsistent with worldwide efforts to decrease pesticide dependency in agriculture. In the short term, the late blight resurgence will be felt primarily in countries whose farmers are too poor to purchase the additional fungicides necessary for adequate control; countries in Africa and eastern Europe are particularly at risk. Just as 150 years ago, the poor are likely to suffer the most. However, a famine of magnitude similar to the one that affected Ireland in the 1840s is unlikely because no country now depends solely on the potato for food.

The most significant long-term impact of the contemporary migrations of *P. infestans* is that both mating types (which are necessary for sexual reproduction) are now widely distributed, so meiotic recombination will occur in locations that previously were spared the sexual phase of this fungus. The resulting new gene combinations and increased survival via sexual spores will make the disease more difficult to control. Sexual reproduction has been confirmed in Europe (Drenth et al. 1995, Sujkowski et al. 1994), and there is preliminary evidence that it is occurring in British Columbia (Goodwin et al. 1995b). Sexual reproduction could also explain the diverse genotypes found recently in populations of *P. infestans* in Oregon and Washington.[2]

[2]R. W. Sandrock, P. B. Hamm, J. Miller, D. A. Johnson, and W. E. Fry, unpublished results.

The effect of sexual reproduction on the epidemiology of late blight is not understood completely. Oospores resulting from sexual reproduction can survive in soil for at least months and possibly years (Drenth et al. 1995, Pittis and Shattock 1994). If oospores provide a reservoir of the fungus, then epidemics might be initiated even earlier in the growing season than previously, and rotation away from potatoes for one year will no longer be adquate to eliminate the pathogen. Sexual reproduction will generate new gene combinations, and these combinations may result in characteristics that were previously unknown, thus creating uncertainty in our attempts to suppress these pathogen populations.

The resurgence of potato late blight illustrates the need to intensify research on the biology of the pathogen, on the events that lead to host susceptibility or resistance, and on additional technologies that can mitigate the disease. The National Association of State Departments of Agriculture and the National Potato Council have called for increased research efforts in the United States. Communication about the current state of knowledge and education of farmers have been the initial parts of this effort, and there have been many conferences, meetings, and symposia sponsored by the private sector, cooperative extension, the USDA, and the American Phytopathological Society. Internationally, there have been conferences in Ireland (1995), Peru (1996), and Mexico (1996). The International Potato Center in Lima, Peru, has called for a global initiative on late blight (CIP 1996). An international late blight project, Progama Internacional Cooperativo del Tizon tardio de la PAPA (PICTIPAPA), has been initiated in Toluca, Mexico.

In addition to new and intensified control efforts, thorough investigations into disease epidemiology and pathogen genetics are needed. It has been difficult to develop potato and tomato varieties with stable resistance to late blight because although specific resistance genes are known, they are overcome rapidly by mutants in local fungus populations (Wastie 1991). Efforts to identify and transfer novel resistance genes

from other plants, bacteria, and even animals into potatoes and tomatoes (Liu et al. 1994, Staskawicz et al. 1995, Wu et al. 1995) are still in their infancy.

The late blight resurgence highlights the threat posed to modern agriculture by migration of plant pathogens. Earlier in the twentieth century, exotic pathogens were responsible for the demise of the American elm and the American chestnut. These hosts were particularly vulnerable to exotic pathogens with which they had not coevolved. Vulnerability to exotic pathogens may be particularly important for clonally propagated crops, such as potatoes, because of the homogeneity of the host crop within fields, within regions, and among regions. Thus, if an introduced pathogen is particularly aggressive on a widely grown cultivar, crops in widely separated growing regions would be at risk. This vulnerability compounds the other risks (other pathogens or insect pests) associated with the limited genetic diversity of modern agricultural crops. The continuous discovery of novel plant pathogens (Fry 1996) indicates that there are additional pathogens in the wild that have not yet come in contact with our main crop plants. Some of these new encounters will probably result in economic hardship and human suffering.

Even if some pathogens do not threaten survival of their host, their migration into new locations can have devastating economic consequences because of quarantines imposed by countries to prevent movement of potentially infected agricultural products. Discovery of a quarantined pathogen in a region may restrict severely the markets available to growers in that region. This economic effect of quarantines is exemplified by the recent discovery of Karnal bunt of wheat in parts of southwestern United States. Before March 1996, this fungal disease had never been detected in the United States and had a limited global distribution. Although the disease itself is not serious, quarantines by other countries jeopardized the $5 billion US wheat export crop. Ironically, before Karnal bunt was discovered within its borders, the United States had been a leading proponent of preventing entry into the United States of all wheat from countries where this disease had been discovered, regardless of its distribution in that country. Fortunately, during the first several months after the initial discovery of Karnal bunt in the United States, many countries agreed to permit importation of wheat from areas in the United States that were still free of the disease.

Accelerated introductions of pathogens due to global commerce and travel and the occurrence of antibiotic-resistant microbes have been widely recognized as public health problems (Berkelman et al. 1994, Cassell 1994, CDC 1994, 1995, Garrett 1994), but these issues also apply to plants and animals. The potato late blight disease illustrates how exotic and chemical-resistant pathogens are a serious plant health concern. Short-term precautions may include quarantines, but the safest long-term precaution is a solid understanding of the biology of the pathogens and the diseases they cause. Only from this understanding will we be able to devise the technology and education necessary to control these diseases.

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