

## Re-emergence of Potato and Tomato Late Blight in the United States



**D**uring the late 1980s and early 1990s, late blight of potatoes and tomatoes re-emerged as an important disease in the United States and Canada. Problems caused by the disease have been so dramatic at times that they attracted national attention of the press, as shown by the sample headlines above. The dark brown background consists of several fields in which all foliage was killed by an epidemic of late blight, lineage US-8, in July and August 1994.

The re-emergence of late blight in the United States and Canada follows similar resurgences worldwide (11,50). The im-

mediate cause of the global re-emergence of late blight is the recent migration (movement) of exotic strains of *Phytophthora infestans*. Globally, there have been several independent migrations from Mexico to other countries (25). The exotic strains in the United States and Canada are different from the exotic strains on other continents and apparently were distributed by different migration events (18,22,30,31,36,47,68,75). The situation in the United States and Canada is unique because the introduction and distribution of exotic strains were recorded year by year, rather than retrospectively. The purpose of this article is to describe the dynamics, magnitude, and impact of the migrations that have led to the recent re-emergence of late blight.

### Biology of *Phytophthora infestans*

A brief review of the biology of this oomycete plant pathogen provides context that will facilitate understanding of recent

events. Oomycetes are a group of organisms in a kingdom separate from the true fungi, plants, or animals. Depending on classification, they are included in the Kingdom Protocista or Chromista (17,21). This group of organisms is characterized by the absence of chitin in the cell walls (true fungi contain chitin), zoospores with heterokont flagella (one whiplash, one tinsel) borne in sporangia, diploid nuclei in vegetative cells, and sexual reproduction via antheridia and oogonia. The genus *Phytophthora* contains some species (including *P. infestans*) that are heterothallic (A1 and A2 mating types) and some that are homothallic. The closest phylogenetic relatives of the oomycetes include some algae (those with heterokont zoospores, and which produce antheridia and oogonia as sexual structures). Acceptance of the concept that oomycetes were unrelated to true fungi (21,46) has occurred gradually over the last 30 years and was strongly influenced by the investigations of Sansome (61–64) and others (6,7,69–72).

Dr. Fry's address is: Department of Plant Pathology, 334 Plant Science Building, Cornell University, Ithaca, NY 14853; e-mail: wef1@cornell.edu

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The western world became aware of *P. infestans* with the devastating late blight epidemics in the northeastern United States and Europe in the 1840s (5,74). The Irish Potato Famine is a well-known result of these early epidemics. Tomato late blight (Fig. 1) was detected sometime later and has also been a persistent problem. While many scientists now consider the mid-nineteenth century to be the first encounter between potatoes and *P. infestans*, some speculate that late blight occurred in South America before it appeared in Europe and the United States (1,4). Potatoes (*Solanum tuberosum*) are native to the South American Andes Mountains (40). Most scientists agree that the center of origin of *P. infestans* is in the highlands of central Mexico and that this region has been the ultimate source for all known migrations (26,30–32,35,59,77). Certainly populations of *P. infestans* in central Mexico are more diverse than populations in any other location, and a large group of tuber-bearing *Solanum* species apparently co-evolved with *P. infestans* there. It was only in central Mexico that both mating types of the pathogen were common prior to the 1980s (29,59,77). The pathogen population in central Mexico is the only one in which we have found more than 25 bands identified by the DNA fingerprint probe RG57, and four and seven alleles at the allozyme loci *Peptidase* (*Pep*) and *Glucose-6-phosphate isomerase* (*Gpi*), respectively (24,28,30,31, 35,47,55,77; W. E. Fry and S. B. Goodwin, unpublished results).



**Fig. 1. Tomato late blight. (A) Field of tomatoes devastated by late blight. The lineage causing this devastation was adapted to tomatoes. Some plants are nearly completely defoliated. Foliage, stems, and fruit (B) are susceptible. Sporulation can occur from lesions on any part of the plant. (B from T. A. Zitter)**

## Clonal Lineages

The concept of a clonal lineage is important to understanding the population biology of *P. infestans*. Clonal lineages also have been identified in populations of other plant pathogens, including *Sclerotinia sclerotiorum*, *Erysiphe graminis*, and *Magnaporthe grisea* (2,8,48,53). A clonal lineage includes the asexual descendants of a single genotype (2). Initial description of a clonal lineage requires a large number of genetic markers. Markers used to define clonal lineages of *P. infestans* have included mating type (A1 or A2), allozyme genotypes (77), and DNA fingerprints (33), developed during the mid-1980s and early 1990s. Pathotype appears to be such a rapidly evolving character (37,76) that it has very limited application in population genetic studies. Thus, it is primarily mating type and the neutral molecular and biochemical markers that have permitted reliable analyses of populations of *P. infestans*.

The conclusion emerging from population genetic studies on *P. infestans* is that prior to the 1980s, world populations of this pathogen were dominated by or consisted solely of a single clonal lineage (termed US-1 by Goodwin et al. [31]). Although pathotypic diversity could be quite large (66), that diversity seemed to occur within a single clonal lineage. Minor differences within a clonal lineage arise from mutation or mitotic recombination, and, as expected, older lineages contain greater diversity than younger ones (37,76). Prior to the 1980s, only A1 mating types were common outside Mexico, and A2 mating types were either very rare or absent (41). The occasional report of oospores (12,60,65) may have resulted from the fact that old cultures sometimes produce oospores (65), especially in the oatmeal agar used by early investigators (32).

The most likely explanation for the previous worldwide predominance by a single clonal lineage of *P. infestans* is that upon transport of this oomycete from central Mexico, first to the United States, then to Europe, and subsequently to the rest of the world, it went through very tight bottlenecks, and the surviving (founder) populations contained very little genetic diversity (31). That is, the first introduction to the United States probably involved very little of the pathogen with little genetic diversity. No one knows how the introduction occurred, although it seems that infected plant material or perhaps infested soil were the likely avenues. If more than a single clonal lineage survived the bottleneck into the northeastern United States, genetic drift (random loss of rare genotypes) before migration into Europe could have reduced the diversity to a single clonal lineage. The mechanism of transport into Europe is also unknown, but movement of infected tubers via trans-Atlantic shipping is certainly feasible. It

thus seems likely that most of the investigations on *P. infestans* from the 1840s to the 1970s were performed on a tiny range of the genetic diversity in the species. Certainly, the classic studies of Crosier and van der Zaag fit the current understanding of the characteristics of the US-1 clonal lineage (13,78).

## Recent Migrations

One hundred fifty years after the first appearance of late blight in the United States and Europe, there are new migrations, and it is logical to ask why there was not evidence of migrations between the mid-nineteenth and late twentieth centuries. There are several reasons: deserts and oceans that limited natural dispersal of *P. infestans*, limited long-range rapid transport of people and produce until recently, limited production of potatoes in central Mexico with limited international trade of Mexican potatoes until the latter part of the twentieth century, and limited international trade in tomatoes between Mexico and other countries until the latter twentieth century. Infected potato tubers probably provide the most efficient means for long-distance transport of *P. infestans*, and potatoes have become a much more important crop in Mexico during the latter part of the twentieth century. Thus, there is now greater opportunity for infected potatoes to be produced in Mexico. Transport of potatoes in the late 1970s may have been the means by which an exotic population was introduced to Europe (59). While the United States does not import potatoes from Mexico, tomato imports are significant, and infected tomato fruits can easily be transported via air freight. Presymptomatic infected tomatoes packed in Mexico would become symptomatic at the destination and would likely be discarded. Because *P. infestans* can sporulate profusely on infected tomatoes (Fig. 1), aerial dispersal from discarded tomatoes to production fields of tomatoes or potatoes is certainly possible.

The first hint of change in the European population of *P. infestans* was reported by Hohl and Iselin in 1984 (41), who found isolates with the A2 mating type in Switzerland. This report startled scientists all over the world, because dogma had been that all isolates outside Mexico were the A1 mating type. This report was followed by many others, first from Europe but subsequently from many other locations, confirming the occurrence of isolates with A2 mating type as early as 1980 (27). It now seems likely that there have been secondary migrations to South America from Europe (22). Detection of a unique A2 mating type clonal lineage in Japan and Korea (47,57) probably indicates a separate migration.

Changes in populations of *P. infestans* in the United States and Canada were signaled by reports at the beginning of the



1990s, when Deahl et al. reported A2 mating types and isolates with metalaxyl resistance (15,16). Subsequent analysis revealed that these isolates were very recently introduced to the United States and Canada from Mexico (30,36,38).

Several factors enabled scientists in the United States and Canada to rapidly accept the concept of migrations of *P. infestans* into the United States and Canada in the 1990s. First, unambiguous markers were available to identify clonal lineages of *P. infestans*. Second, reports of worldwide migrations had been published and were widely accepted (73). Third, several laboratories around the world were now interested in the population genetics of *P. infestans*. Fourth, most early analyses in North America were concentrated at the USDA in Beltsville, Maryland, and at Cornell. This concentration enabled these laboratories to develop continent-wide understanding of changes in populations.

From studies at both Beltsville and Cornell, it was clear that recent immigrant strains were nearly all resistant to metalaxyl (16,38). Metalaxyl had been the only systemic late blight fungicide available in the United States and Canada, and it had excellent after-infection efficacy (23). The exotic strains were overwhelmingly resistant whether or not metalaxyl had been used in the production region in the United States or Canada from which they were obtained, so they were probably already resistant to metalaxyl when they were introduced (38). Although the introduced strains were nearly monomorphic for metalaxyl resistance, the previous dominant strains remained sensitive (30,38).

The possibility that immigrant strains might be more aggressive is a concept that has been considered seriously only within the last few years. Other factors were initially thought to be responsible for severe late blight. For example, during the early 1980s, the severe late blight in Europe was attributed to metalaxyl resistance in the resident pathogen population (14) or to weather that was especially favorable to the disease. The relative prevalence of new strains was not known until the 1990s (73). Thus, when immigrant genotypes of *P. infestans* were first detected in the United States and Canada in the early 1990s, it was not immediately expected that these would worsen late blight. The locally severe epidemics could be attributed to wet weather and the occurrence of metalaxyl-resistant strains. While metalaxyl resistance would clearly create a need for changes in management, and the potential for sexual reproduction provided by the A2 mating type created opportunity for long-term changes, it was not generally expected that these strains had increased aggressiveness and that this would exacerbate late blight.

Selection by R-gene resistant cultivars appears to have played no role in the pre-

dominance of recently introduced strains. In the United States and Canada, the vast majority of potato production area is planted to cultivars with no known R-genes. Despite this, the exotic strains contain characteristics enabling them to overcome many R-genes (37). In western Europe, there are more cultivars with R-genes, but still the majority of acreage is planted to cultivars without R-genes. Again, as in the United States and Canada, the exotic strains in Europe also can overcome many R-genes (20,76). Thus, the exotic strains on both continents contain unnecessary virulences.

### Characteristics of *P. infestans* Populations

The appearance of exotic strains has stimulated comparisons among them and between the old and new strains. Because only a few lineages have been dominant in the United States and Canada, it has been possible to characterize them individually (Table 1) and then to associate specific epidemiological characteristics with individual clonal lineages. These comparisons have provided insight into disease management and have emphasized that population structures may differ from one location to another.

In most locations in the United States and Canada (with a few important exceptions), epidemic populations have been composed of a single clonal lineage. For example, when an epidemic in a small field of potatoes in upstate New York in 1987 was analyzed for population diversity, all 38 isolates were of the US-1 clonal lineage (Table 1) (30). Even over large regions, there can be homogeneity of isolates. In 1993, an epidemic of tomato late blight in rural, central New York involved hundreds

and perhaps thousands of home gardens in five different counties (Fig. 2). A subset of the samples was sent to Cornell, and all were of the US-7 clonal lineage (Table 1). Predominance by a single clonal lineage also characterized epidemic populations in the Columbia Basin of Washington and Oregon (56). Important exceptions have been reported along the Pacific Coast (10,36), where sexual reproduction appears to have contributed to the diversity of the *P. infestans* population (36).

Pathogen populations contain very low diversity where sexual reproduction has not yet had an impact. In the United States and Canada, where most local populations consist of a single clonal lineage (either A1 or A2), there has been no opportunity for sexual reproduction. In Japan and Korea, where both A1 and A2 lineages have been reported, the inability of oospores to germinate (58) apparently prevents diversification of the very simple pathogen population structure (47). In Ecuador, epidemic populations have consisted almost exclusively of single lineages (22). Although two lineages have been reported in Ecuador, both are A1, and host preferences apparently maintain separation. One lineage (EC-1) occurs commonly on potatoes, but another (US-1) occurs primarily on tomatoes, even when potatoes and tomatoes are interplanted in the same field (G. A. Forbes, *personal communication*).

In contrast, sexual reproduction appears to have a role in locations containing diverse populations. In northern Europe, epidemic populations of *P. infestans* can be very diverse (19,20), and there is evidence of sexual reproduction of *P. infestans* (19,75). Oospores have been shown to survive the winter in the Netherlands (19), and local populations in the Netherlands and Poland fit the expected structures of

**Table 1.** Clonal lineages of *Phytophthora infestans* detected in the United States and Canada. US-1, US-6, US-7, and US-8 have been locally predominant, and US-8 has been nationally predominant since 1995

Lineage	Mating Type	Metalaxyl sensitivity <sup>a</sup>	Pathogenicity <sup>b</sup>	<i>Gpi</i> <sup>c</sup>	<i>Pep</i> <sup>c</sup>	Dates <sup>d</sup>
US-1	A1	S	P(t)	86/100	92/100	? - (1993)
US-6	A1	(R) <sup>e</sup>	P-T	100/100	92/100	1980s - 93
US-7	A2	R	P-T	100/111	100/100	1992 - ?
US-8	A2	R	P	100/111/122	100/100	1992 - ?
US-11	A1	R	(P-T)	100/100/111	100/100	1994 - ?
US-17	A1	R	T(?)	100/122	100/100	1996 - ?

<sup>a</sup> S = sensitive, R = resistant.

<sup>b</sup> Pathogenicity refers to pathogenic specialization. Some lineages are primarily pathogens of potatoes (P), and others are pathogens of both potatoes and tomatoes (P-T). The US-1 clonal lineage contained a few individuals that were pathogenic on both potatoes and tomatoes, but most were pathogenic mainly on potatoes [P(t)]. The US-17 lineage has been detected in the field only on tomatoes to date but causes lesions on potatoes in the lab (C. D. Smart, *personal communication*).

<sup>c</sup> *Gpi* and *Pep* identify genotypes at the *Glucose-6-phosphate isomerase* and *Peptidase* loci, respectively.

<sup>d</sup> Dates refer to the time during which these lineages were common. The US-1 clonal lineage seems rare since 1993. The US-6 lineage has declined in frequency since 1993.

<sup>e</sup> Some isolates of US-6 have been sensitive to metalaxyl.

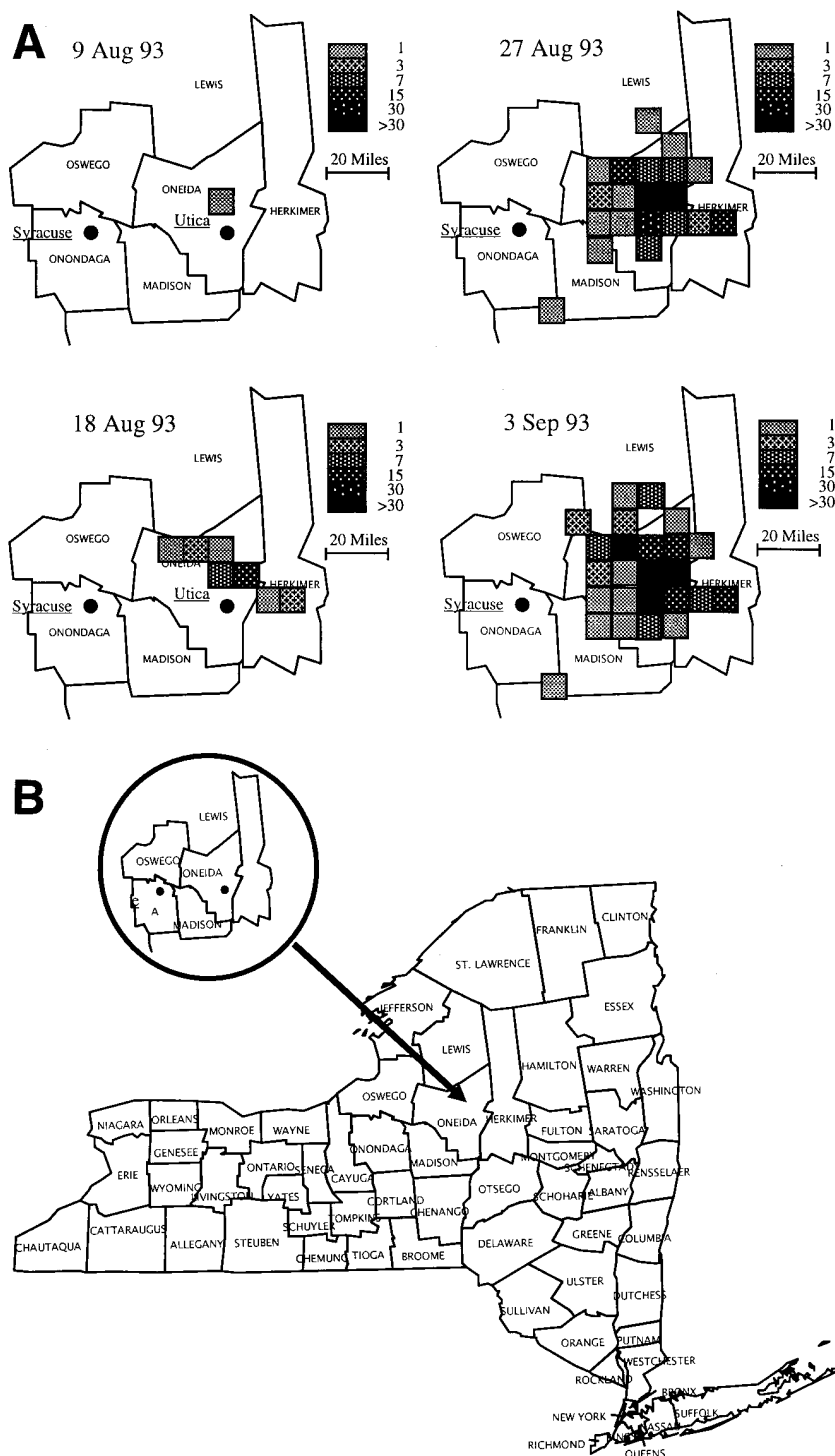
sexually reproducing populations (20,75). However, the importance of oospores in initiating epidemics relative to other sources of the pathogen is yet unknown. Thus, some European situations have some

similarity to central Mexico, where sexual reproduction is thought to have occurred for a long time and epidemic populations are very heterogeneous. For example, among 75 isolates from a single field in the

Toluca Valley, the majority had unique genotypes (54). Thus, population structures of *P. infestans* can differ dramatically among locations; predictions concerning population behavior need to be based on the correct population structure.

The common occurrence of monomorphic populations in much of the United States and Canada, combined with our newly acquired ability to detect individual lineages, has enabled important new insights. One insight concerns the role of residential populations of *P. infestans*. For example, an implicit assumption by many scientists, including the authors, was that resident populations of *P. infestans* were important in most of the United States and Canada. However, we have now seen situations where a resident population had little impact relative to the introduced population. In several New York counties, the clonal lineage responsible for disease changed radically from 1992 to 1993 to 1994 (Fig. 3), indicating either the absence of a residential population during those years or that the residential population was not important to subsequent epidemic populations. Another insight is that clonal lineages can differ from each other in important ways. The differences include pathogenicity factors (host preference, specific virulence, aggressiveness, fungicide sensitivity) (37,38,45,52) and ecological factors (responses to physical factors such as temperature) (E. S. G. Mizubuti and W. E. Fry, *unpublished*). If the differences can be documented and if the lineages can be detected easily, then management actions can be tailored to the lineages found in each field. Fortunately, during the early 1990s, the most common lineages were distinguishable via *Gpi* genotype. A simple technique for detecting *Gpi* genotype directly from sporangia from diseased tissue provided a very rapid (sometimes a few hours) preliminary assessment of the particular lineage involved (34). Knowledge of lineage enabled lineage-specific management options. For example, the indigenous strain (US-1) is sensitive to metalaxyl, and when this lineage is responsible for disease, metalaxyl can be used successfully to suppress disease.

The occurrence of new lineages with different pathogenicities and ecological characteristics means that diversity of epidemiology of late blight in the United States and Canada will now be greater than in the past. This prediction may seem odd for a pathogen that has been considered highly variable (e.g., consult recent reviews and books on late blight [3,39,42,67]). However, most historic analyses of diversity involved pathogenicity characteristics, and it seems that genes governing these traits are highly variable (37,76). Previous reports of temperature and moisture effects on *P. infestans* (13,78) are very consistent with characteristics of the US-1 clonal lineage, but not with all



**Fig. 2.** Dynamics (A) of an epidemic of tomato late blight in upstate rural New York (B) in 1993. Home gardeners in a several-county area were affected by tomato late blight in 1993. The maps identify the minimum cumulative number of phone calls and samples received by the county extension offices. Extension personnel estimated that many additional gardens were affected, and the event was widely reported in local newspapers. All samples investigated were the US-7 lineage (36). The origin of the epidemic is not certain but may have involved a single greenhouse and infected/infested transplants. Subsequent aerial dispersal of sporangia led to an intense regional epidemic. Interestingly, commercial potatoes and tomatoes in the area were unaffected – perhaps because they had been sprayed with fungicide. (Maps by Kiyoshi Ishiguro and Kenneth P. Sandlan)

other lineages (E. S. G. Mizubuti and W. E. Fry, *unpublished*). The details of differences are now under investigation, and if they are discovered to be of sufficiently large effect, there will need to be adjustments in disease management.

### Heightened Threat

After several years of observation and investigation, it is now clear that the exotic strains are a greater threat in the United States and Canada to potato and tomato production than was the previous dominant lineage (US-1). Newspaper reporters came to this conclusion very quickly, and it turns out that although their headlines may have been dramatically overstated, the overall message was correct.

Three exotic lineages (Table 1) contributed to the heightened threat during the early 1990s. The first was the US-6 lineage (Table 1) (A1, very pathogenic to tomatoes and usually, but not always, resistant to metalaxyl [38,52]). It was first detected in the Pacific Northwest but eventually also in Florida and California (30). However, this genotype was rare after 1993. Next was the US-7 lineage (A2, pathogenic on tomatoes, and metalaxyl resistant), which was detected in three states in 1992, eight states in 1993, five states in 1994 (36), and sporadically since. It was this lineage that caused the tomato late blight epidemic depicted in Figure 2. The lineage that has created the most problem and notoriety is the US-8 lineage (A2, very pathogenic on potato foliage and tubers, and metalaxyl resistant [45,49]). In 1992, this lineage was detected only in a single county in north central New York (Fig. 4), and in 1993 only in Maine. However, in 1994, US-8 was detected in the southeastern U.S. growing regions and throughout the north-eastern and midwestern growing regions (Fig. 4). In 1995, US-8 was detected in western growing areas, and by 1996 it was distributed into most production regions, including eastern Canada (Fig. 4). Although some additional genotypes and lineages have been detected (Table 1), it seems that as of early 1997, the US-8 lineage is still the most widely distributed, dominant, and problematic. Most of the recent dominant lineages are resistant to metalaxyl, and suppression of an initiated epidemic is now very difficult (38).

The rapid distribution of exotic strains, but particularly of US-8, is due to several factors. First, transcontinental shipment of potato seed tubers is an effective method of long-distance movement. Although most infected seed tubers probably do not lead to infections in the foliage, some do (78), and when tons of potatoes are shipped, there can be successful transport of strains of *P. infestans*. Second, US-8 is especially pathogenic to foliage and tubers (45,49). Tuber infections result from sporangia produced on infections in the foliage, so

that US-8 is particularly likely to cause tuber infections. Third, aerial dispersal of *P. infestans* sporangia leads to local dispersal. This combination of factors has enabled rapid distribution of strains.

The greater aggressiveness of US-8 compared with US-1 (faster lesion expansion rate) is sufficient to explain the field observation that the exotic strains (espe-

cially US-8) require more fungicide for adequate suppression of late blight than was required to suppress disease caused by the US-1 lineage (45). The new strains are not more resistant to protectant fungicides than the old strains (45). When differences between US-1 and US-8 were analyzed with the aid of a complex computer simulation model of late blight (9), more pro-

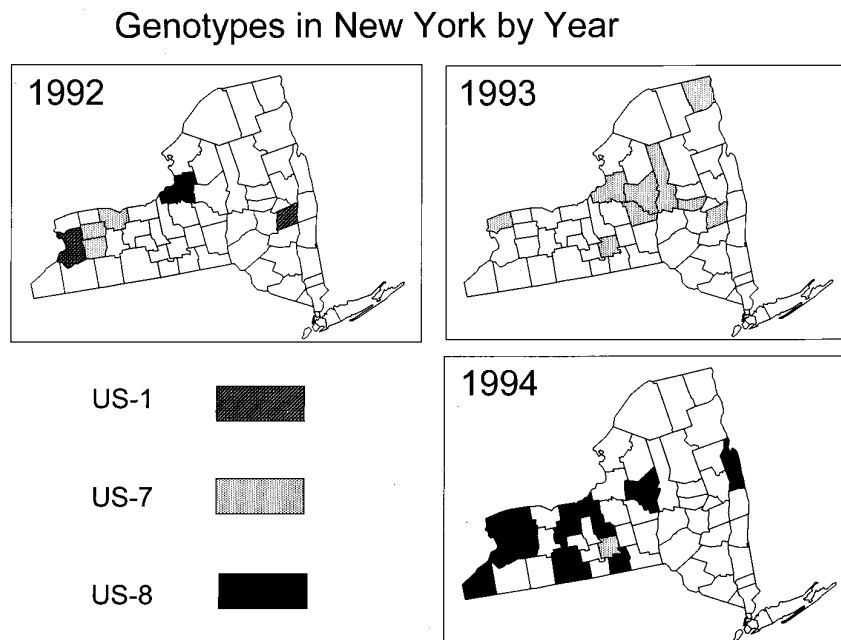


Fig. 3. Occurrences of clonal lineages of *Phytophthora infestans* in New York in 1992, 1993, and 1994 (36). In no case was there more than one lineage found in any county. While some counties are represented by one or two samples, others had 10 to 20 samples.

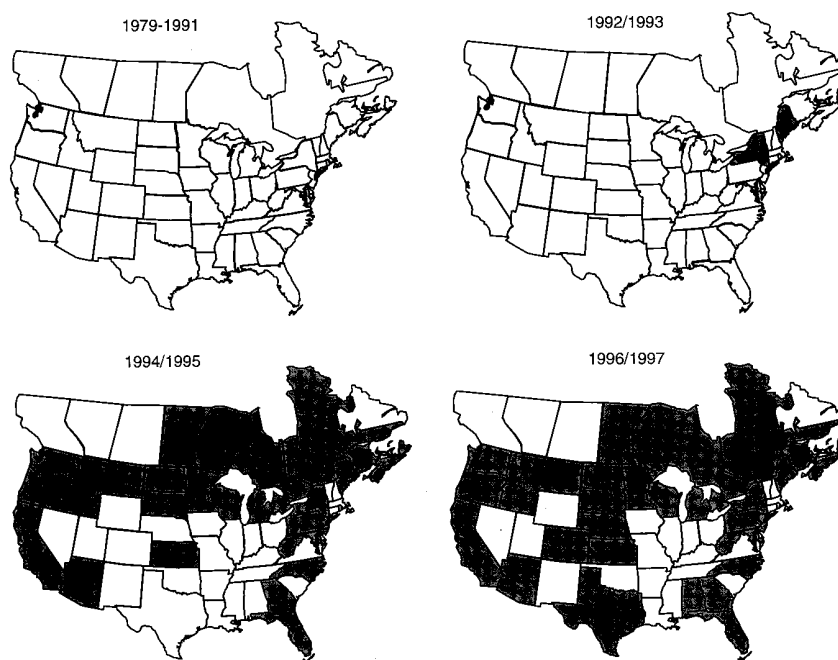


Fig. 4. Occurrences of the US-8 clonal lineage of *Phytophthora infestans* from 1992 into 1997 in the United States and Canada. Data are from a variety of sources including (36,57; J. Hill, H. Schwartz [CO], A Pavlista [NE], and W. E. Fry and S. B. Goodwin, *unpublished results*). (Map by Kenneth P. Sandlan)

tectant fungicide was required to suppress epidemics caused by US-8 than those caused by US-1. The differences were in the range of an additional 25% (measured as increased frequency of application) needed to suppress US-8-induced epidemics (45).

Some of the recent immigrant strains are particularly pathogenic to tomatoes and have caused severe tomato late blight (Fig. 1). The California Tomato Board identified late blight as its top priority for research in the mid-1990s. The lineages US-6 and US-7 were demonstrated to be especially

pathogenic on tomatoes from observation, greenhouse, growth chamber, and field studies (51,52). Other lineages (perhaps US-11 and US-17) appear to share some of these characteristics.

### Impacts of New Strains

There have been many impacts of the new strains, including psychological stress on individual potato growers dealing with devastating epidemics, reduced yields, increased tuber blight, heightened fear of the new late blight even in its absence, and economic hardship caused by dramatically

increased fungicide costs. Although the aggregate economic impacts of the exotic strains are not yet known completely, some regional data have been published.

One economic analysis for the 1995 epidemic in the Columbia Basin of Washington and Oregon has been published recently (44). Late blight was not a serious threat in this production region during the previous several decades (44). Instead, early blight, caused by *Alternaria solani*, was a more persistent concern. However, during the early 1990s, late blight was discovered. Initially, the US-1 and US-6 lineages dominated (56), but in 1994, US-8 was detected in low frequency. In 1995, weather favored late blight and the disease was severe. The overwhelming majority of isolates analyzed from the severe epidemics of 1995 were the US-8 clonal lineage (43,56). The average number of fungicide sprays increased from 2.5 in 1994 (little late blight) to 10 in 1995 (severe late blight) at a cost of \$450/ha or a total cost estimated at \$30 million for 1995 (44).

Comprehensive economic impact data from other regions are not yet available. However, plant pathologists throughout the United States and Canada are aware of severe losses to individual producers, and anecdotal data abound. For example, late blight was especially severe in the northeastern United States in 1994. Destruction of foliage was common in many locations, and this was accompanied by severe tuber blight (Fig. 5). Most of the severe problems were caused by the US-8 clonal lineage. In early September 1994, we visited one farmer who had just discarded 300 metric tons of potatoes—only 8 days after harvest. These stored potatoes were decomposing rapidly due to bacterial soft rot in the late-blighted potatoes. Late blight in the tubers provided entry points for soft-rotting bacteria. The value of potatoes in that single storage was estimated at more than \$30,000. The same grower had potatoes in other storages that also were affected. Unfortunately, some of his neighbors had even worse losses, and decaying potatoes were discarded without regard to proper disposal (Fig. 6) to prevent the infected tubers from serving as overwintering sites for the pathogen. In a different production region, one grower with approximately 200 ha of potatoes suffered an 80 to 85% crop loss in 1994 and subsequently was forced out of business. Potato production in the northeastern United States is sometimes locally concentrated such that this single crop can contribute noticeably to the economy of an entire county. Some of these counties were severely affected in 1994. The severity of loss suffered in 1994 in the northeastern United States was usually not repeated in this production region in 1995, because 1995 was drier and because growers had intensified their late blight management activities.



Fig. 5. Tubers decaying due to a variety of causes after being initially infected by *Phytophthora infestans*.



Fig. 6. Decaying late blight-infected potato tubers discarded along a roadside into a deep gully. The potatoes were discarded within a few days of harvest because of rapid decay. Tubers were harvested under wet, warm conditions and were infected with *Phytophthora infestans*. The high moisture, warm temperatures, and late blight infections enabled soft rotting bacteria to initiate a meltdown of the entire storage. The magnitude and speed of decay quickly saturated the grower's ability to discard cull tubers appropriately, and this was a hasty response to a terrible situation. Recommended methods to dispose of infected tubers are to bury them in deep pits covered by at least two feet of soil, or (in northern latitudes) to spread the tubers on the soil surface and allow them to freeze during the winter.



## Responses by the Scientific and Agricultural Community

After the 1994 season, plant pathologists in the eastern and midwestern United States agreed that a multistate action plan was necessary. Some components of the plan have been implemented already. Effective communications among and within states were initiated. Several e-mail networks were established so that scientists could have some knowledge of where late blight had been detected and which clonal lineages were involved. Several states initiated communication systems so that the occurrence of weather favorable to late blight could be quickly and effectively relayed to growers. Another component was to petition the U.S. EPA for emergency registrations (section 18) for fungicides with some systemic activity. The Departments of Agriculture of several states became involved in pressing for these emergency registrations. The petitions were delivered in February 1995 and were granted in time for the majority of the 1995 season. Eventually these emergency registrations were granted to 23 states for 1995 and to 25 states by 1997. Clearly the regulatory personnel agreed with scientists, growers, and state departments of agriculture that the situation was a bona fide emergency.

The US-8 lineage continued to be distributed over more growing regions in 1995 (Fig. 4), while plant pathologists were developing an accurate understanding of the pathogenicity characteristics of the immigrant strains. Accurate knowledge of the aggressiveness of the immigrant strains was confounded by weather favorable for disease and by metalaxyl resistance. Thus, although we knew that the immigrant strains posed some additional risk, it was difficult to certify the causes and to quantify the added risk. Predictions that overstated the potential danger of the immigrant strains would cause unnecessary fear and perhaps economic stress by demanding too much fungicide. Predictions that understated the potential would put growers at risk. The compromise was to publicize the existence of immigrant strains as widely as possible.

While growers in locations already affected by exotic strains understood the significance of the warnings, growers in locations not yet affected by immigrant strains were sometimes unconvinced that the new strains posed any heightened risk. Growers who did not heed the warning or who denied the risk were often those who were particularly vulnerable. Unfortunately, weather favorable for late blight sometimes enabled the immigrant strains to overcome the management efforts even of growers who attempted to take precautions. It appeared that the most effective factor in causing growers to view immigrant strains with extreme caution was for

them to see the devastation they caused (especially the US-8 lineage).

Plant pathologists have been on the front line of this issue, warning growers and consultants of the heightened risks posed by the immigrant strains and investigating improved management activities. Massive educational efforts have been launched. Programs have been redirected to develop knowledge that will enable more effective management of the new strains, and new investigations have been initiated. By late 1996, plant pathologists had developed a good understanding of the problems posed by the immigrant strains, and the appropriate extension and research activities were being defined. A series of important recommendations was endorsed in January 1997 in Tucson, Arizona, at a North American Potato Late Blight Workshop (Sidebar). The next steps are to put those recommendations into effect. The recommendations include implementation of fundamental disease management activities, new initiatives in epidemiology and plant breeding, and more basic research into pathogen biology and host-pathogen interactions (Sidebar).

In addition to the practical impacts, the recent migrations have provided startling insight into the potential for changes in the population biology of pathogens. The concept of colonization of a whole continent by a single clonal lineage in a few years

seems startling to persons who are accustomed to thinking of indigenous populations. The magnitude of the event is staggering to those most familiar with it, and the mechanisms of population replacement or displacement are not completely known. The potential of *P. infestans* to be transported on seed potato tubers, tomato transplants, and tomato fruits has been re-emphasized.

Knowledge that global migration of exotic strains is possible and that subsequent colonization of large areas can occur within just a few years raises many questions. Some of these are:

- If migrations can occur so easily with rapid continental colonization, what is the role of quarantines?
- Have similar migrations and population displacements of other pathogens gone undetected for lack of appropriate genetic markers?
- Are there other, even more aggressive, genotypes of this oomycete in Mexico or other locations that may soon be introduced into the United States?
- Will sexual reproduction and oospore production alter the epidemiology of late blight in the United States and Canada?
- Is the US-8 lineage so aggressive that it will be eliminated because it destroys its host too rapidly?
- Does mating type influence pathogen fitness?

### Selected (consolidated) recommendations from the North American Potato Late Blight Workshop, Tucson, Arizona, 8 to 11 January 1997. Original recommendations collated by W. Brown (Colorado State University).

#### Identification

- Develop rapid, reliable methods for species and lineage identification.

#### Biology/Genetics of *Phytophthora infestans*

- Learn factors influencing oospore development and germination.
- Identify genotypic diversity in North America and characterize population structures.
- Develop additional genetic markers and locate specific genes on chromosomes.

#### Pathology

- Learn mechanisms of pathogenesis.
- Learn biotic factors influencing tuber infections.

#### Epidemiology

- Quantify biological/environmental factors that influence epidemic development.
- Improve disease forecasting systems.
- Quantify influences of diverse pathogen genotypes on epidemic development.
- Improve understanding of environmental factors influencing tuber blight.
- Quantify factors influencing dispersal of *P. infestans*.
- Determine contribution to epidemic development of different types of lesions.

#### Disease Management

- Determine relative importance of various sources of initial inoculum.
- Quantify effects of different fungicides and application techniques on epidemic development.
- Evaluate potential of reduced tolerances in certified seed to limit epidemics.

#### Host Resistance

- Develop resistant cultivars through traditional breeding.
- Improve understanding of field resistance.
- Investigate molecular and biochemical bases of resistance.
- Attempt to create host resistance through emerging and novel technologies.

#### Education

- Organize and publicize educational materials concerning late blight.
- Develop comprehensive programs for integrated management of late blight.
- Develop an electronic network for distributing educational materials, real time status reports of late blight occurrences, and warnings of potential outbreaks.

## What's Next?

The re-emergence of late blight in the United States and Canada as well as throughout the world illustrates that even old diseases can become serious again. Mutation or migration brings new characteristics to a pathogen population. Our experience with *P. infestans* is not necessarily unique, and it illustrates that changes might be possible in other pathogen populations. There may still be pathogens of which we are not aware, and these might be introduced to susceptible host crops. Thus, plant pathologists need to maintain a vigilance for new pathogens and for signals of changes in populations of old ones. Characterization of pathogen populations with robust neutral genetic markers appears to be a useful first step in developing that awareness.

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**William E. Fry**

Dr. Fry is professor of plant pathology at Cornell University, Ithaca, New York. He received his B.A. in chemistry from Nebraska Wesleyan University in 1966 and his Ph.D. from Cornell University in 1970. He joined the faculty at Cornell in 1971. He served as chairman of the department from 1981 to 1995; he was president of the American Phytopathological Society from 1995 to 1996. His research interests include the ecology, genetics, and pathology of *Phytophthora infestans*, with emphasis on epidemiology and disease management. His teaching has included epidemiology, disease management, and introductory plant pathology.



**Stephen B. Goodwin**

Dr. Goodwin is a research plant pathologist with the USDA-ARS at the Department of Botany and Plant Pathology, Purdue University, West Lafayette, Indiana. He received his B.S. in botany from Duke University in 1981 and his Ph.D. in genetics from the University of California, Davis, in 1987. His work on *Phytophthora infestans* was initiated at Cornell University in 1987. His current research interests, in addition to a continuing interest in late blight and *P. infestans*, include biology and pathology of *Septoria nodorum* and the biology of the interaction between wheat and *S. nodorum*.