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Historical and Recent Migrations of *Phytophthora infestans*: Chronology, Pathways, and Implications

The 1984 report of A2 mating types of *Phytophthora infestans* (Mont.) de Bary in western Europe (20) was the first indication of new and dramatic developments in populations of that fungus. This discovery stimulated plant pathologists all over the world to analyze local populations, since previously only the A1 mating type had been detected outside of central Mexico (Fig. 1). The analyses of a large number of dispersed local populations indicated, surprisingly, that the changes were not restricted to western Europe but, rather, were worldwide (Fig. 2) (3,10,23,26,36,41). The recent worldwide changes in populations most certainly result from migration. Indeed, migration has played an essential role in the entire history of potato late blight. In this article we illustrate that role. To provide context, however, we first present background concerning the basic biology/pathology of *P. infestans*, the genetic tools used to investigate populations of *P. infestans*, and the characteristics of the source population of *P. infestans*.

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Background

Biology/pathology. The biology of *P. infestans* is typical of all oomycetes, but the pathology resembles that of a downy mildew (a specialized group of oomycetes) rather than that of most other *Phytophthora* spp. The oomycetes appear to have closer affinities with algae and higher plants than with ascomycetes and basidiomycetes (4,21). They are diploid and coenocytic, lack chitin in the cell walls, and produce biflagellate zoospores. For *P. infestans* there are two mating types, A1 and A2. Sexual structures (antheridia and oogonia) are induced only in the presence of the opposite mating type, and genetic fusion results in oospores (Fig. 3). Oospores are probably survival structures, whereas infections of foliage (Fig. 4) or tubers (Fig. 5) are initiated by asexual sporangia (Fig. 6) and/or zoospores. Individuals in asexual populations most commonly survive as mycelium in infected tubers. Hosts include the economically important crops of potatoes and tomatoes, as well as a large number of tuberous *Solanum* spp.

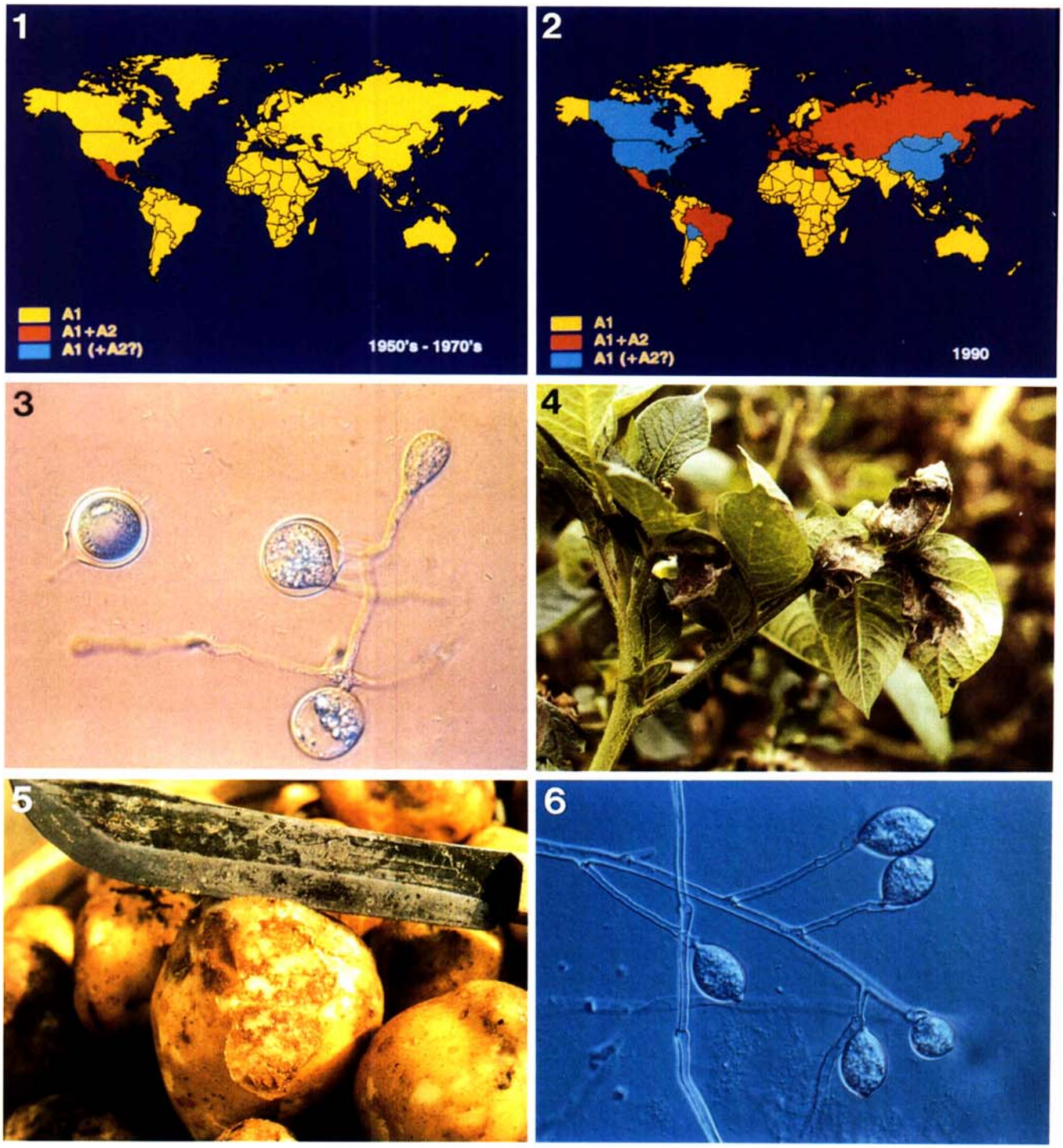
Genetic and phenotypic markers. Characterizations of *P. infestans* populations have relied on a series of markers (Table 1), which have contributed significantly to our understanding of the population genetics of *P. infestans*. (For our purposes, a population is those isolates from a geographically defined unit.) Mating type provided the first indication of major changes in *P. infestans* populations. Allozyme alleles at glucose-6-phosphate isomerase (*Gpi*) (Fig. 7), malic enzyme (*Me*), and peptidase (*Pep*) loci provided the first genetic evidence of diploidy in *P. infestans* and enabled the first comparisons of genetic diversity in populations from different locations (35,42). More recently, nuclear DNA fingerprinting (Fig. 8) has enabled much greater resolution of population structures (16,18). Alleles at the allozyme loci and most bands revealed by one fingerprint probe (RG57) are inherited according to Mendelian expectation (16,18,27, 38,39). Polymorphisms in the mitochondrial genome have also provided useful markers (5,15). These markers are presumably neutral and can be scored unambiguously.

Some phenotypic markers have also been helpful. Although the genetic bases of all specific virulence phenotypes have not yet been determined, the genetic control of some has been, and these traits have been important historically in terms of genetic characterizations and of disease management (12). Additionally, metalaxyl resistance is an agriculturally important trait that can be used to characterize isolates.

Source population. Since the discovery that the A2 mating type was common in central Mexico (14,28), evidence has been accumulating that the central highlands of Mexico (Figs. 9–12) represent the center of origin of *P. infestans* (29). Several convincing lines of evidence support this hypothesis: 1) This region historically has been the only one in which

both mating types occur in equal frequency (12,29,42); 2) the population of *P. infestans* in central Mexico (particularly in the Toluca Valley) is especially diverse for virulence characteristics (25, 29,31,43); and 3) populations of *P. infestans* in central Mexico are remarkably diverse for neutral markers. This is the only location in which all of the known allozyme alleles have been detected, as well as the only location containing all of the known DNA fingerprint bands (18; J. M. Matuszak et al, *unpublished*).

The high level of diversity in central Mexico is exemplified in a temporal analysis of genotypes in an epidemic on unsprayed potatoes in the Toluca Valley (24). Isolates were collected at a single location in the valley over a 2-week period in 1988. Twenty-five isolates from each of three sampling dates—11, 19, and 26 July—were analyzed for mating type, allozyme genotype, and DNA fingerprint. Unique genotypes (i.e., detected



Figs. 1–6. (1) Known distribution of the A2 mating type of *Phytophthora infestans* from the 1950s to the late 1970s. (2) Known distribution of the A2 mating type of *P. infestans* in 1990. The A2 mating type was firmly established in Europe, western Asia, eastern Asia, the Middle East, and Brazil. Sporadic or anecdotal reports of A2 mating types in the United States, Canada, and China indicate probable establishment of the A2 mating type in these locations. (3) Dormant and germinating oospores, approximately 35–45 μm in diameter, of *P. infestans*. (Courtesy R. C. Shattock) (4) Sporulating lesions of potato late blight on potato foliage. (5) Lesions on potato tubers infected by *P. infestans* produce a dry rot that is "grainy" or "corky." (6) Sporangia, approximately 25–35 μm in length, of *P. infestans*. (Courtesy R. V. James)

only once in the entire period) were accumulated steadily. At each sampling date, at least half of the individuals had unique genotypes (24). This is entirely consistent with previous observations that most isolates from the Toluca Valley were genetically unique (18). This diversity occurred despite the predominance of asexual reproduction during the epidemic. The obvious conclusion is that there is an extremely large number of distinct genotypes of *P. infestans* in the Toluca Valley.

Other lines of evidence are consistent with the idea that the population in the Toluca Valley reproduces sexually whereas populations elsewhere have been asexual. *Gpi* alleles were present in Hardy-Weinberg equilibrium in the Toluca Valley but not in other populations (11,18,42). The occurrence or predominance of only one mating type in most other locations in the world suggests that asexual reproduction is the only means of propagation in those locations.

Coevolution in the *P. infestans*–*Solanum* pathosystem probably involved many *Solanum* species in the highlands of central Mexico. This area is a secondary center of diversity for the genus *Solanum* (6,19) and has many endemic tuber-bearing species of *Solanum*. In contrast, domesticated potatoes (*Solanum tuberosum* subsp. *tuberosum*) originated in the South American Andes and were not intensively grown in the Toluca Valley until the 1950s (29). Many of the wild Mexican *Solanum* species have resistance to *P. infestans*. For example, *S. demissum*, which is common in the central highlands, including the Toluca Valley (Figs. 9 and 11), has been the source of many specific resistance genes (R-genes) (47).

The 1840s Migration

The first global migration of *P. infestans* about which we have any information occurred in the 1840s. A new disease of potatoes (probably potato late blight) was reported from locations near Philadelphia beginning in 1843 (30,40). With time, reports of this disease came from locations increasingly distant from the initial locations, and by 1845 the disease was detected throughout the maritime provinces of Canada and the northeastern United States and into the Midwest (Fig. 13). The first reports of a new potato disease in Europe came from Belgium in June 1845, and the disease was reported at increasing distances from Belgium during the summer of 1845 (1) (Fig. 14). By mid-October 1845, most of Ireland was affected and the disease had spread east into Germany (1). Two paths of migration seem most plausible (Fig. 15): The fungus may have been transported from Mexico to the United States and then to Europe or it may have been transported directly from Mexico to Europe.

Once introduced to Europe, *P. infestans* could have been distributed to much of the rest of the world via the international trade in seed potatoes (7). The cool climate and resulting relatively low incidence of aphid vectors of potato viruses have aided potato growers in northern Europe to produce potatoes with low virus incidence. These have been used as seed potatoes by potato growers in South America, Africa, the Middle East, and Asia. Unfortunately, some infected tubers may have been planted in these areas because not all tubers infected by *P. infestans* are readily detectable. Thus, the potato seed trade has probably provided an efficient mode of migration for *P. infestans*.

Recently, the relationships among *P. infestans* populations (>250 isolates) from 10 different countries on five continents have been investigated (12,17). Because there is no definite evidence for migrations during the interval between the 1840s and the 1970s, these populations may have been separated from the source population in central Mexico until relatively recently. A single genotype predominated (frequency >50%) in all samples from locations not affected by the 1970s migration. This genotype and those very closely related (differing only by loss of one of the 14 fingerprint bands or loss of one of the four allozyme alleles) constitute a clonal lineage that dominated extra-Mexican populations until recently (17). In some samples, this historical lineage has been the only one detected (Table 2). The genotype of this historical clonal lineage is mating type A1, 86/100 for *Gpi*, 92/100 for *Pep*, and the DNA fingerprint shown in Figure 16.

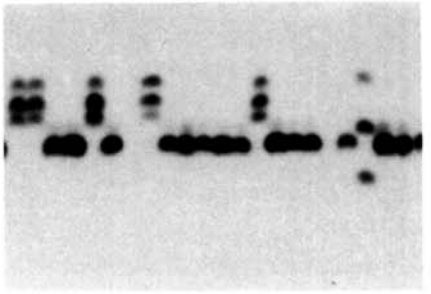


Fig. 7. Allozyme gel stained for glucose-6-phosphate isomerase, a dimeric enzyme; the direction of migration is from bottom to top. If a diploid organism is heterozygous (*AB*) for electromorphs of the isozyme, three active species of the dimeric enzyme can result (AA, AB, BB). This gel illustrates five individuals that were heterozygous (111/122), 14 individuals that were homozygous (100/100), and one individual that was heterozygous (86/122).

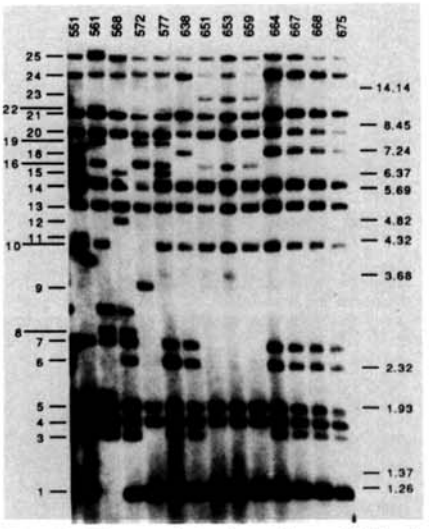
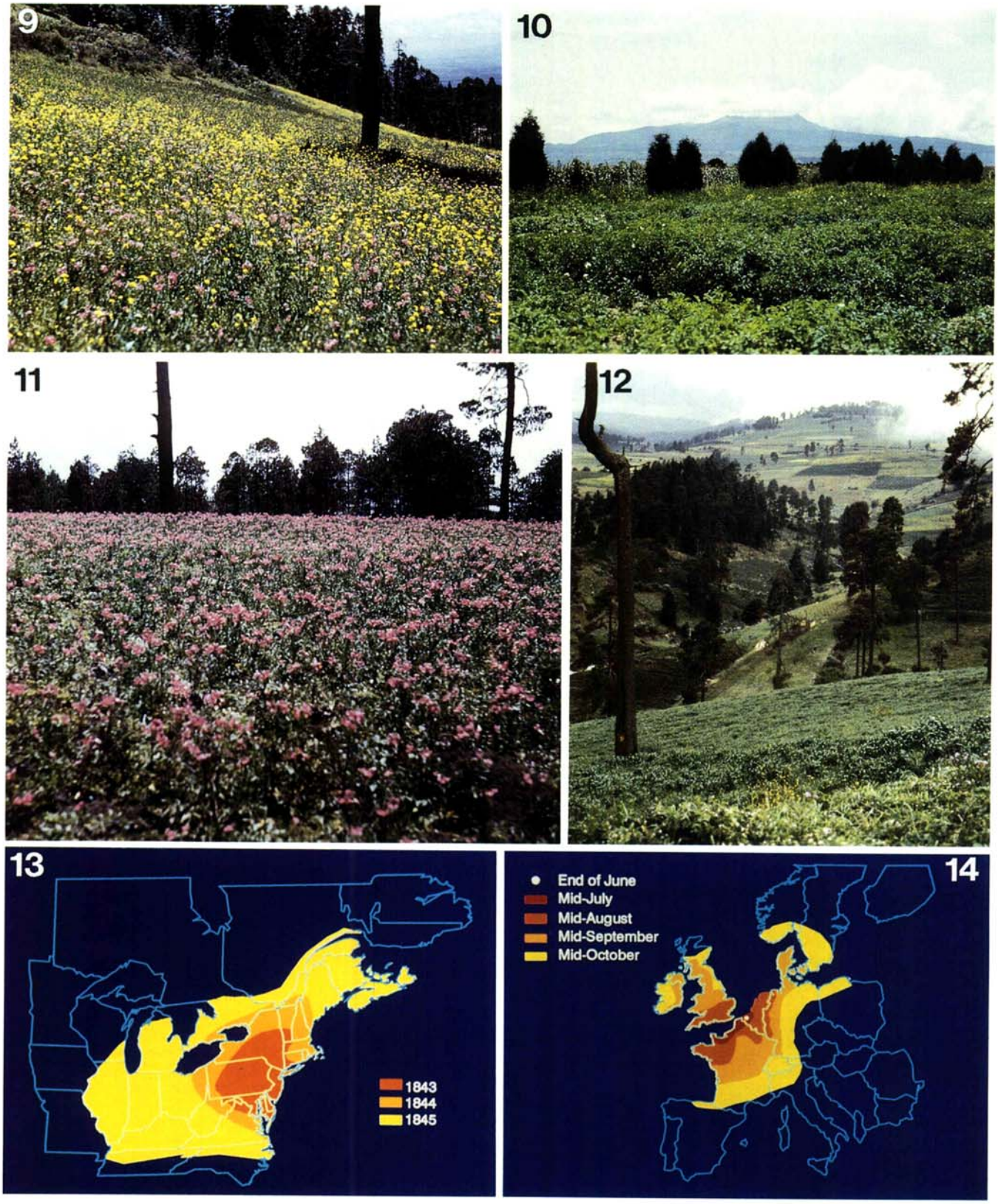


Fig. 8. Fingerprint of nuclear DNA of *Phytophthora infestans*. Total genomic DNA of *P. infestans* was digested with the restriction enzyme *Eco*RI, subjected to Southern analysis, and probed with ^{32}P-labeled clone RG57. The direction of migration is from top to bottom. Band numbers are designated on the left, molecular weights (kb) on the right, and isolates along the top. Isolates in the 500s were collected from central Mexico, and each has a distinct fingerprint—a situation common in collections from the Toluca Valley in central Mexico. Isolates in the 600s were collected from northwestern Mexico, and only two have distinct fingerprints, which is consistent with a clonal population structure. (From Goodwin et al [18])

Table 1. Genetic and phenotypic markers used to characterize populations of *Phytophthora infestans*

Marker	Description
Mating type	A1 and A2
Allozymes	Three allozyme loci: glucose-6-phosphate isomerase (*Gpi*) (seven alleles), peptidase (*Pep*) (five alleles), and malic enzyme (*Me*) (two alleles)
Nuclear DNA fingerprinting	Probe RG57 reveals up to 30 loci in *P. infestans* populations
Mitochondrial DNA	At least five different mitochondrial DNA haplotypes are known
Fungicide resistance	Isolates can be classified as resistant or sensitive to the fungicide metalaxyl
Virulence	At least 11 different specific compatibilities have been described



Figs. 9–14. (9) Wild *Solanum* spp. in the highlands of central Mexico near the Toluca volcano, in the 1950s. (Courtesy J. S. Niederhauser) (10) Commercial potato production in the Toluca Valley; the Toluca volcano is in the background. (11) *S. demissum* near the Toluca volcano, in the 1950s. (Courtesy J. S. Niederhauser) (12) Typical potato production in the highlands of central Mexico near the Toluca volcano in the 1980s. (13) Distribution of a new disease of potatoes (probably late blight) in the United States from 1843 to 1845. The disease was first detected near Philadelphia in 1843 and by 1845 had extended into the Midwest of the United States and into the maritime provinces of Canada. (After Bourke [1]) (14) Distribution of potato late blight during the summer of 1845 in western Europe. The disease was first detected in Belgium at the end of June and by mid-October had been reported from as far west as Ireland and as far east as Germany. (After Bourke [1])

These data are consistent with the concept that *P. infestans* populations in Eurasia, Africa, and South America consisted of a single clonal lineage until very recently. (Populations in the United States and Canada contained this historical clonal lineage plus a few others [17], and populations in central Mexico were sexual—without clonal lineages.) If these historical collections represent descendants of the first migration, then the path of migration was likely from locations of greater diversity to locations of lesser diversity, i.e., from Mexico to the United States, from the United States to Europe, and from Europe to Asia, Africa, and South America.

The 1970s Migration

A second migration, including the A2 mating type, probably occurred out of Mexico in the late 1970s. The A2 mating type has now been reported from countries representing all continents except Antarctica and Australia (Fig. 2) (12, 23,26,33,34,36). In Europe, the A2 mating type was not collected before 1981 (20). The characteristics of the newly migrating population can be deduced by comparison with historical isolates, i.e., those collected before 1977, or before detection of the A2 mating type (37). The initial characteristics, deduced on the basis of specific allozyme alleles, were: mating types A1 and A2; *Gpi*, 100/100 and 90/100; *Pep*, 100/100 (96/96) and 83/100; and RG57 fingerprint, >35 types (37; A. Drenth and W. E. Fry, *unpublished*). Prior to detection of the A2 mating type, the 90 allele for *Gpi* and the 83 allele for *Pep* had not been detected in Europe (37). Subsequently, DNA analyses have corroborated conclusions based on allozyme data (17).

The mechanism of migration was probably via infected potato tubers. Apparently, large quantities of potatoes for domestic consumption were imported from Mexico to western Europe in the late 1970s (29) (Fig. 17). If some infected tubers were stored and subsequently planted or if they were discarded in a compost pile or cull pile, the fungus would likely have survived winter in the tuber. In spring, some infected tubers would produce foliage, which would then support growth of the pathogen. Sporangia from this infected tissue would then be dispersed to nearby potato or tomato plants. If this was the mechanism, then many distinct strains were probably involved, because the new migrating population was more diverse than the previous population (Fig. 18) (*A. Drenth*, unpublished; L. S. Sujkowski, *unpublished*). It again seems likely that subsequent global migrations were via infected seed tubers associated with the seed trade between western Europe and countries on other continents (Fig. 17). This new population now occurs in much of the world (Table 3). However, there are a few locations in which representatives of this new "European" migration have not yet been detected, including the Philippines, Taiwan, the United States, Canada, and perhaps Peru (Table 2). We expect that representatives of the new migrating population will soon be dispersed to all locations that have seed potato trade with western Europe.

Temporal analysis of *P. infestans* populations in Europe indicated that the new population displaced the old population in only a few years (37) (Fig. 19). Independent analyses of populations from Germany, the Netherlands, and Poland all led to this conclusion (Fig. 19). Similarly, when isolates from Wales collected in 1978 were compared with isolates collected in 1987, results were consistent with population displacement (R. T. Folkertsma, R. C. Shattock, and D. S. Shaw, *personal communication*). The phenomenon in Europe appears not to be a melding of two populations via hybridization. Recombinants of "new" and "old" genotypes have not been discovered, and allozyme alleles characteristic of the "old" lineage are absent from the current population. Displacement of the old clonal lineage by the new migrating population indicates (by definition [32]) that the newly migrating population is more fit than the population consisting of the old clonal lineage. Increased fitness might possibly be associated with increased aggressiveness—thus explaining at least partially the heightened visibility of late blight in Europe and the Middle East during the 1980s.

Although the mechanisms of displacement have not yet been determined, initial observations indicate that they differ from R-gene selection (which occurs when only compatible isolates can persist on a race-specific, resistant cultivar). Selection by R-genes has been common in the "old" lineage of *P. infestans* and has been described repeatedly (22,44,45). However, R-gene selection is an inadequate explanation for this population displacement because: 1) most cultivars in western Europe do not contain R-genes, 2) there were adequate virulences in the "old" population, and 3) there has been no major change in the R-gene composition of European cultivars coincident with displacement. Instead, the phenomenon with *P. infestans* is probably most similar to the displacement described for *Ophiostoma ulmi* by

Table 2. Locations and dates in which only the same single clonal lineage of *Phytophthora infestans* was detected[a]

Location	No. of isolates analyzed	Date collected or acquired
China	6	1981
Europe	3	1977–1981
Japan	4	1984
Peru	34	1985–1987
Philippines	28	1989–1990
Taiwan	3	1991

[a] From Goodwin and Fry (17) and *unpublished.*

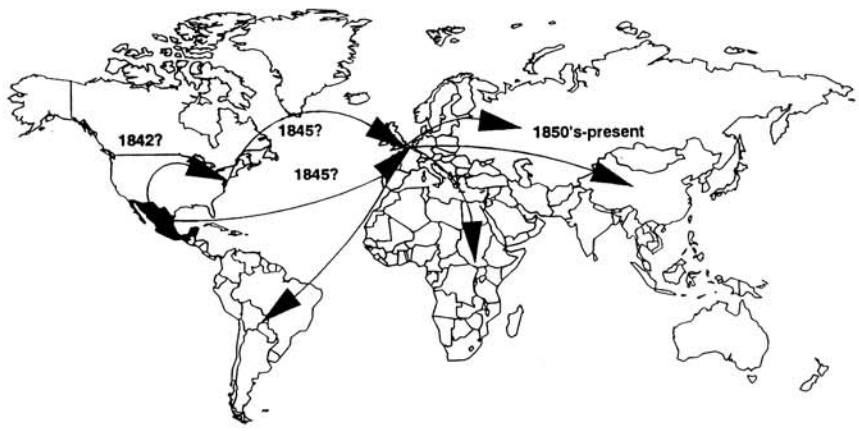


Fig. 15. Possible paths of migration of *Phytophthora infestans* in the 1840s and 1850s.

Fig. 16. DNA fingerprints of worldwide isolates (excluding those from central Mexico) with the 86/100 genotype for *Gpi*.

Brasier (2), in which a recently invasive aggressive strain has displaced the previously dominant (but nonaggressive) strain in western Europe. In contrast with the situation with *P. infestans*, however, the aggressive strain of *O. ulmi* is morphologically distinct from the nonaggressive strain.

Displacement of *P. infestans* populations may also be occurring on other continents. Reports from Japan indicate that the frequency of the old single clonal lineage has been declining recently (27), and representatives of a "new" population predominated in a recent collection of *P. infestans* isolates from many areas in Korea (Y. J. Koh et al, *unpublished*).

One practical implication of migration may concern sensitivity/resistance to the oomycete-specific fungicide, metalaxyl. This fungicide has been especially effective in suppressing many diseases caused by oomycetes, and therefore the occurrence of resistance has been particularly troublesome (8). Metalaxyl resistance was not widespread among isolates belonging to the "old" population. It may be that metalaxyl resistance has been problematic because of migration and selection of resistant individuals—in addition to or instead of de novo mutation and selection.

The 1980s Migrations: Canada, Mexico, and the United States

Until very recently, populations of *P. infestans* in the United States consisted of the old clonal lineage plus a few other clonal lineages (12,13,17), but that situation may be changing. Populations from tomatoes in Florida in the spring of 1991, from potatoes and tomatoes in western Washington in 1990, and from potatoes from eastern Washington in 1991 were predominantly of a single clonal lineage (Fig. 20) previously undetected in the United States or Canada (12,13; S. B. Goodwin et al, *unpublished*). Unfortunately, this genotype is resistant to metalaxyl (10,12)—which may have increased the difficulty of disease suppression. Additionally, Deahl et al (9) reported two A2 isolates in the late 1980s—one from Pennsylvania in 1987 and one from British Columbia in 1989. Others were detected in summer 1992 (S. B. Goodwin et al, *unpublished*).

The occurrence of a single clonal lineage in the geographic extremes of the continental United States demonstrates the pathogen's capacity for rapid, long-distance migration and illustrates the need to consider migration in the development of a management strategy.

Any of a number of migration mechanisms may have been operating. One suggestion is that the newly migrating population arrived in the Pacific Northwest on potted tomatoes (D. Ormrod, *personal communication*). Because the fungus can survive in tomato fruits (46), it seems plausible that individual isolates could also have traveled via infected tomato fruits. The connection of Florida tomatoes with California tomatoes, with tomatoes from Mexico, or with potatoes and tomatoes from the Pacific Northwest is not yet clear.

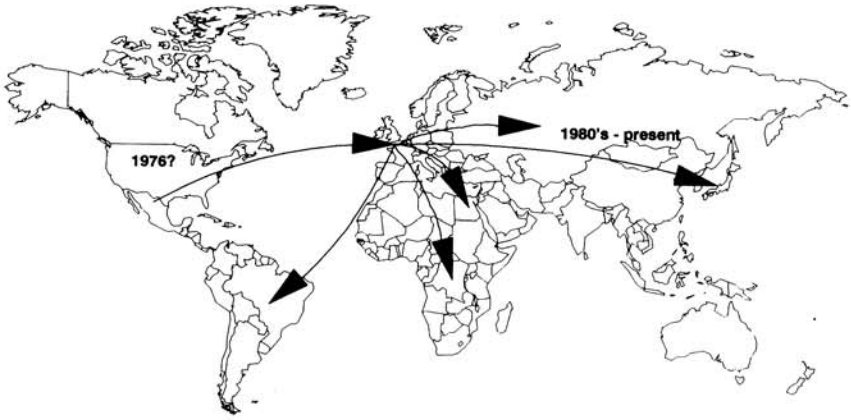


Fig. 17. Possible paths of migration of *Phytophthora infestans* in the late 1970s and 1980s.

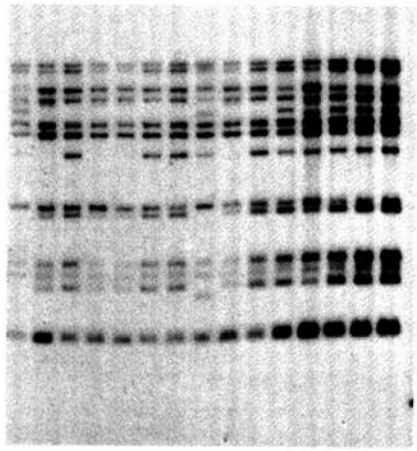


Fig. 18. DNA fingerprints of 15 isolates collected from tomatoes in community gardens in Wageningen, Netherlands. All isolates are 100/100 for *Gpi*, but five different fingerprints are discernible. (A. Drenth and W. E. Fry, *unpublished*)

Table 3. Locations in which new "European" genotypes of *Phytophthora infestans* have or have not been detected in recent collections

	New genotypes detected			New genotypes not detected		
Continent	**Country**	**When collected**	**Reference**	**Country**	**When collected**	**Reference**
Asia	Japan	1985 to present	26,27	Philippines	1990, 1991	Y. J. Koh, *unpublished*
	Korea	1991	Y. J. Koh, *unpublished*	Taiwan	1991	Y. J. Koh, *unpublished*
Africa	Egypt	1984	36			
	Israel	1980s	12			
	Rwanda	1980s	17			
Europe	Germany	1981–1989	S. Daggett, *personal communication*			
	Ireland	1989	P. W. Tooley, *unpublished*			
	Netherlands	1981–1989	37			
	Poland	1988–1991	37			
	Switzerland	1981	20			
	United Kingdom	1984	R. T. Folkertsma, *personal communication*			
South America	Bolivia	1990	S. B. Goodwin, *unpublished*	Peru	1985, 1986	17,42
	Brazil	1988	3,17			
	Colombia	1990	A. T. Dyer, *unpublished*			
	Ecuador	1989	A. T. Dyer, *unpublished*			

The migration of a new population of *P. infestans* into and throughout the United States, its widespread distribution, the occurrence of metalaxyl resistance in that population, and the diversity of mechanisms of dispersal of this fungus, all emphasize that persons interested in suppressing late blight of potato or tomato should be alert for new outbreaks of the disease and occurrences of fungicide resistance. If the greater fitness of the new population results from greater aggressiveness, then we also need to be vigilant for evidence of rising rates of disease and more damaging epidemics of late blight.

Probable Effects of Sexual Reproduction

With the migration of the A2 mating type to locations all over the world (now coincident with the A1 mating type in many locations), it is important to investigate the probable effects of sexual reproduction. If it occurs, sexual reproduction will generate a larger number of genotypes, but this phenomenon alone seems unlikely to exacerbate rates of failure of R-genes in most parts of the world. Asexual populations of the fungus have not lacked plasticity of virulence (22,47). Consequently, R-gene resistance has not contributed substantially to disease control in western agriculture. However, the genetic diversity generated by sexual reproduction might lead to more aggressive genotypes. Additionally, if oospores are formed in potato and tomato agroecosystems throughout the world, and if oospores function effectively as initial inoculum, the epidemiology of late blight outside Mexico may change dramatically. When populations were strictly asexual, the fungus had no survival structure to allow it to overwinter in soil apart from the host. It now seems possible (via extrapolation from other *Phytophthora* species) that oospores can survive for months or years in soil. Thus, if *P. infestans* reproduces sexually, oospores might be another very important source of initial inoculum for late blight of tomato and potato.

Although sexual reproduction is expected in locations where both mating types occur, there is still no clear evidence that it influences the population structure in locations outside Mexico. For example, analyses from populations in the Netherlands (where the A1 and A2 mating types had coexisted for at least 9 years) provided no clear evidence for the contribution of sexual reproduction (11). New data may soon alter that conclusion, however. For example, analyses of *P. infestans* populations in Poland may indicate sexual recombination (L. S. Sujkowski et al, *unpublished*). Collections of *P. infestans* from 1986 to 1991 in Poland contained a high frequency of unique genotypes that resembled the diversity in the Toluca Valley of central Mexico (L. S. Sujkowski, *unpublished*). Thus, our prediction, which is similar to that of many other scientists, is that evidence of sexual recombination is likely to emerge in the near future.

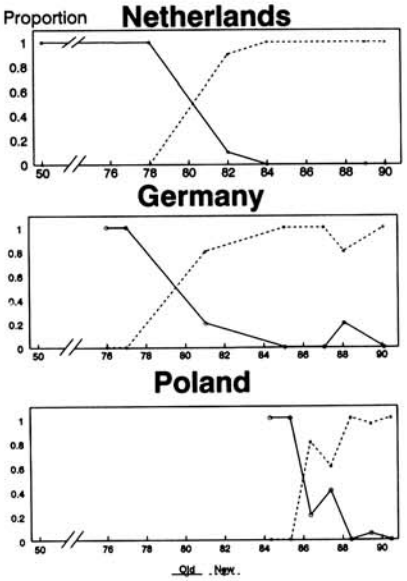


Fig. 19. Temporal dynamics of "old" and "new" genotypes of *Phytophthora infestans* in the Netherlands, Germany, and Poland. Old genotypes are 86/100 for *Gpi* and apparently represent a single clonal lineage (see Figure 16). New genotypes are 90/100 or 100/100 for *Gpi* and are genetically diverse, representing a large number of clonal lineages (see Figure 18). Data for the Netherlands from Spielman et al (37); for Germany from S. Daggett, C. D. Therrien, and E. Goetz (*personal communication*); and for Poland from L. S. Sujkowski et al (*unpublished*). Sample sizes for the Netherlands, Germany, and Poland were about 200, 76, and 200, respectively.

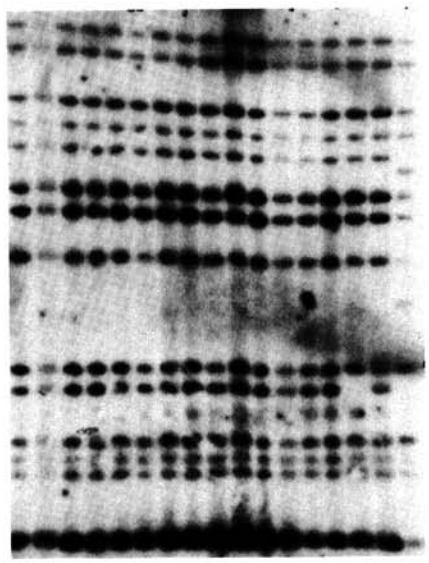


Fig. 20. DNA fingerprints (with probe RG57) of 17 isolates from the state of Washington in 1990. Sixteen have the same fingerprint. This pattern was identical to that detected in isolates from Florida in 1991 (12; S. B. Goodwin, *unpublished*).

Next Steps

Increased investigation and education are needed. To establish the most effective control strategies for late blight, we need to know much more about dispersal and genetic variation in local populations of *P. infestans*. Studies of the epidemiology of A1 and A2 isolates are in progress in Europe (A. Drenth, *personal communication*; R. C. Shattock, *personal communication*). Cultivar resistances and fungicides effective against the old population should be evaluated against the new populations. But, perhaps most important, persons involved in managing late blight of potato or tomato must understand the continued need for integrated disease management. All factors that reduce population size and pathogen population growth rates need to be considered in the management program. An integrated approach will include sanitation (elimination of sources of inoculum), resistant cultivars, increased effort to exclude novel pathogen genotypes, and judicious use of appropriate fungicides.

Acknowledgments

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