CHAPTER 10

WORLDWIDE MIGRATIONS, HOST SHIFTS, AND REEMERGENCE OF *PHYTOPHTHORA INFESTANS*, THE PLANT DESTROYER

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INTRODUCTION

Phytophthora species are oomycete plant pathogens that are responsible for devastating diseases on a wide range of food and ornamental crops, natural vegetation, and forest trees worldwide (Erwin and Ribeiro, 1996). Indeed the name *Phytophthora* is derived from the Greek phytó, "plant," and phthorá, "destruction"; thus, members of this genus are often referred to as "the plant destroyer." They represent an emerging food security threat, in large part due to increases in plant movement via international trade (Brasier, 2008). *Phytophthora infestans*, the causal agent of late blight, exemplifies this threat; it was the first species in the genus described and left a path of devastation on potato in its wake in the United States, Ireland, and Europe in the 19th century (Berkeley, 1846; Bourke, 1964; deBary, 1876). Movement of infected potato tubers led to the potato famine epidemics of the 19th century that resulted in widespread human hunger, disease, and, ultimately, death of two million people in Ireland. The pathogen causes a destructive foliar blight of potato and also infects potato tubers and tomato fruit and is still wreaking havoc more than 160 years after the Irish famine (Fry, 2008; Hu et al., 2012).

Late blight is the most important biotic constraint to potato production worldwide and is major threat to food security, particularly in the developing world where use of fungicides is often uneconomical (Anderson et al., 2004; Pennisi, 2010). Worldwide losses due to late blight on potato and tomato exceed $7 billion annually (Haverkort et al., 2008). Evolution of strains varying in sensitivity to fungicides and novel pathotypes of *P. infestans* continue to challenge the sustainable production of potatoes. The pathogen has also reemerged as a significant disease threat to the organic tomato industry in the United States where synthetic chemicals are not used (Stone, 2009). In 2009, late blight epidemics of potato and tomato in the eastern United States were the worst in recent history due to widespread inoculum distribution and weather conducive for disease (Hu et al., 2012; Moskin, 2009).

This chapter provides an overview of the emergence of potato late blight in the 19th century and the evolutionary position, life

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history, and the population biology of *P. infestans*. Worldwide migrations of the pathogen and the role of host shifts in the evolution of species in the genus are also discussed.

EMERGENCE OF LATE BLIGHT IN THE UNITED STATES AND EUROPE

The potato blight struck with a vengeance in the United States in 1843 before it appeared on the European continent and in the British Isles. The potato crop in the United States was a good one in 1842, but the situation changed in 1843. One account documented in the *Annual Report of the Commissioner of Patents in the United States* stated that the "potato yield was nearly 50 percent less owing to a rot which seized them before the time for taking them out of the ground" (Ellsworth, 1843). A second report in the same document stated "The potato crop has been attacked. The cause is generally attributed to the peculiarity of the weather" (Ellsworth, 1843). The late blight epidemic began in 1843 in the United States in a five-state area starting from the ports of New York and Philadelphia (Bourke, 1964; Ellsworth, 1843). The first appearance of the disease near the two port cities of Philadelphia and New York suggested an introduction via imported tubers. Potatoes and bat guano, which were used as fertilizer, were being shipped at that time from Peru in South America into many ports in both the United States and Europe to improve the stock of seed potatoes that had declined from *Fusarium* dry rot. These potatoes probably provided the source of inoculum for the first disease outbreaks.

In Europe, late blight was first noticed on the coast of Belgium in the Courtrai area in June of 1845 (Bourke, 1964). By mid-July, late blight had moved southward into Flanders and parts of the Netherlands and France. By August, it had spread into the lower Rhineland, Switzerland, and to southern England. In 1845, on the 16th of August, it was seen in the Isle of Wight and on the 23rd of August in the South of England. The potato blight made its way into Ireland by the 7th of September, 1845, and later in the year to Scotland (Bourke, 1964).

The farmers and learned men and women of the time immediately started to speculate as to the cause of the "evil" that was now widely affecting potatoes grown everywhere. James Teschemacher, a member of Boston's Natural History Society, associated a minute fungus with the lesions on the potato plant (Teschemacher, 1844). There was by no means a consensus on the subject of the cause of the potato blight, which was attributed to the weather, insects, the wrath of God, and the minute "fungus." Speculation began about the cause of the disease and lively discussions ensued in the *Gardeners Chronicle* in the United Kingdom: "That minute and rapidly propagating fungi have been observed on the affected potato plants, there is no reason to doubt. But when we know, from other facts, that such appear on plants after they are dead or diseased, and not when they are alive and healthy, we are justified in affirming that we are yet ignorant of the cause of the malady in question" (MacKenzie, 1845). Others wrote, "It is certain that a fungus appears in the leaves, stems, and tubers of the plants which have been attacked, but it is uncertain how far the fungus is the cause or the consequence of the disease - how far it is to be considered as a parasite upon the living potato, or as a mere devourer of its dying parts" (Johnson, 1845).

The pathogen was described and named by C. Montagne in France and in the United Kingdom by the Reverend M. J. Berkeley in 1846 as *Botrytis infestans* (Berkeley, 1846; Montagne, 1845). Morren had observed the disease in Belgium in 1844 and called it *Botrytis devastatrix* and

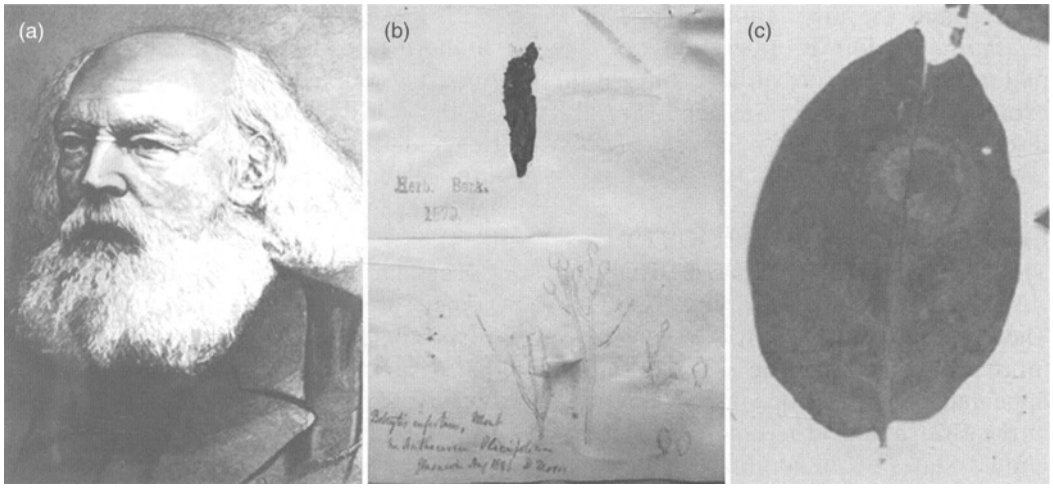


Figure 10.1 (a) M. J. Berkeley, the British mycologist who described the pathogen *Botrytis infestans* (later named as *Phytophthora infestans*) on potato in 1846; (b) sporangia and sporangiophores of *P. infestans* drawn on a famine-era archival specimen label by David Moore, Glasnevin, Ireland, that was sent to Berkeley for confirmation; (c) herbarium specimen with leaf lesion caused by *P. infestans* collected by Krieger in Germany in 1888. See color insert.

presented a paper to the Royal Society of Lille (Morren, 1844). Libert had also called the pathogen *B. devastatrix* (alternate spelling *vastatrix* or *devastrix*) Lib. 1845, but Berkeley chose to use the species name *B. infestans*. In 1847 and 1852, Unger and Caspary, respectively, considered it in the genus *Peronospora* (Unger, 1847). deBary initially accepted this opinion (Cline et al., 2008; deBary, 1863).

Miles J. Berkeley (Fig. 10.1a) and later deBary resolved the debate by documenting that the "fungus-like" organism *P. infestans* was the cause and not the consequence of the disease. David Moore of Ireland drew pictures of the sporangiophores and sporangia of the pathogen and sent specimens to Berkeley, who named the pathogen *B. infestans* (Fig. 10.1b). The work on late blight by Berkeley predated work by Louis Pasteur on the germ theory and clearly documented that a microbe could cause disease (Berkeley, 1846) and was a major contribution to both the development of the science of plant pathology and the germ theory. In 1876, deBary elucidated the life cycle of the pathogen and based on sporangial development and sporangiophore characteristics and changed its name from *Peronospora* to *P. infestans* (deBary, 1876).

EVOLUTIONARY POSITION AND PHYLOGENETIC RELATIONSHIPS OF *PHYTOPHTHORA* SPP.

The genus *Phytophthora* now encompasses more than 100 species that are classified within the diploid, algae-like Oomycetes in the Kingdom Stramenopila (Adl et al., 2005; Bauldauf, 2003; Gunderson et al., 1987). The number of *Phytophthora* species described has increased rapidly due to intensive monitoring of plants and waterways in the past 10 years for *Phytophthora ramorum*, the cause of sudden oak death (Cline et al., 2008; Rizzo et al., 2002). Oomycetes were once grouped with true fungi (ascomycetes and basidiomycetes) since they share a filamentous habit of growth by mycelium and obtain nutrition

by absorption. However, oomycetes differ from true fungi in many ways and are now placed in a separate kingdom in a distinct branch in the eukaryotic tree of life and are more closely related to brown algae and diatoms than true fungi (Adl et al., 2005).

Based upon sequences of ribosomal genes and the introns associated with them (internal transcribed spacer [ITS] regions), 10 clades in the genus *Phytophthora* were described (Cooke et al., 2000). Subsequently, multilocus sequencing more clearly elucidated phylogenetic relationships among a larger group of *Phytophthora* species (Blair et al., 2008; Kroon et al., 2004; Martin and Tooley, 2003). *P. infestans* is a member of the Ic clade of *Phytophthora* (Blair et al., 2008; Cooke et al., 2000). Other species in this clade include the more distantly related *Phytophthora phaseoli* and *Phytophthora mirabilis*, *Phytophthora ipomoeae*, and *Phytophthora andina* (Flier et al., 2002; Galindo and Hohl, 1985; Oliva et al., 2010).

LIFE HISTORY OF THE PATHOGEN

P. infestans is a hemibiotrophic pathogen that infects host tissue and after a few days produces symptoms. One reason why late blight is such a destructive disease is due to the explosive, polycyclic nature of asexual sporulation that can occur on plant tissue (Fig. 10.2a).

The pathogen produces black water-soaked lesions on potato leaf tissue that produce asexual sporangia (Fig. 10.2a). Tuber infections lead to a purplish brown discoloration of the tissue and eventually the tuber rots (Fig. 10.2b). The pathogen causes brown lesions on the stems, petioles, and leaves of tomatoes (Fig. 10.2c). Brown zonate lesions occur on the fruit (Fig. 10.2d).

Sporangia (Fig. 10.2e) can be dispersed by wind and rain at local and national scales (hundreds of meters) and entire fields can be destroyed in a few days. Late blight is truly a community disease, and

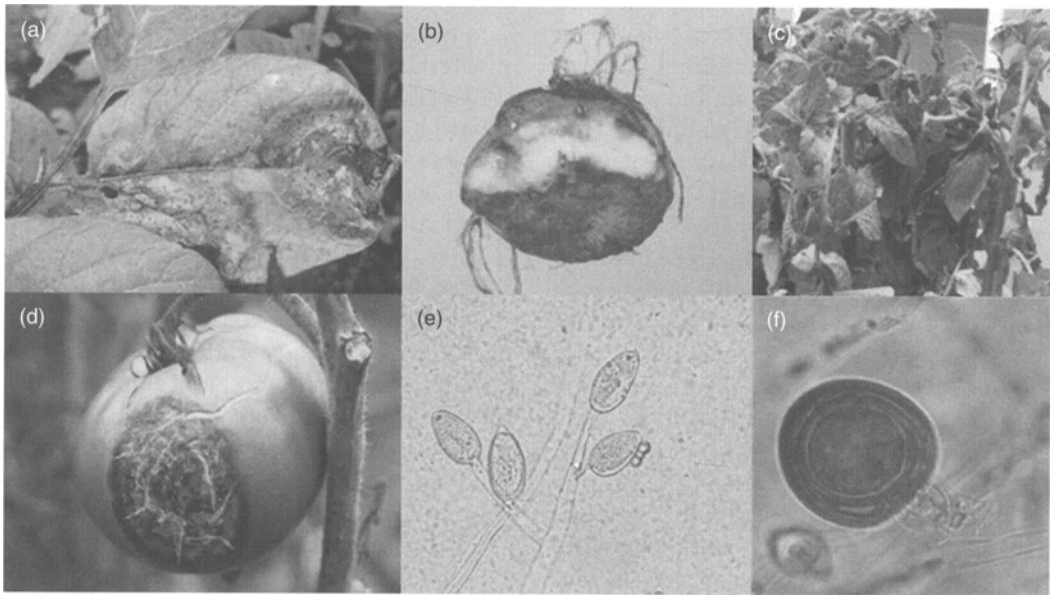


Figure 10.2 Symptoms of late blight caused by *P. infestans* on (a) potato leaf, (b) tuber, (c) tomato stem, (d) tomato fruit, (e) plants in a field, and (f) a tomato transplant. (e) Asexual sporangia borne on compound sporangiophores; (f) sexual oospore produced by fusion of anisogamous gametangia (male antheridium and female oogonia) of opposite mating types (A1 and A2). See color insert.

inoculum in fields left untreated can spread to neighboring fields due to aerial dispersal of sporangia. The pathogen has caused major losses mostly by the unintentional spread on infected plant materials such as potato tubers or tomato transplants that are transported over large areas, as is illustrated by the widespread epidemics of 2009 on tomato in the United States (Hu et al., 2012). The pathogen typically survives from season to season as mycelium in infected potato tubers, volunteer potato plants, or infected culled potatoes that contribute to epidemic development on subsequent crops.

P. infestans is heterothallic and requires two opposite mating types, A1 and A2, for sexual reproduction and formation of the nonmotile sexual oospore (Fig. 10.2f) (Gallegly and Galindo, 1958). Oogonia develop when A1 and A2 mating type isolates are paired. Hormonal heterothallism is involved in the formation of gametangia with one thallus producing a hormone that stimulates gametangia formation of the opposite mating type (Ko, 1988). Each thallus is bisexual and can act as either male or female and induces either oogonia or antheridia formation in the opposite mating type (Judelson, 1997). Studies of the inheritance of mating type show that the A1 types have a heterozygous locus (Aa), while A2 types are homozygous recessive (aa) (Fabritius and Judelson, 1997; Judelson et al., 1995). The position of the mating type locus on a genetic linkage map (Van der Lee et al., 2004) and in genomic clones (Randall et al., 2003) has been identified and in several strains has been linked to genetic abnormalities such as balanced lethality and translocations (Judelson, 1996a,b). The A1 mating-type hormone MH-1 α has been purified and reported to be a diterpene (Harutyunyan et al., 2008; Qi et al., 2005). A second structurally similar mating hormone, MH-2 α, has recently been purified, and both hormones are proposed to be derived from the chlorophyll-related plant hormone phytol (Ojika et al., 2011). This exciting discovery may lead to novel methods to stop sexual reproduction in the pathogen.

Oospores were reported first in Mexico, but the pathogen now also reproduces sexually in the Netherlands, Scandinavia, and Canada (Andersson et al., 2009; Drenth et al., 1994; Gavino et al., 2000). Oospores are not common in the United States and sexual reproduction is rare. The oospore is a thick-walled survival structure and can persist in soils at low temperatures for several years but does not survive well at higher temperatures (Fay and Fry, 1997; Maytoun et al., 2000).

POPULATION BIOLOGY: MIGRATION THEORIES OF *P. INFESTANS* IN THE 19TH CENTURY

P. infestans has four mitochondrial haplotypes (Ia, Ib, IIa, and IIb) (Avila-Adame et al., 2006). The mitochondrial genomes of all the extant haplotypes have been sequenced (Avila-Adame et al., 2006; Carter et al., 1990; Lang and Forget, 1993). Phylogenetic and coalescent analysis revealed that although the type I and II haplotypes share a common ancestor, they form two distinct lineages that evolve independently. Type II haplotypes diverge earlier than type I haplotypes. The type II lineages did not evolve from type I lineages, as previously suggested (Gavino and Fry, 2002). Our data support the hypothesis that all the extant mitochondrial lineages of *P. infestans* evolved from a common ancestor in South America since the most ancient mutations in the four lineages occur there (Gomez et al., 2007).

The center of origin and diversity of the late blight pathogen is proposed to be in Mexico (Fry and Goodwin, 1997; Goodwin et al., 1994b; Reddick, 1939). This hypothesis is based on the fact that in Mexico, (i) both mating types occur;

(ii) host resistance genes are present in wild *Solanum* populations; and (iii) pathogen populations are highly diverse for neutral DNA markers and pathotypes. Isolates of *P. infestans* of the A2 mating type and oospores in infected plant material were first discovered in central Mexico in 1956 (Fry et al., 1992; Gallegly and Galindo, 1958; Goodwin et al., 1994a). Prior to 1980, both the A1 and A2 mating types were reported only in Mexico, while the A1 mating type was reported elsewhere in the world (Fry, 2008; Fry and Goodwin, 1997; Gallegly and Galindo, 1958). This situation changed in the 1980s when the A2 mating type was observed in Switzerland (Hohl and Iselin, 1984).

Mexico has also been proposed to have provided the source inoculum for the late blight epidemics of the 1840s (Fry and Goodwin, 1997; Goodwin et al., 1994b). Genetic analysis of modern worldwide populations of *P. infestans* demonstrated that they were dominated by a single clonal lineage known as the US-1 "old" genotype (mtDNA haplotype Ib) in the mid 20th century (Fry, 2008; Goodwin et al., 1994b). Mexican populations are highly diverse for genotypic and phenotypic markers and thus, Mexico clearly represents a present-day center of diversity of the pathogen. However, since Mexican populations are highly diverse, the pathogen population is proposed to have undergone a genetic bottleneck in the 1840s during the first migration event, thus greatly reducing genetic diversity, in order to explain the dominance of a single "old" clonal genotype (US-1 genotype) in modern worldwide populations prior to the 1980s (Fry and Goodwin, 1997; Goodwin et al., 1994b). Domesticated potatoes were not grown for export in Mexico in the 1840s and tuber blight was not common. It was suggested that the pathogen may have been accidentally introduced by a plant collector (Goodwin et al., 1994b). These authors have provided well-documented evidence for migrations of the pathogen from Mexico after the 1970s (Goodwin et al., 1994a), but no definitive evidence for migrations of *P. infestans* during the interval between 1840 and 1970. The fact that the putative "old" US-1 lineage (Ib mtDNA) has not been widely reported in Mexico, does not support the theory that Mexico provided the source inoculum for the late blight epidemics of the 1840s.

The Mexican "bottleneck" theory of Goodwin and colleagues was challenged by several authors (Abad and Abad, 1997; Andrivon, 1996; Ristaino, 1998; Tooley et al., 1989). A second "hybrid" migration theory was proposed, suggesting that Mexico may represent the center of origin of the disease but that inoculum that caused the 19th century potato famine epidemics originated in Peru (Andrivon, 1996; Bourke, 1964). This theory was based on both historical data and an evaluation of the published population genetic data at the time. A third theory suggests that the Peruvian Andes represents both the center of origin of the disease and the source of inoculum for 19th century epidemics. Evidence to support this theory has been provided by both historical data about disease occurrence (Abad and Abad, 1997; Berkeley, 1846) and population genetic data (Gomez et al., 2007).

WHAT mtDNA HAPLOTYPE CAUSED THE FAMINE AND WHERE DID IT COME FROM?

Nineteenth and early twentieth century scientists collected and preserved potato and tomato leaves infected with *P. infestans*, and specimens exist from the Irish potato famine (Fig. 10.1b,c). Historical specimens have been analyzed to answer questions about the evolution and population biology of the pathogen (Ristaino, 1998, 2002, 2006; Ristaino et al., 2001; May and Ristaino, 2004). The polymerase

TABLE 10.1 Identity of the Mitochondrial DNA Lineage of *P. infestans* in Archival Herbarium Specimens Collected Worldwide

Geographic Region	Year Collected	Number of Specimens	mtDNA Haplotype[a]
Central and South America	1889–1969	18	Ia Ib—Bolivia (1944), Ecuador (1967) IIb—Nicaragua (1956)
North America	1855–1958	88	Ia Ib (1931 and later—potato, tomato, *Solanum sarrachoides*)
Northern Europe	1866–1905	11	Ia
Western Europe	1845–1982	52	Ia
Eastern Europe	1892–1978	7	Ia
United Kingdom	1845–1974	52	Ia
China	1935–1982	10	Ia Ib (1952—potato, 1956—tomato) Ib (1982—*Solanum lyratum*)
Southeast Asia and Australia (Japan, Philippines India, Nepal, Peninsular Malaysia, Thailand)	1901–1987	13	All Ia except: Ib India (1968, 1974—potato) Ib Thailand, (1981—tomato)

[a] Based on PCR methods of Griffith and Shaw (1998).

chain reaction (PCR) has been used to amplify minute amounts of DNA from leaves infected with *P. infestans* from historic epidemics.

The mtDNA haplotypes present in specimens collected during the Irish potato famine and later in the 19th and early 20th century were identified (May and Ristaino, 2004; Ristaino et al., 2001). First, a 100-bp fragment of DNA from ITS region 2 specific for *P. infestans* was amplified from 90% of the leaves tested, confirming infection by *P. infestans* (Trout et al., 1997; Ristaino, 1998, 2001). Primers were then designed that amplify short segments of mtDNA around variable restriction sites that separate the four mtDNA haplotypes (Griffith and Shaw, 1998) and the DNA was sequenced. Surprisingly, 86% of lesions from leaves collected during historic epidemics were caused by the mtDNA haplotype Ia (May and Ristaino, 2004), not Ib (Goodwin et al., 1994b), as previously believed.

Interestingly, both the Ia and IIb haplotypes were found in potato leaves from specimens collected in 1954 and 1956 in Nicaragua (Table 10.1). These data challenge the hypothesis that a single clonal lineage of *P. infestans* existed outside of Mexico since two mtDNA haplotypes were present in samples from Nicaragua. Thus, late blight populations in the mid 20th century in Central America did not consist of a single clonal lineage and pathogen diversity was greater than previously believed.

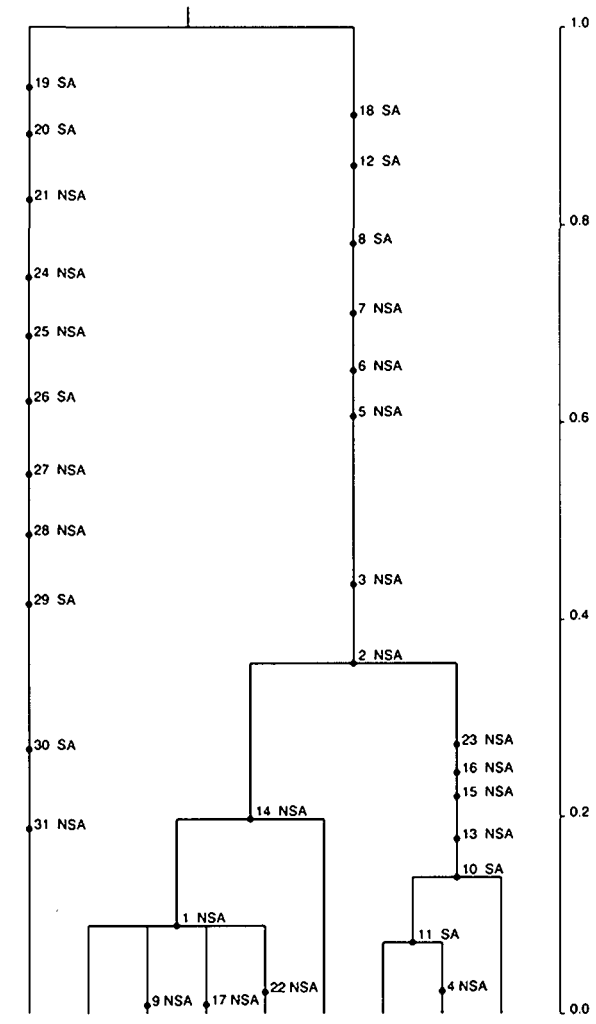
WHEN DID THE mtDNA HAPLOTYPE Ib MIGRATE FROM SOUTH AMERICA?

The Ib haplotype of *P. infestans* was found in several plant samples from herbaria including a sample from Bolivia (1944) and Ecuador (1967) in the mid-20th century (Table 10.1) (May and Ristaino, 2004). We

recently examined mtDNA haplotypes of several hundred samples of *P. infestans* from infected potato and tomato leaves from herbaria collected from the United States between 1855 and 1953. In all cases, haplotype Ia was predominant and found before the haplotype Ib was detected (May and Ristaino, 2004; Ristaino and Lassiter, unpublished data). The Ia haplotype of *P. infestans* was also found earlier in China than other mtDNA haplotypes (Ristaino and Hu, 2009). In contrast, the earliest record of haplotype Ib in China was in 1952 on potato in 1954 and in 1956 on tomato (Beijing). The haplotype Ib still occurs in the Beijing area on tomato (Guo et al., 2010). Modern Chinese populations contain all four haplotypes, suggesting more recent introductions of IIa and IIb haplotypes (Guo et al., 2010). In other areas in Asia, the earliest documentation of haplotype Ia was in Japan in 1901. The haplotype Ia was also found in India (1913), the Philippines (1910), and Russia and Australia (1917). In contrast, haplotype Ib was only found in three samples in India on potato (1968 and 1974) and in Thailand on tomato (1981). All the rest of the herbarium plant samples from other Southeast Asian countries and regions were infected with haplotype Ia (Table 10.1). Thus, our data suggest that the haplotype Ib was dispersed in early to mid-20th century migrations into the United States, China, and Southeast Asia, whereas earliest introductions during the famine-era epidemic in the United States and Europe, no doubt on imported potato tubers, were the Ia haplotype (May and Ristaino, 2004; Ristaino and Hu, 2009).

HISTORIC MIGRATIONS: "OUT-OF-SOUTH AMERICA" MIGRATION HYPOTHESIS

We have assessed the genealogical history of *P. infestans* using multilocus sequences from portions of two nuclear genes (*β-tubulin* and *ras*) and several mitochondrial loci P3 (*rpl14*, *rpl5*, tRNA) and P4 (*Cox1*) from 94 "modern" isolates from South America, Central America, North America, and Ireland in order to test an out-of-South America migration hypothesis (Gomez et al., 2007). Summary statistics, migration analyses, and the genealogy of populations of *P. infestans* for both nuclear (*ras* gene) and mitochondrial loci are consistent with an out-of-South America origin for *P. infestans* rather than an "out-of-Mexico" origin. Mexican populations of *P. infestans* from the putative center of origin in Toluca Valley of Mexico harbored less nucleotide and haplotype diversities than Andean populations. Coalescent-based genealogies of mitochondrial and nuclear loci were congruent and demonstrate the existence of two lineages leading to present-day haplotypes of *P. infestans* on potatoes. Mitochondrial haplotypes found in Toluca, Mexico, were from the type I haplotype lineage, whereas those from Peru and Ecuador were derived from both type I and type II lineages (Fig. 10.3). Mitochondrial and nuclear haplotypes in populations from both the United States and Ireland were derived from both ancestral lineages that occur in South America, suggesting a common ancestry among these populations. The oldest mitochondrial lineage was associated with isolates from the botanical section Anarrhichomenum including *Solanum tetrapetalum* from Ecuador (Gomez et al., 2007). This lineage (EC-2 Ic) shares a common ancestor with *P. infestans* and has been named *P. andina* (Gomez et al., 2008; Kroon et al., 2004; Olivia et al., 2010). The geographic distribution of mutations on the rooted coalescent tree for both nuclear and mitochondrial loci demonstrates that the oldest mutations in *P. infestans* originated in South America; this is consistent with an Andean origin (Gomez et al., 2007).



	H6	H2	H3	H4	H5	H8	H1	H7	H9	
	3	24	1	1	3	2	23	5	1	Count
	3	7	.	.	.	.	20	.	1	South American
	.	17	1	1	3	2	3	5	.	Non-South American
	.	.	.	.	.	.	3	.	.	Bolivia
	.	.	.	.	.	.	5	.	.	Brazil
	.	6	.	.	.	.	5	.	1	Peru
	3	1	.	.	.	.	7	.	.	Ecuador
	.	7	.	.	.	.	.	.	.	Costa Rica
	.	5	1	1	3	.	.	.	.	Mexico
	.	3	.	.	.	2	.	5	.	United States
	.	2	.	.	.	.	3	.	.	Ireland
	A2	A1/A2	A1	A2	A1/A2	A1/A2	A1/A2	A1/A2	A1	Mating type
	Ic	Ia/1b	Ia	Ia	Ia	Ia	IIa	IIb	IIa	Carter's mtDNA haplotype
		Type I					Type II			

Figure 10.3 The rooted coalescent-based gene genealogy showing the distribution of mutations for South American (SA: Peru, Ecuador, Bolivia, Brazil) and non-South American (NSA: Costa Rica, Mexico, United States, Ireland) populations for the mitochondrial (P3 + P4) loci of *Phytophthora infestans* generated using GENETREE. The timescale is in coalescent units of effective population size and the direction of divergence is from the top (past–oldest) to the bottom (present–youngest). Numbers below the tree from top to bottom designate each distinct haplotype (H) and its count (i.e., the number of occurrences of the haplotype in the sample, where. = 0), the count of each haplotype in each population, the mating type of the isolates, and the mtDNA haplotype according to Carter et al. (1990). Image reproduced with permission from the *Proceedings of the National Academy of Sciences of the United States of America* from Gomez et al. (2007).

HOST SHIFTS AND JUMPS

P. infestans can infect other *Solanum* species and has shifted hosts on numerous occasions to exploit new niches. After its first introduction into Ireland, it was reported by Moore in Dublin in 1846 on *Anthocercis illicfolia*, a solanaceous shrub that had been imported from Australia into the Botanic Gardens at Glasnevin (Fig. 10.1b). The pathogen was also reported in petunia in the United Kingdom by M. C. Cooke in 1856. The haplotype Ib of *P. infestans* was identified on *Solanum lyratum*, a weed host that occurs commonly alongside potato fields in China in 1982 (Table 10.1). The occurrence of the Ib haplotype on a weed host in China, coincident with the time of first use of the fungicide metalaxyl in the country, suggests that this host shift may have enabled this fungicide-sensitive strain to survive by avoiding the fungicide that had been sprayed in fields. Weeds such as *Solanum nigrum*, *Solanum dulcamara*, and *Solanum sisymbriifolium* are also hosts of *P. infestans* and may act as a refuge and overwintering host for the pathogen in the absence of a potato or tomato crop in the field (Flier et al., 2003).

P. infestans occurs sympatrically in Mexico with *P. mirabilis* a pathogen that infects *Mirabilis jalapa* and *P. ipomoeae* a pathogen that infects *Ipomoea longipedunculata* (morning glory). The evolution in the Ic clade has involved host jumps to these unrelated plant species (Raffaele et al., 2010). *P. infestans* occurs sympatrically in Ecuador with *P. andina*. *P. andina* can infect other non-tuber-bearing species of *Solanum*, *Solanum muricatum*, and *Solanum betaceum*, indicating that *P. andina* has an expanded host range compared to *P. infestans*. *P. mirabilis* was first considered the closest known relative of *P. infestans* (Galindo and Hohl, 1985). However, phylogenetic analysis of *P. andina* from Ecuador suggests that *P. andina* (EC-2 Ic lineage) is also closely related to *P. infestans* (Adler et al., 2004; Blair et al., 2008; Gomez et al., 2008; Kroon et al., 2004; Oliva et al., 2010), and this lineage shares a common ancestor with *P. infestans* in the Andean region (Gomez et al., 2007).

Whether *P. andina* evolved as a hybrid of *P. infestans* and *P. mirabilis* (Gomez et al., 2008) or some other very close relative of *P. mirabilis* is still uncertain (Goss et al., 2011; Lassiter et al., 2010). The *ras* intron 1 sequence suggests that *P. andina* may have arisen from a hybridization possibly between *P. infestans* and *P. mirabilis* (Gomez et al., 2008). However, *P. mirabilis* has not been found in Ecuador where *P. andina* occurs. *P. mirabilis* was first reported in Mexico (Galindo and Hohl, 1985), so further exploration for *P. mirabilis* in the Andean region is warranted. *P. infestans* and *P. mirabilis* can hybridize in the laboratory and produce viable progeny (Goodwin and Fry, 1994). *M. jalapa* has not been widely surveyed for *P. mirabilis* in South America, although the host evolved in Peru and its common name is the flower of Peru. We are currently using nuclear gene

genealogies and Bayesian statistics to clarify the evolutionary relationships of the Ic clade species (Lassiter et al., 2010). One interpretation of the data is that the Andean region is the center of evolutionary origin for all the species in the Ic clade since *P. andina* and *P. infestans* coexist there and the oldest mutations in the *P. andina* EC-2 Ic lineage are of South American origin. Further surveys are needed to test the possibility of the occurrence of *P. mirabilis* and *P. ipomoeae* in the Andean region.

The patterns of sequence variation involved in host jumps in several Ic clades species of *Phytophthora* including *P. phaseoli*, *P. ipomoeae*, and *P. mirabilis* have been compared to *P. infestans* by resequencing genomes of the sister species in the Ic clade (Raffaele et al., 2010). Patterns of gene-sparse, repeat-rich regions in the genomes of the sister species were found, as reported in the *P. infestans* genome (Haas et al., 2009; Raffaele et al., 2010). These repeat-rich regions contained many effector genes that showed evidence of positive selection, uneven rates of evolution, and enrichment of genes induced *in planta* during preinfection and infection, suggesting host adaptation has led to the evolution of species in the clade. Unfortunately, these authors (Raffaele et al., 2010) did not analyze the genome of *P. andina* due to difficulties in reassembly of the genome, which contained many loci with heterozygous sites, a situation suggestive of *P. andina* being a hybrid (Brasier et al., 1999; Gomez et al., 2008; Goss et al., 2011; Kroon et al., 2004; Lassiter et al., 2010).

WHY IS LATE BLIGHT A REEMERGING DISEASE?

Late blight has reemerged as a significant threat to production for both tomato and potato. The varied dispersal mechanisms of the pathogen including the ability to move both as airborne inoculum (sporangia) and in plant material (tubers or transplants) have made local and long-distance spread common. The polycyclic nature of the life cycle enables the pathogen to produce copious amounts of spores in a short time period on aerial parts of plants, and these spores can move rapidly (within days) through untreated fields. Pathogen populations became resistant to the phenylamide fungicides shortly after their introduction. The widespread monoculture of highly susceptible potato cultivars in the United States has made them vulnerable to disease. *P. infestans* also can infect a wide range of other solanaceous plants. The plasticity of the genome as revealed by genome sequencing and the diversity of pathogen effectors that can overcome host R (resistance) genes make host resistance a moving target at best and make the search for more durable host resistance a more viable goal then single-gene resistance (Haas et al., 2009; Kamoun and Smart, 2005).

Populations of *P. infestans* in the United States have consisted of a series of mostly asexual clonal genotypes and 19 genotypes have been reported previously in regional populations (Fraser et al., 1999; Fry et al., 1992; Gavino et al., 2000; Goodwin et al., 1994a, 1998; Wangsomboondee et al., 2002). We recently identified five new multilocus genotypes in the eastern United States (US-20 to US-24) (Hu et al., 2012). The presence of sexually reproducing populations in several European countries has been documented by restriction fragment length polymorphism (RFLP) markers and later by microsatellite markers (Cooke et al., 2007; Drenth et al., 1994; Knapova and Gisi, 2002; Lees et al., 2006; Zwankhuizen et al., 1998, 2000).

In 2009, a widespread strain, US-22 (Hu et al., 2012), identified by multilocus genotyping, spread in the northeast of the United States on tomato transplants and rapidly infected potatoes. There was a complete loss of the tomato crop in many states and an increase in fungicide use on pota-

toes to curb epidemic spread. A combination of weather conducive for disease and widespread inoculum distribution exacerbated the epidemic. Strain US-22 was very widespread (12 states) on tomato and potato, and this genotype accounted for about 60% of all the isolates genotyped in 2009 in the United States. Organic tomato growers were devastated by the disease since few options for control besides copper-based sprays are available in these production systems. At least 400 farms were affected by the disease in New England (Moskin, 2009). The lack of deployment of resistant varieties of both potato and tomato in the United States has exacerbated the situation. On the basis of sequence analysis of the *ras* gene, US-22, US-23, and US-24 appear to have been derived from a common ancestor (Hu et al., 2012).

The US-8 genotype, which is still common on potatoes in the United States, is mefenoxam resistant. Isolates of the widespread US-22 genotype and several other clonal lineages (US-23 and US-24) are largely sensitive to mefenoxam. However, this information was not known until after the widespread epidemics in the United States in 2009 since fungicide sensitivity assays are not rapid enough to deliver information to growers in a timely manner. Microsatellite markers can be used to quickly separate some of the genotypes. The phenylamide fungicides such as mefenoxam target RNA polymerase I (Davidse, 1988; Gisi and Cohen, 1996). Markers associated with mefenoxam resistance mapped to a single locus (Fabritius et al., 1997; Lee et al., 1999). A rapid molecular assay to identify mefenoxam insensitivity in the pathogen is needed.

Hundreds of effector genes are present in the genome (Haas et al., 2009). Avirulence alleles can now be tracked in the field to study the evolution of these genes in pathogen populations in response to deployed host resistance genes. This should help document the pathogen–host arms race and lead to strategies to slow the development of new pathotypes of *P. infestans*.

CONCLUSIONS

Further field and laboratory studies are under way exploiting the *P. infestans* genome sequence to develop rapid genotyping methods and to monitor further changes in the structure of both selectively neutral and evolving traits of the populations of the pathogen in North America. A national portal (http://www.USAblight.org) has been developed to track disease occurrences, send disease alerts, provide a venue for submitting samples for diagnosis and genotyping, publish pathogen multilocus genotypes, and provide management information to growers developed by the extension and research community on late blight. Over 160 years after the great famine, late blight continues to challenge the sustainable production of potato worldwide.

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