

REVIEW

The origin of *Phytophthora infestans* populations present in Europe in the 1840s: a critical review of historical and scientific evidence

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A critical review of available historical and scientific evidence related to the question of the origin of the first European populations of the potato late blight pathogen *Phytophthora infestans* is presented. It shows that the bases for the current theories of a direct introduction of the fungus into Europe and North America from either a Mexican or an Andean centre of origin and of diversity are questionable. An alternative theory, involving a three-step process: (i) migration from central Mexico to South America several centuries ago; (ii) migration from South America to the US in 1841–1842, and (iii) migration to Europe from either South America, US or both in 1843–1844, is in good agreement with both the historical records and with the genetics and structure of current populations.

INTRODUCTION

The European epidemics of *Phytophthora infestans* in 1845–1847, which led to the Great Irish Famine, are probably amongst the best-documented epiphytotics of all time. The explosive nature and spectacular effects of the disease, as well as the magnitude of its scientific (Peterson, 1995) and also political, social and economic consequences, made these catastrophic outbreaks a favourite topic of investigation for historians and plant pathologists alike, and resulted in a very large corpus of written material. To quote Bourke (1964), ‘the major problem is the sheer volume of relevant data’ for the scientist willing to trace back the origin of the epidemic and of its fungal agent.

The many studies devoted to this problem focused on two sources of evidence: (i) historical accounts of the outbreaks published by contemporary observers, scientists or amateur naturalists, and (ii) inference from modern observations of the structure of genetic and phenotypic variability in populations of *P. infestans* in different parts of the world. Two different theories on the origin of the first inoculum successively emerged: the Andean theory, suggesting that the fungus

came from South America, predominated during the second part of the 19th century and up to the 1950s, to be then supplanted by the currently widely accepted view of a Mexican source. However, recent papers supporting the Mexican theory (Goodwin *et al.*, 1994) or trying to reinforce the Andean hypothesis (Abad & Abad, 1995; Abad *et al.*, 1995) brought new elements to the debate.

Aside from its historical interest, a better understanding of the sources of the inoculum that started the disastrous epidemics of the 1840s in Europe is of scientific relevance today, as another global migration of the fungus, that took place in the mid 1970s, dramatically modified the structure of *P. infestans* populations in many areas of the world (Spielman *et al.*, 1991; Fry *et al.*, 1992, 1993; Goodwin *et al.*, 1994). It is now well established that the source of this recent migration is Mexico (Fry *et al.*, 1992, 1993). Therefore, a Mexican origin of the pathogen when it first spread into Europe and North America, if demonstrated, could provide clues for possible changes to be expected in the future in current populations of the fungus. This paper reviews the scientific and historical evidence available concerning the original introduction of the blight fungus to Europe and North America, and supports an alternative

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theory for the origin of late blight in these areas a century and a half ago.

EVIDENCE RELATED TO THE ANDEAN AND MEXICAN THEORIES

Three factors have been considered in the discussions of the origin of *P. infestans* inoculum: (i) the location of the centre of origin of the pathogen, (ii) the availability and/or opportunity for importation into previously blight-free areas and the correlative question of the date of importation, and (iii) analyses of the establishment and evolution of recent population structures in the different parts of the world.

Centre of origin of the pathogen

The Andean theory, first proposed by Berkeley (1846) and later supported and documented by de Bary (1861), was originally based on the assumption that *P. infestans* originated from the same part of the world as its host, namely the northern part of the South American Andes (currently Peru and Bolivia). This view gained widespread acceptance until the mid-1950s, when the original assumption was challenged by work in central Mexico showing that both mating types were present, in about equal proportions, only in that part of the world (Gallegly & Galindo, 1958), and that pathogenic diversity was also greatest in that area (Mills & Niederhauser, 1953). The extreme diversity of central Mexican populations of *P. infestans* has now been confirmed for a number of neutral markers, such as isozymes and DNA RFLPs (Goodwin *et al.*, 1992). These data support the theory, first proposed by Reddick (1939, 1943) and commonly accepted today, that *P. infestans* is native from the central highlands of Mexico, and consequently that the original introduction to Europe and North America was from Mexico.

The Andean theory was further weakened by the difficulty to establish the antiquity of late blight presence in the Andes. According to Niederhauser (1991), the first reports of certified blight outbreaks in South America all indicate that the fungus was introduced there only recently, in most cases during the first decades of the 20th century. However, Abad & Abad (1995) looked back at the historical reports of blight epidemics in the Andes, and claimed that the pathogen has been endemic there for centuries. This claim was based primarily on a different analysis, focusing

on the semantics of local names for blight, of sources already studied by earlier workers, such as reports by Boussingault (1845), Marchal (1845) and a citation of Acosta (1590) by de Bary (1861). The discrepancy between interpretations of Niederhauser (1991) and Abad & Abad (1995) mainly arises from the difficulties in attributing with certainty the damage or diseases reported as the manifestation or consequences of late blight. Niederhauser (1991) pointed out that, whilst the historical descriptions of damage to potato crops in Peru are in good agreement with the situation that can be observed today, a number of causes interact and result in rotting of the tubers or death of the foliage, and that even now late blight is often a minor component of that complex of factors. Abad & Abad (1995) nevertheless provide some evidence that the disease was known in Peru and some other parts of South America at or before the time of the first European outbreaks. Of particular interest in this respect are citations of consul Pazos from Bolivia by Marchal (1845) indicating that 'the excess of equatorial rains ... that is sometimes experienced in Peru causes the same disease that affected European potatoes during the present year ...', and of French explorer d'Orbigny by Rozé (1898), stating that 'the Aymaras indigenous people, living in the vicinity of La Paz, in Bolivia, have known since the most remote antiquity the disease that struck potatoes in Europe this year'. D'Orbigny provides a detailed description of a potato disease, named *casagui* by Bolivian Aymaras, that he considers identical to late blight, and cites the fact that the General Consul of Bolivia in England, don Antonio Acosta, also confirmed that disease symptoms observed in Europe in 1845 were identical to *casagui*. Further support to Abad & Abad's (1995) claim about the antiquity of blight in South America can be found in Farlow's (1875, p. 334) statement that 'the disease prevails amongst the wild species of Peru, where the potato is indigenous' (although no facts or references supporting this claim are provided by the author), as well as, more convincingly, in the presence of race-specific resistance genes to *P. infestans* in several wild Andean potato species (Hawkes, 1958).

However, the ancient presence of the fungus in South America is not by itself sufficient to prove that the Andean area is indeed the centre of origin of *P. infestans*. In an attempt to support this hypothesis, Abad *et al.* (1995) reviewed some factual evidence that they consider decisive. Unfortunately, most of their effort is unconvincing

at best. Contrary to Abad *et al.*'s (1995) claim, no evidence is given that late blight is actually 'much older in [Andean countries] than it is in central Mexico'. Many statements of the authors apply just as well to central Mexico and to Andean countries, such as those related to the wide host range of *P. infestans* and to the resistance found in a number of *Solanum* species. Investigations of the resistance and host range of *P. infestans* in central Mexico have shown that the pathogen is able to infect a number of wild species growing in that area (Hawkes, 1958; Rivera Peña, 1990a,b) and that both race-specific and race-nonspecific, partial resistance are widespread amongst these accessions (Rivera Peña, 1990b). Race-specific resistance genes are actually more frequent in central Mexico than in the South American Andes (Hawkes, 1958), which may indicate that the latter location is only a secondary home to the fungus. The low diversity found in the Andean area for isozymic and molecular markers, virulence combinations (races) and mating types (Tooley *et al.*, 1989) also supports this interpretation, rather than South America being the centre of origin of *P. infestans* as claimed by Abad *et al.* (1995).

Opportunities and possible dates for migration of *P. infestans*

The development of late blight in North America is quite well known since the classic study of Stevens (1933). The first outbreaks of the disease were recorded in the five States of the North Eastern coast nearest Philadelphia and New York in 1843, and then spread to most potato growing areas during the following two years. Although Farlow (1875) reported severe blight attacks in 1842, Stevens (1933) considered that these reports did not refer to the situation in the United States, but in Europe. Jones *et al.* (1912) joined Jensen (1887) in his hypothesis that the opportunity for *P. infestans* importation into Europe and North America from the Andean region arose from the guano trade started in the early 1830s, the faster crossings of the Atlantic allowed by steam-powered ships, and the use of ice to prevent deterioration of the potato shipments.

Stevens (1933) had little doubt that the disease was actually imported to the US from Europe (probably from the British Isles), stating that 'as the disease was well established in Ireland, England, and on the continent of Europe in 1842, its prompt introduction on the Atlantic Seaboard of North America can be easily understood'. The idea that

blight had been present in Europe for several years before the great epidemics of 1845–1847 was widely accepted by the naturalists who described the outbreak (Decaisne, 1846, reviewed evidence for the presence of the disease in France as early as the second half of the 18th century), and persisted amongst scientists in the first years of the 20th century. This is evident in the extensive report of Jones *et al.* (1912), who based their position on a monograph by von Martius (1842) which, they believed, described late blight epidemics in Europe as early as 1830. Jones *et al.* (1912; pp. 20–21) credit von Martius for the first descriptions and figures of the disease. They had no doubt that the disease under consideration was indeed late blight and the fungus *P. infestans*, stating (p. 24) that 'so far as we can learn, the first scientific writer to ascribe the disease to a parasitic fungus was von Martius (1845), whose figures of the fungus, whilst crude, are easily recognizable' (*sic*). Incidentally, the only reference to a paper by von Martius in Jones *et al.*'s (1912) list of references is the 1842 monograph, so the citation of von Martius (1845) is either a printing error or a reference to a paper not listed. In either case, a closer examination of the figures published by von Martius in his 1842 monograph, particularly those numbered 30 and 31 which could most easily recall *P. infestans*, does not support the statements of Jones *et al.* (1912). A number of features, such as the form of the spores or the septate mycelium, clearly indicate that these drawings do not represent *P. infestans* (see Fig. 1 a,b). Furthermore, amongst the other figures von Martius (1842) gave of the fungi he observed in diseased tubers are very good drawings of *Fusarium* macroconidia (Fig. 1 c-e). It is therefore safe to consider, as did Bourke (1964), that von Martius's 1842 monograph deals with *Fusarium* dry rot, not late blight. It can be noted that the differences between the two diseases were detailed by von Martius himself (1845) in a letter to Professor Bergsma in Utrecht, and later on by Decaisne (1846) who cautioned against the frequent confusions of the 1845 'potato disease' with the 'gangrene dry rot' described and investigated by von Martius in his 1842 monograph.

One of the first authors to consider that *P. infestans* reached the United States before it arrived in Europe was Bourke (1964), who stated that 'there is some evidence that blight was already present in parts of Europe in 1844; there are isolated grains of evidence that the disease may have dated back locally to 1843 or even 1842, but they are far from conclusive. For 1841 and earlier, there is little risk in a dogmatic

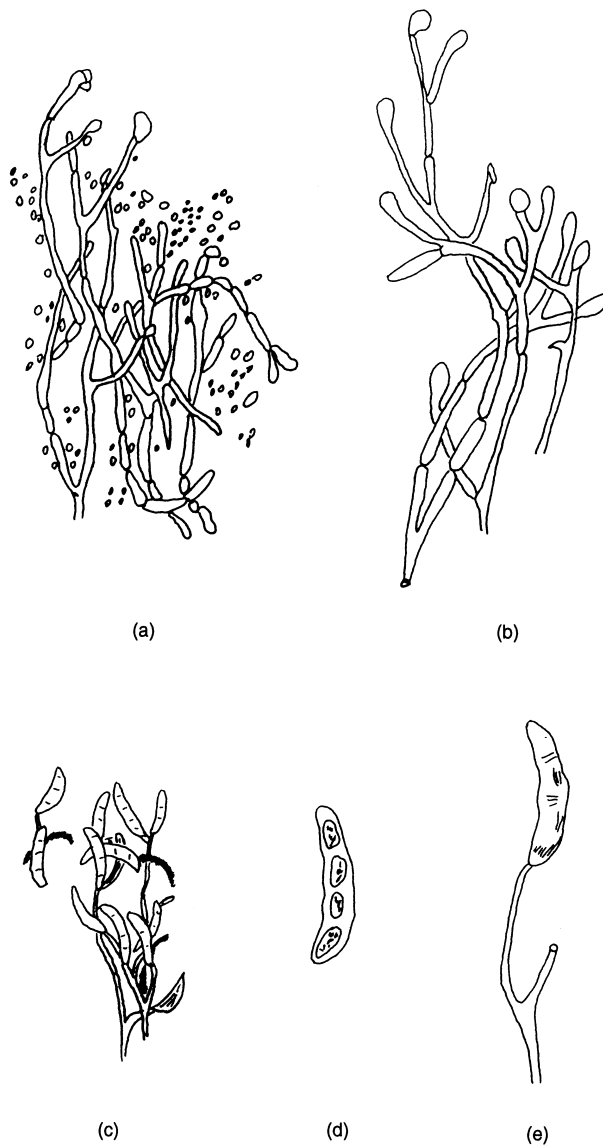


Fig. 1 Reproduction of drawings of fungal potato pathogens from von Martius (1842). The septate mycelium and rounded spores (a and b) were probably those mistaken by Jones *et al.* (1912) for the first *P. infestans* drawings; c, d and e are clearly *Fusarium* macroconidia and mycelium.

claim that potato blight did not then exist in Europe'. None of the reports providing the evidence for the 1842–1844 outbreaks are cited in Bourke's paper, but his reconstruction of the sequence of events has been little questioned since, and was endorsed by most subsequent authors (e.g. Klinkowski, 1970; Fry *et al.*, 1993). This chronology is further supported by evidence

provided by Bourke & Lamb (1993), who showed that *P. infestans* most probably reached Europe with a shipment of potatoes officially imported into Belgium late in 1843 or early in 1844, in an effort to restore the potato stocks then badly affected with viral diseases and *Fusarium* dry rot. They cite a decision of the Provincial Council of West Flanders, dated 4 July, 1843, to import

commercial cultivars of various origins to this effect, and trials of these in the Cureghem farm. However, no precise indication as to the origins of these stocks is given, and it is likely that both South American and US genotypes were imported, so that the origin of the inoculum cannot be ascertained from these records (Bourke & Lamb, 1993).

Structure and evolution of recent populations of the pathogen

The many methodological developments made during the last 10 years, particularly the use of isozymes and of molecular markers, allowed in-depth analysis of the phenotypic and genetic structure of *P. infestans* populations from diverse geographical areas. Amongst the most striking results of these investigations is the discovery that *P. infestans* populations present in all regions of the world except central Mexico before the world-wide migration of the mid 1970s belonged to a single clonal lineage, called US-1 (Goodwin *et al.*, 1994) or PO-1 (Sujkowski *et al.*, 1994), as determined by nuclear DNA fingerprinting with the moderately repetitive probe RG57 (Goodwin *et al.*, 1994). Meanwhile, the population present in central Mexico was found to be a collection of varied, and in most cases unique, RFLP genotypes (Goodwin *et al.*, 1992), a further strong argument in favour of central Mexico as the centre of diversity of *P. infestans*. Goodwin *et al.* (1994) postulated that US-1/PO-1 has been present in Europe and the US since the first global migration of the fungus. They proposed that the original migration was a two-step process, first from Mexico to the US around 1842–1843, and then from the US to Europe in 1843 or 1844. In this view, the US-1/PO-1 lineage is considered the only survivor of an originally larger group of genotypes, submitted to a series of founder effects out of its Mexican birthplace and, subsequently, out of its North American secondary home. No extensive, long-range migrations of *P. infestans* genotypes are thought to have taken place between the 1840s and the 1970s (Fry *et al.*, 1993).

However, this scenario is faced with several difficulties. The lack of further migrations between the 1840s and the 1970s requires that US-1 was either fixed in the US before migration to Europe occurred, or became fixed independently in the US and Europe. Both possibilities seem rather improbable if the original source of migration

was central Mexico. Fixation of US-1 in the US before immigration into Europe, i.e. in only one or two growing seasons, is highly improbable if the original migration involved a number of different genotypes, as would certainly have been the case if central Mexico were the source of the inoculum. Goodwin *et al.* (1994) postulated that a severe demographic bottleneck restricted to the absolute minimum size of one the number of clones that escaped central Mexico in the first migration event to the US, leading *ipso facto* to its fixation in this country, and subsequently in Europe. This is theoretically possible, but would require a conjunction of highly uncommon circumstances. Furthermore, Goodwin *et al.* (1994) showed that the most probable sources of inoculum in their scenario are infected tubers of wild potato species, which were of no commercial or agricultural value. However, tuber blight is virtually nonexistent on wild potato species growing in the Toluca valley (Rivera Peña, 1990a), possibly because of the extremely high level of soil suppressiveness to the pathogen (A. Velarte Garcia, unpublished data). This observation, together with the lack of economic value of wild potato species in the 1840s, make blighted tubers unlikely candidates for successful propagation of *P. infestans* outside central Mexico.

Independent fixation of this genotype in the US and Europe is also improbable, as recognized by Goodwin *et al.* (1994). If local founder effects were the only evolutionary mechanism acting, the probability for the same genotype surviving and becoming fixed on both sides of the Atlantic is extremely low. Strong evidence exists that genetic drift and local founder effects are major factors in the evolution of *P. infestans* populations (Fry *et al.*, 1992; Andrivon, 1994 a,b), but the stochastic nature of these processes makes their outcome impossible to predict in a deterministic way. Drenth *et al.* (1994) showed that over 90% of the genotypes present in a given year and place will not survive to the next growing season, and that extinction affects genotypes irrespective of their frequency in the population. Under these conditions, the probability is close to zero that drift and founder effects alone could lead to fixation of the same clonal lineage in two independent areas as large as Europe and North America, not to mention other locations such as Africa or Asia (the pathogen was probably introduced more recently to these continents, possibly from Europe, on infected potato seed). Furthermore, current population structures of *P. infestans* in Europe and North America are consistent with a metapopulation pattern, i.e. a

mosaic of local populations with their own evolutionary history and occasional gene flow between them (Fry *et al.*, 1992; Andrivon, 1994b). Although the probability of extinction is higher in the small patches of a metapopulation than in large, continuous habitats (e.g. Burkey, 1995), metapopulations are efficient ways of maintaining genetic diversity over large areas provided migration between patches occur (Hanski *et al.*, 1995), since equilibrium in individual patches is not necessary to maintain the stability and, hence, the diversity at the metapopulation level (Olivieri *et al.*, 1990). A metapopulation structure thus makes fixation of a single genotype in two separate continents under the exclusive action of genetic drift next to impossible on a purely statistical basis.

Selection of US-1 is hardly an alternative explanation for the ubiquitous presence of this lineage before the 1970s, since selection for a particular genotype was weak at best, and most probably nonexistent, in Europe and the US at the time of the original introduction. Most potato cultivars grown during the 1840s were very susceptible to blight, as the numerous reports from the 1845 and subsequent epidemics demonstrate (Bourke, 1964). These cultivars carried no known R genes, and their general resistance was extremely low (Vanderplank, 1957). Therefore, cultivar resistance was probably not a selective factor at all for the initial populations of *P. infestans*. As no selection forces other than cultivar resistance could conceivably have acted strongly enough to fix one genotype in a couple of years in a biotrophic fungus like *P. infestans*, it is difficult to postulate that the exclusive presence of the US-1 lineage could result from host-mediated selection of this clone amongst a variety of other pathogen genotypes.

The 1970s migration showed that migration from the Mexican centre of origin most likely would have involved isolates belonging to both the A1 and A2 mating types. A2 isolates would have probably survived in at least some of the regions they reached, and would have eventually predominated or become fixed as they have in the Far East (Japan, Korea) following the 1970s migration (Mosa *et al.*, 1989; Lee *et al.*, 1993). This was not the case during the first migration. As pointed out by Bourke (1964), the large variability of *P. infestans* genotypes in central Mexico 'give strong support to the claim of Mexico as the primary site of origin of potato blight; they are less convincing as evidence that Mexico was the immediate source of the less

versatile fungus which invaded the United States and Europe'. Indeed, this very variability is one of the strongest arguments in favour of an area of origin of the inoculum outside the centre of diversity of the pathogen.

MIGRATION FROM OUTSIDE THE CENTRE OF ORIGIN: A POSSIBLE SOLUTION

The current Andean and Mexican theories are both based on the hypothesis that the pathogen was introduced into Europe and North America from its native home, i.e. its centre of origin and diversity. The above review highlighted some of the difficulties raised by this initial postulate. However, an alternative theory, first suggested by Bourke (1964) and later explicitly exposed by Tooley *et al.* (1989), adequately addresses these difficulties.

This theory postulates a three-step migration process, first from Mexico into South America, and subsequently from there to North America and Europe. Little doubt remains now that central Mexico constitutes the area of origin of *P. infestans*. However, some of the evidence reviewed above, including several of the historical accounts of potato diseases in the Andes but first and foremost the presence of race-specific resistance genes to *P. infestans* in wild Andean potato species, point to a relatively ancient introduction (at least several centuries ago) of the late blight fungus into the Andean regions of South America from its original home in Central Mexico. Opportunities for such an introduction arose before and after the Spanish conquest, particularly during the several human migrations from North America to the Southern hemisphere. Although potatoes were not cultivated by inhabitants of North and Central America until very recently, several groups collected and ate wild tuber-bearing *Solanum* species in these regions since remote times (Salaman, 1949). Furthermore, the possibility that *P. infestans* was introduced on some plant other than potatoes to the South American Andes should not be overlooked, as the host range of South American isolates of the fungus extends to a number of genera and species of the Solanaceae and Nolanaceae (Turkensteen, 1978; Abad *et al.*, 1995). It is, however, difficult to obtain direct (archaeological or historical) evidence of such an introduction.

Once introduced into South America, populations of *P. infestans* most probably evolved in almost complete isolation. Even today, when

communications and trade are very active, US-1 is still the only *P. infestans* lineage found in Peru and Ecuador (G. A. Forbes, personal communication), indicating very limited or no gene flow in Andean populations of the pathogen. Furthermore, the original introduction was probably restricted to a few genotypes: no large cargoes of potatoes or other solanaceous crops susceptible to blight could possibly be introduced in remote times, given the means of transportation and conservation of fresh plant material available at the time. The narrow genetic base of Andean populations of *P. infestans*, combined with the often limited incidence of blight in Andean regions (Niederhauser, 1991) – reducing the absolute size of the populations – and with the high extinction rates observed in *P. infestans* lineages (Drenth *et al.*, 1994), probably resulted in the relatively rapid fixation of a single clonal lineage (today known as US-1) in the South American Andes. Migration of the fungus to the US and Europe from South America in the 1840s, either as two separate events or as a two-step process (first to the US and subsequently to Europe from North America), would then easily account for the panglobal distribution of the US-1 lineage before the migration of the mid 1970s.

Under this theory, migrations of *P. infestans* before 1850 can be regarded as a three-step process, as follows: (i) several centuries ago, limited migration(s) from central Mexico to the South American Andes followed by fixation of the US-1 clonal lineage in this area; (ii) 1841–1842, migration from South America to the US; (iii) 1843–1844, migration to Europe from South America, US or both. In this hypothesis, the actual chronology of introductions to the US and to Europe, as well as the actual origin of the tubers introduced into Europe in 1843–1844 (US, South America, or both), whilst of historical interest, are of no consequence as to the outcome in terms of structures of *P. infestans* populations, since only one lineage would have been present in the source area of the inoculum at the time of the introduction. This theory has the advantage of addressing in a plausible way the major contradiction between the high diversity of the fungus in its centre of origin and the very homogeneous populations found everywhere else before the 1970s migration.

Bourke (1964) indicated that a South American inoculum would be easier to trace back, as far as transportation means are concerned, than a Mexican one. Indeed, the guano trade offered ample opportunities for rapid transportation of potato tubers from South America to the US and Europe.

Data from population analyses, but also from the population biology of *P. infestans*, are in good agreement with the three-step theory proposed by Tooley *et al.* (1989) and further elaborated here. However, this three-step process is not easy to demonstrate, particularly because of the difficulties to establish unequivocally the antiquity of *P. infestans* presence in South America. Further investigations, particularly concerning this first migratory step, are now needed to validate and test it.

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