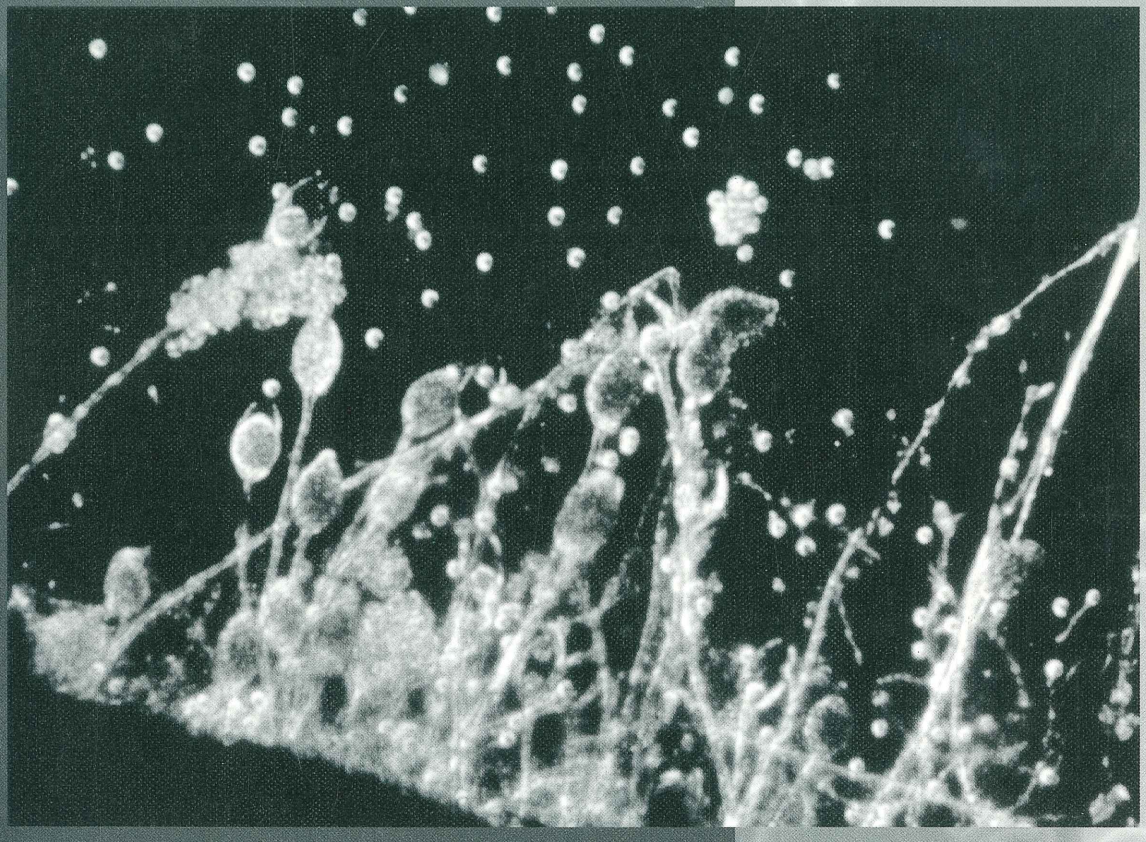
Phytophthora Diseases Worldwide



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CHAPTER 33

Phytophthora infestans

(Mont.) de Bary (1876)

Synonyms of the causal agent of late blight of potato include *Gangraena tuberum solani* Martius (1842), *Botrytis vastatrix* Libert (1845) (see Waterhouse 1970a), *Botrytis infestans* Montagne (1845), *Botrytis fallax* Desmazieres (1845), *Botrytis solani* Harting (1846) (see Waterhouse 1970a), *Peronospora trifurcata* Unger (1847) (see Waterhouse 1970a), *Peronospora fintelmani* Caspary (1852), *Peronospora devastatrix* (Libert) Caspary (1855), *Peronospora infestans* (Montagne) de Bary (1863), *Phytophthora thalictri* Wilson and Davis (1907) (see Waterhouse 1970a), and *Phytophthora devastatrix* (Libert) Puttemans (1937).

Martius (1842) in Prussia was the first to claim that a fungus caused the malady that devastated the foliage and tubers of potato in nearly all of Europe and named the fungus *Gangraena tuberum solani.* According to Bourke (1991), who reviewed the scientific controversy, Martius's report was unorthodox at that time and "to some smacked of heresy." Berkeley, a country parson in England who was also interested in plant diseases, viewed Martius positively and published the translated paper in the *Gardener's Chronicle.* The volume *Phytophthora* (Lucas et al. 1991) was dedicated to Reverend Berkeley because of his active participation in the quest to determine the cause of late blight of potato (Buczacki 1991).

It must be remembered that in 1842 the germ theory of disease was not universally accepted, and many considered fungi and bacteria the results but not the causes of disease. Bourke (1991) relates an interesting saga about the many theories to explain this new disease of the potato that appeared in continental Europe, the British Isles, and Ireland. The majority of comments blamed the miserable weather, an idea we still hear frequently. I once told an Irish priest that my research involved problems such as the potato blight, and his reply was, "Ah sure, Don, and that is caused by the damp!"

In 1845, Dr. August Morren from Belgium wrote several letters that supported the thesis that a fungus was the cause of the potato blight. His theory was vigorously contested, as Bourke (1991) relates, by Dieudonné, who declared that Morren's concept would hang forever like Damocles's sword over his head and that this depressing thesis must be exposed so that the fungus did not "become the terror of the farmers." Morren's replies were equally sharp. After some doubts, Montagne eventually accepted Morren's thesis and with information that he had developed, described *Botrytis infestans,* the fungus that we now know as *Phytophthora infestans.* Little did Montagne know that his description would be so vigorously denounced that even he would back off for a time. Bourke (1991) graphically relates the many controversies that followed Montagne's report until Anton de Bary (1876) determined conclusively that the fungus, which he renamed *Phytophthora infestans,* was the cause of late blight.

P. infestans is perhaps the best known of all the species of *Phytophthora* because of the tremendous destruction it wreaked on the potato crops in Ireland during the 1840s. The economic chaos from the loss of this vital food crop directly led to so many deaths that a high proportion of the surviving Irish people emigrated overseas to Europe and America. A fascinating and detailed account of this tragic period in history is told by Large (1960). Although *P. infestans* has a limited host range (mainly solanaceous hosts) (Table 33.1; Commonwealth Mycological Institute Map 109 ed. 5, 1982), it is of considerable importance, since it severely affects potato, a food crop that is vital to the diets of many people worldwide. Now, more than 125 years later, it has yet to be totally controlled, mainly because of the bewildering array of new biotypes known as physiologic or pathologic races that appear with each succeeding introduction of race-specific, blight-resistant potato cultivars (Vanderplank 1963, 1984, 1988; Malcolmson 1976). The production of new races of *P. infestans* is discussed in Chapters 3 and 6. Volume 7 of *Advances in Plant Pathology* is devoted entirely to *P. infestans* (see Fry and Spielman 1991). Fry et al. (1993) relate new information on how the A2 mating type migrated to different areas of the world, most likely from its native home in central Mexico. Gillis (1993) presents an excellent popular rendition of how the A2 mating type migrated from Mexico to nearly every potato-producing nation of the world. Until the 1980s, only the A1 mating type was present outside Mexico. The book *Phytophthora infestans 150* contains numerous papers on *P. infestans* given at an international meeting on the 150th anniversary of the description of *P. infestans* (Dowley et. al. 1995).

Until the early 1980s, A1 was the predominant mating type detected in all regions of the world except central Mexico, where both A1 and A2 mating types coexisted at a 50:50 ratio (Gallegly and Galindo 1958; Gallegly and Niederhauser 1959). Since the report of Hohl and Islen (1984), A2 isolates are now found in nearly all potato-growing regions of the world, but usually at ratios less than 50:50 (Fry et al. 1992, 1993; Spielman et al. 1991; see also Chapter 3 for more citations). However, Sujkowski et al. (1994) reported an approximate 50:50 ratio of A1 to A2 mating types in Poland.

On the basis of isozymic studies of more than 200 isolates of *P. infestans* collected in 20 countries, S. B. Goodwin et al (1994) hypothesized that a single clonal line of *P. infestans* migrated in about 1842 or 1843 from Mexico first to the northeastern United States and from there in about 1845 to Europe, where late blight of potato became an international problem. Subsequent migrations of *P. infestans* to Africa and Asia were likely from Europe because most of the seed potatoes of the world emanated from there. Variants of the single clonal line of *P. infestans* most likely were the result of mutations or mitotic recombination. Fry and Goodwin (1995) summarize the migration of the A2 mating type and the new population of *P. infestans* from Mexico to the northeast United States and to Europe, East Asia, and South America. See also S. B. Goodwin et. al. (1994) for a description of the panglobal distribution of a single clonal line of *P. infestans* from Mexico to the United States and other parts of the world.

It is not understood why the A2 type did not spread

throughout the world with the movement of potato as a food crop during the 1800s. It was not until the early 1980s that A2 mating types were reported in several European countries, Egypt, the Middle East, Asia, and South America (Spielman 1991). Spielman et al. (1991) and Fry et al. (1993) summarize and map the migration of the new genetic types of *P. infestans* throughout the world. Use of molecular isozyme techniques (described in Chapter 3 in the section Genetics and Cytology of *Phytophthora,* in an excerpt from Spielman [1991], and in Chapter 4 under Molecular Technology for Differentiation of Species) allowed the determination of the change in the genetic makeup of *P. infestans.* The detection of particularly unique isozyme characteristics in isolates collected after 1980 that did not occur in isolates from previous years supports the assumption that a change in the genetic makeup of *P. infestans* is in progress. The available evidence indicates that the newer genotypes are pathologically more fit than populations made up of the older isolates (discussed by Spielman [1991] and Spielman et al. [1991]). These and the studies summarized by Fry et al. (1993) and Fry and Goodwin (1995) confirm that *P. infestans* is highly variable and because of its ability to become adapted to different hosts and different situations remains a major threat to world food production.

Recent information on migration of the A2 type into Great Britain, other areas of Europe, the United States, and British Columbia (K. L. Deahl in Gillis [1993]) raises the question of whether natural mating of the A1 and A2 types could lead to even greater genetic variability than existed when only the A1 mating type was present. Although the extensive monitoring of isozyme groups representing "old" and "new" populations appearing in Europe during the 1980s has not thus far indicated that recombinant types have arisen, the possibility exists. Fry et al. (1993) state that some evidence indicates that the high frequency of unique genetic biotypes in Poland resembles the diversity that exists in central Mexico where A1 and A2 types coexist (W. Sujkowsky, *unpublished*). Sujkowski et al. (1994) present evidence that the introduction of the A2 mating type into Europe has influenced the increase in virulent biotypes (see the section in Chapter 3 Genetics and Cytology of *Phytophthora*).

Characteristics of *Phytophthora infestans*

P. infestans is classified in group IV (Stamps et al. 1990). See Tables 4.2 and 4.3 for tabular keys and Appendix 4.9 for a dichotomous key (Ho 1992). Morphology is shown in Figures 33.1 and 4.12G. See Fry et al. (1993) and Hooker (1981) for photomicrographs of spore structures.

Sporangia

Sporangia are ovoid, ellipsoid to limoniform, tapering at the base, caducous (pedicel <3 μm), and semipapillate. Average size of sporangia ranges from 36 × 22 μm (Tucker 1931) to 29 × 19 μm (Waterhouse 1963). These dimensions are similar to those of de Bary (1876), Rosenbaum (1917), Haskell (1921), K. O. Müller (1928), and Leonian and Greer (1929). Sporangiophores are compound sympodial (Figure 4.5B) with a small characteristic swelling just below the sporangium (Figures 4.5C and 4.6).

Hyphal Swellings and Chlamydospores

Neither hyphal swellings nor chlamydospores have been reported, except in a paper from Russia by Patrikeyeva (1979), who noted chlamydospores with a two-layer wall after incubation for 4 to 9 months on oat-pea agar at 9 to 10°C.

Sex Organs

P. infestans is heterothallic. Antheridia are amphigynous; oogonia are 31 to 50 μm in diameter (average 38 μm); oospores formed in plant leaves are aplerotic, 24 to 35 μm in diameter (average 30 μm); in artificial culture they measure 24 to 56 μm in diameter. Until the early 1980s, A1 was the only mating type found in most of the world, but in central Mexico both A1 and A2 isolates coexisted at a 50:50 ratio (Niederhauser 1991). Inoculation of a leaf with an A2 isolate of *P. drechsleri* and *P. infestans* (A1) induced oospore production (Skidmore et al. 1984). Whether or not unique biotypes could arise from unrelated species is unknown. Most likely, formation of oospores results from stimulation by hormonelike substances emitted by the opposite mating type (see Chapter 3).

Growth Temperatures

The minimum temperature for growth is 4°C, optimum 20°C, and maximum 26°C.

Some Diseases Caused by *Phytophthora infestans*

Late blight of potato and tomato are the most important diseases caused by *P. infestans.* Both hosts are members of the plant family Solanaceae. Other hosts and their distribution are listed in Table 33.1.

Late Blight of *Solanum tuberosum* L. (Potato)

Late blight of potato is described in detail in the *Compendium of Potato Diseases* (Hooker 1981), by Rich (1983) in a text on potato diseases, and in a publication on potato health management (Rowe 1993). Shorter descriptions are given by Stamps (1985h) and Holliday (1980). Late blight with an emphasis on the disease in Mexico is discussed by Niederhauser and Cobb (1959) and Niederhauser et al. (1954).

Infected foliage first becomes yellow and then water soaked and eventually turns black (Plate 33.1). The leaf symptoms, which may appear on the foliage any time during the development of the plant, consist of purple black or brownish black lesions (Plate 33.2) and usually appear first at the tip or margins of the leaf. Later, lesions may occur anywhere on the leaf, petiole, or even the stem. Symptoms often resemble those caused by frost. If cool, moist conditions prevail, whitish masses of sporangia (Figure 33.1) appear on the underside of the leaf. A pungent odor usually becomes prevalent in potato fields before the more obvious symptoms of late blight are apparent.

The tubers become affected later in the season. In the early stages, slightly brown or purple blotches appear on the skin. In damp soils the disease progresses rapidly, and the tuber decays either before or after harvest. Tuber infection is followed by secondary invasion by bacteria and fungi. This type of rot is known as "wet rot." When soil is dry, the brown discoloration extends only to a depth of about 1 cm into the tuber; however, in storage, infection progresses throughout the tuber.

Disease Development. Sporangia are produced rapidly on infected leaves at temperatures near 21°C when relative humidity is near 100%. The deciduous sporangia are readily splashed by water or spread by wind. Disease development is favored by cool (16 to 21°C), cloudy, moist weather, during which new sporangia are continually being formed. Slightly warmer weather favors the infection process. If the weather clears and the relative humidity stays low, the progress of the disease is checked, and the characteristic white mycelium and

Table 33.1. Distribution of hosts of *Phytophthora infestans*

Host	Common name	Disease	Geographical distribution
Acer sp.	Maple	Leaf blight	Soviet Union (Pshedetskaya 1968)[a]
Anthocercis viscosa R.	Anthoceris	Leaf blight	Germany (Kuhn 1859)
Aster thomsonii Clarke	Aster	Leaf blight	India (Raj et al. 1976)
Atropa belladonna L.	Deadly nightshade	Leaf blight	United States (Peterson 1942)[a]
Bupleurum maddeni Clarke	Thoroughwax	Leaf blight	India (Raj et al. 1976)
Capsicum annuum L.	Red pepper	Leaf blight	United States (Cox 1948)
Datura metel L.	Datura	Late blight	Israel (Sztejnberg and Wahl 1966)[a]
Datura meteloides DC	Datura	Flower and fruit blight	United States (Vartanian and Endo 1985b)[a]
Datura stramonium L.	Jimsonweed; jamestown weed; thorn apple	Leaf blight	United Kingdom (Hirst and Moore 1957); India (Anon. 1962b)[a]; Israel (Sztejnberg and Wahl 1966); United States (Vartanian and Endo 1985b)[a]
Erigeron multicaulis DC	Fleabane	Leaf blight	India (Raj et al. 1976)
Galinsoga parviflora		Leaf blight	India (Raj et al. 1976)
Geranium nepalense Sweet	Cranebill	Leaf blight	India (Raj et al. 1976)
Hyoscyamus aureus L.		Late blight	Israel (Sztejnberg and Wahl 1966)[a]
Hyoscyamus niger L.	Henbane; stinking nightshade	Leaf blight	England (W. G. Smith 1884); Poland (Garbowski 1913); Germany (Vowinckel, 1926)[a]; United States (Reddick 1928)[a]
Ipomoea hederacea Jacq.	Morning-glory	Leaf blight	India (Raj et al. 1976)
Ipomoea purpurea (L.) Roth	Common morning-glory	Leaf blight	India (Raj et al. 1976)
Lycium chinense Mill.	Chinese matrimony vine	Leaf blight	United States (Vartanian and Endo 1985b)[a]
Lycium halimifolium Mill.	Matrimony vine	Leaf blight	Germany (Vowinckel 1926)[a]; United States (Reddick 1928)[a]; England (Moore 1945)
Lycium obovatum L.		Leaf blight	Germany (Vowinckel 1926)[a]
Lycium turcomanicum Turcz.	Box thorn; matrimony vine	Leaf blight	Germany (Vowinckel 1926)[a]; United Kingdom (Hirst and Moore 1975)[a]
Lycopersicon esculentum Mill.	Tomato	Late blight; damping-off	France (Tulasne 1854); Germany (Kuhn 1859); United Kingdom (W. E. Smith 1881); United States (Thaxter 1989b); Russia (Speschnew 1896); India (Butler 1903); Worldwide distribution by 1930
Mandragora officinarum L.	Mandrake	Late blight	Israel (Sztejnberg and Wahl 1966)[a]
Mirabilis jalapa L.	Four o-clock	Leaf blight	Mexico (Servin 1953; Romero and Fourton 1962; Perches and Galindo 1967)
Nicandra physalodes (L.) Gaertn.	Apple of Peru	Leaf blight	United States (Peterson 1947)[a]
Nicotiana acuminata (R.C. Grah.) Hook	Nicotiana	Leaf blight	United States (Vartanian and Endo 1985b)[a]
Nicotiana clevelandii Gray	Nicotiana	Leaf blight	United States (Vartanian and Endo 1985b)[a]
Nolana humifusa (Gouan) Johnst.	Nolana	Leaf blight	Peru (Turkensteen 1978)
Petunia Juss.	Petunia	Seedling blight	England (W. G. Smith 1884); Australia (Samuel 1932); United Kingdom (Hirst and Moore 1957)
Petunia hybrida Hort.	Petunia	Leaf blight	Sweden (Lagerheim 1891); United States (Peterson 1947, Cox 1948)[a]; United Kingdom (Hirst and Moore 1957); India (Anon. 1962b)[a]; Mauritius (Anon. 1965); Israel (Sztejnberg and Wahl 1969)[a]
Physalis alkekengi L.	Winter cherry; Chinese lantern plant	Leaf blight	Germany (Vowinckel 1926)[a]; United States (Reddick 1928)[a]
Physalis angulata L.	Ground cherry	Leaf blight	United States (Peterson 1947)
Physalis ixocarpa Brot.	Tomatillo	Leaf blight	Mexico (Gandara 1909)
Polygonum alatum Buch-Ham	Smartweed, knotweed	Leaf blight	India (Raj et al. 1976)
Rumex acetosa Linn.	Garden sorrel	Leaf blight	India (Raj et al. 1976)
Salpichroa origanifolia (Lam). Baill.	Cock's eggs	Leaf necrosis	Japan (Hori 1964)
Salpiglossis sp.		Leaf blight	United States (Peterson 1947)[a]
Salpiglossis sinuata Ruiz and Pav.	Painted tongue	Leaf blight	India (Dastur 1913)
Schizanthus sp.	Butterfly flower	Leaf blight	India (Anon. 1962b)
Schizanthus grahamii Gill.	Butterfly flower	Stem, leaf, and bud blight	Germany (de Bary 1876); United States (Reddick 1928)
Schizanthus pinnatus Ruiz and Pav.	Butterfly flower	Leaf blight	United States (Vartanian and Endo 1985b)[a]
Solanum andigenum Juz. and Bukasov.		Leaf blight	Peru (Niederhauser 1953)
Solanum antipoviczii		Blight	Mexico (Reddick 1932)
Solanum atropurpureum Schr.		Leaf, stem, and fruit infection	Sweden (Hammarlund 1933)
Solanum aviculare Forst. (*Solanum laciniatum* Ait.)	Kangaroo apple	Leaf and tuber blight	Germany (Kühn 1859); Australia (Brittlebank 1920); United States (Berg 1926); France (Marchal and Foex 1932); Sweden (Hammarlund 1933); New Zealand (Driver 1957); Czechoslovakia (Brejcha et al. 1959)
Solanum boreale Gray		Leaf blight	Mexico (Niederhauser and Mills 1953)
Solanum brachycarpum		Leaf blight	Mexico (Toxopeus 1960)
Solanum bulbocastaneum Ait.		Leaf blight	Germany (Kuhn 1859); Australia (Brittlebank 1920); France (Marchal and Foex 1932); Sweden (Hammarlund 1933)
Solanum caldasii Humb. and Bonpl.		Leaf blight	France (Marachal and Foex 1932)

(*continued on next page*)

Table 33.1. (*continued*)

Host	Common name	Disease	Geographical distribution
Solanum capripense Kunth.		Leaf blight	Ecuador (Lagerheim 1891)
Solanum cardiophyllum Lindl.		Leaf blight	United Kingdom (Smith 1884); Mexico (Niederhauser and Mills 1953)
Solanum commersonii Dun.		Leaf blight	France (Labergerie 1904); England (Jones 1905); United States (Stuart 1906)
Solanum crispum Ruiz and Pav.		Leaf blight	England (Beaumont and Staniland 1937); India (Butler and Jones 1949)
Solanum demissum Lindl.	Wild potato	Leaf blight	United Kingdom (Lindley 1848); Mexico (Gandera 1909)
Solanum dulcamara L.	Bittersweet, nightshade	Leaf blight	Germany (Corda 1847); Mexico (Gandara 1909); United Kingdom (Smith 1913; Hirst and Stedman 1960); Russia (Bondartzeva-Monteverde 1926); United States (Reddick 1928; Cox 1948)
Solanum edinense Berthault.		Leaf blight	United Kingdom (Salaman 1910); Germany (Broili 1921)
Solanum ehrenbergii		Leaf blight	Mexico (Fernández and Galindo 1989)
Solanum etuberosum Lindl.		Leaf blight	Netherlands (Bergsma 1845); France (Tulasne 1854); United States (Stuart 1906); United Kingdom (Salaman 1910)
Solanum fendleri Gray.		Leaf blight	United States (Reddick 1928)[a]
Solanum goniocalix Juz. and Bukasov		Leaf blight	Peru (Bazan de Segura and Carrera 1953)
Solanum humboldti Dun.		Leaf blight	Germany (Röder 1935)
Solanum incanum L.		Leaf blight	Kenya (Nattrass and Ryan 1951); Israel (Sztejnberg and Wahl 1966)[a]
Solanum indicum L.		Leaf blight	New Zealand (Driver 1957)
Solanum iopetalum (Bitter) Hawkes		Leaf blight	Mexico (Niederhauser and Mills 1953)
Solanum jamesii Torrey		Leaf blight	United States (Reddick 1928)[a]; Mexico (Nierderhauser and Mills 1953)
Solanum laciniatum Ait. (*S. ariculare* G. Forst.)	Kangaroo apple	Leaf blight	New Zealand (Driver 1957)
Solanum maglia Molin.	Darwin potato	Leaf blight	Germany (de Bary 1861); United Kingdom (Jones 1905); United States (Stuart 1906)
Solanum marginatum L.		Leaf, stem, and fruit infection	Sweden (Hammarlund 1933)
Solanum medians Bitt.		Leaf blight	Peru (Ochoa 1955)
Solanum melongena L.	Eggplant	Fruit rot and calyx blight	Germany (Corda 1847); United States (Haskell 1921); France (Simonet 1925); Russia (Bondartzeva-Monteverde 1926); Santo Domingo (Ciferri 1927b); Rhodesia (Bates 1959)
Solanum muricatum Ait.	Sweet pepino; Peruvian cucumber; pear melon	Leaf blight	Ecuador (Lagerheim 1891); Peru (Revilla 1963); Colombia (Guzman-Naranjo 1966)
Solanum nigrum L.	Black nightshade; poison-berry	Leaf necrosis	Germany (DeBary 1863); United States (Peterson 1947); United Kingdom (Hirst and Stedman 1960); Japan (Hori 1964)
Solanum panduraeforme Drege		Leaf blight	Kenya (Nattrass and Ryan 1951)
Solanum pinnatisectum Bitter.		Leaf blight	Mexico (Niederhauser and Mills 1953)
Solanum pyracanthum Jacq.		Leaf, stem, and fruit infection	Sweden (Hammarlund 1933); Israel (Sztejnberg and Wahl 1966)[a]
Solanum racemigerum Zodda.		Leaf blight	Germany (Röder 1935)
Solanum rostratum Dunal	Buffalobur	Leaf blight	United States (Peterson 1947)[a]
Solanum sambucinum Rusby.		Leaf blight	Mexico (Niederhauser and Mills 1953)
Solanum sarachioides Sendt.		Leaf and stem blight	United States (Gardner and Yarwood 1942; Vartanian and Endo 1985b)
Solanum senecioides Domb. ex Dun.		Leaf blight	Peru (Turkensteen 1978)
Solanum simile Muell.		Leaf and stem blight	New Zealand (Driver 1957)
Solanum sisymbriifolium		Leaf blight	United States (Vartanian and Endo 1985b)[a]
Solanum stoloniferum Schlect.		Leaf blight	France (Tulasne 1954); United States (Stuart 1906); Mexico (Niederhauser and Mills 1953)
Solanum tomatillo Phil.	Tomatillo	Leaf and stem blight	Germany (Röder 1935)
Solanum tuberiferum Dun.		Leaf blight	Peru (Turkensteen 1978)
Solanum tuberosum L.	Potato	Late blight	Europe (Eriksson 1917, 1918 , noted occurrence from 1830 to 1842); United States (G. Smith 1913, noted occurrence in 1842); Taiwan (Sawada 1919); world-wide by 1930 (Tucker 1933)
Solanum utile Klotzsch		Leaf blight	Germany (Munter 1849)
Solanum verrucosum Schl.		Leaf blight	France (Tulasne 1854); Mexico (Reddick 1932)
Sonchus oleraceus Linn.	Compositae	Leaf blight	India (Raj et al. 1976)
Tilia sp.	Lime tree	Leaf blight	Soviet Union (Pshedetskaya 1968)[a]
Withania somnifera (L.) Dun.		Late blight	Israel (Sztejnberg and Wahl 1966)

[a] Artificially inoculated host.

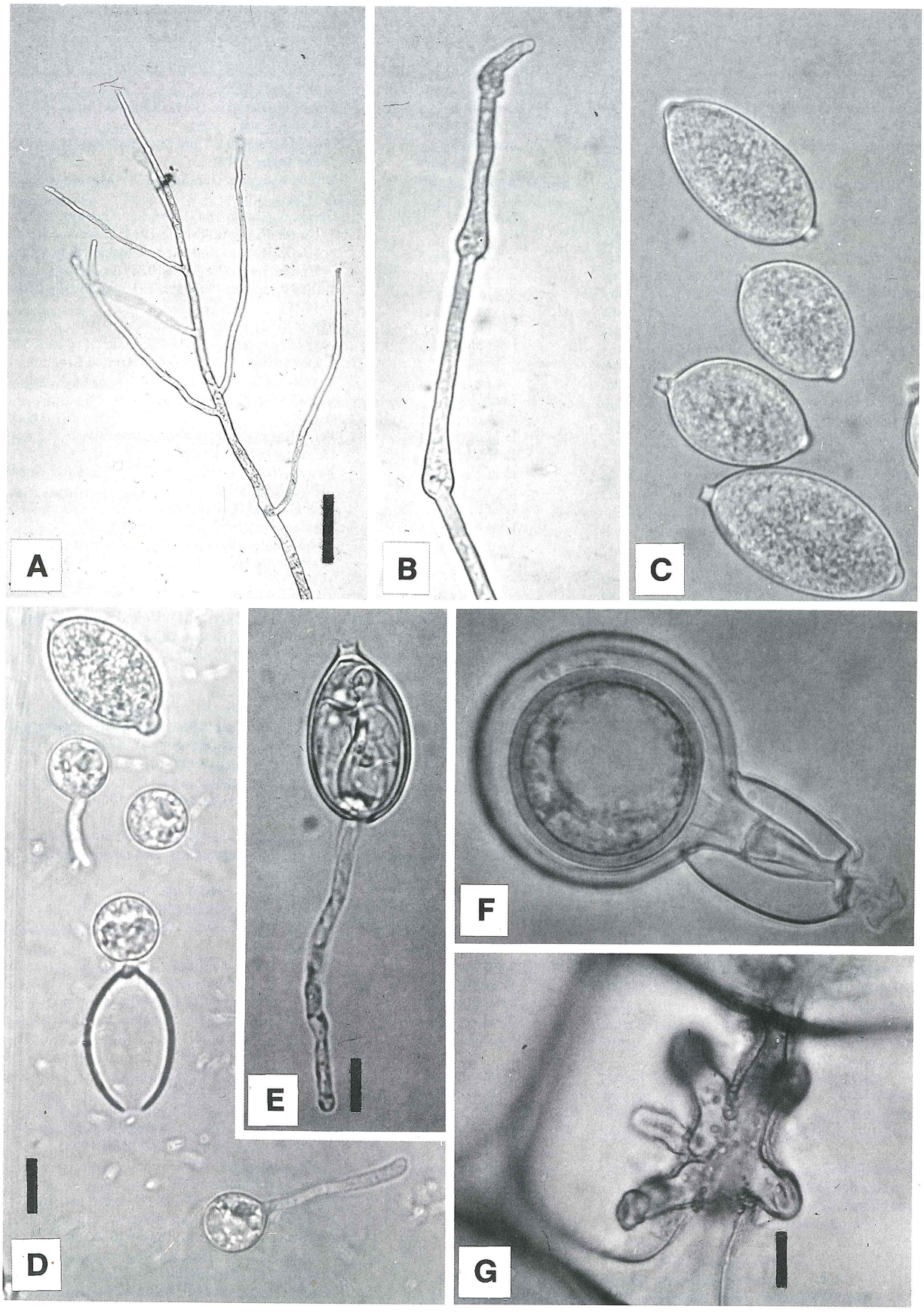

Figure 33.1. *Phytophthora infestans.* **A,** Sporangiophore; **B,** sporangiophore branch showing swellings at successive sites of sporangium formation; **C,** sporangia germinating by zoospores (**D**) and germ tube (**E**); **F,** oospore with antheridium; **G,** haustoria within tuber cells. Bar in A = 50 μm; bars in D, E, and G = 10 μm. (Reproduced by permission of the American Phytopathological Society, courtesy Hooker 1981)

sporangia are not produced on the undersides of the leaves. With the return of cool, moist weather, the fungus again becomes active and symptoms reappear. Long periods of cold weather are unfavorable for disease development, however. In warmer regions, the development of late blight depends on how long the temperature remains below 21°C. An extensive discussion of the environmental conditions favoring late blight of potato worldwide is given by Cox and Large (1960). The role of cultural factors on the development of late blight is discussed at more length in Chapter 5.

The mechanisms by which *P. infestans* persists in soil and becomes the primary source of inoculum have long been controversial (Andrivon 1995; Gregory 1983). According to many reports, inoculum survives in infected tubers and in areas where both A1 and A2 mating types exist (e.g., in Mexico). Oospores are considered to be a source of both inoculum and pathologic variability (see Andrivon [1995] for an intensive review of this subject). Andrivon (1995) states that despite 150 years of research, there are more questions than answers about the soil stages in the life cycle of *P. infestans.* The worldwide migration of a new and diverse group of genotypes and the ability to utilize a wider range of genetic markers (S. B. Goodwin et. al. 1995) should allow researchers to resolve many questions about the role of oospores in the soil phase of the life cycle of *P. infestans.* The reports of Chang and Ko (1991) and Drenth et. al. (1995) indicate that spores can form, survive, and germinate.

P. infestans is disseminated by airborne sporangia (distant spread) and from point sources by water splash of sporangia from infected leaves. Thus, epidemics can result from the dispersal of inoculum from distant fields or from infected seed pieces (Hirst and Steadman 1960).

The following conditions usually precede late blight epidemics: 1) night temperatures below the dew point for at least 4 h; 2) low night temperature that does not drop below 10°C; 3) the mean period of cloudiness not less than 0.8 of a day; and 4) rainfall of at least 0.1 mm on the following day.

Tubers, especially those near the soil surface, may be infected by sporangia or zoospores that wash downward from infected leaves during rain storms. Infection appears to be limited to periods when soil temperature is 18°C or below (Sato 1979).

Forecasting of late blight probability is complex. Hartill et al. (1990) determined the times required for production of lesions on leaves, for production of sporangia during periods of leaf wetness, and for development of new lesions over a range of 5 to 24°C. Equations were developed by use of the data to relate time to sporulate with temperature. A 2-h break in leaf wetness at any time during the first 3 h of incubation after inoculation reduced the numbers of lesions formed. Michaelides (1985) reported a simulation model of the production of disease by *P. infestans* on potato. His discussion contains a basic literature review and a mathematical model.

Control. The application of fungicides prior to infection in the field has been one of the main approaches to control (Fry 1977b; Fry et al. 1979; Rich 1983; Schwinn and Margot 1991). Chemical control has been greatly facilitated by the advent of late blight forecasting (Bourke 1970; Krause et al. 1975; Krause and Massier 1975; MacKenzie 1981; Fry and Fohner 1985; Fry and Doster 1991), which has resulted in more timely and more economical use of fungicides (see Chapter 7 for further discussion of chemical control and disease forecasting).

Until the late 1970s, fungicides commonly used for control of late blight included Bordeaux mixture, which is somewhat toxic to foliage, other copper-based compounds that are less toxic, pthalimides, and the dithiocarbamates (Rich 1983). The dosage rates and application times varied with the weather conditions (Schwinn 1983; Heitefuss 1989). Since then, improved control has been facilitated by use of the systemic phenylamide fungicides such as metalaxyl, which are usually applied with a protective fungicide such as mancozeb to help suppress the development and increase in the population of phenylamide-resistant biotypes of *P. infestans* (Young et al. 1979; Schwinn 1983; Schwinn and Morton 1990). See Chapter 7 for a full discussion of control of *P. infestans* with fungicides and in particular Table 7.6 for a listing of references on control with phenylamide compounds such as metalaxyl and furalaxyl. Fosetyl-Al, although effective against several other *Phytophthora*-caused diseases, has not been found to be effective for control of late blight (Chapter 7). Integration of polygenic host resistance and use of fungicides, as discussed by Fry (1975, 1977a, b, 1983) and Fry et al. (1983), can be efficacious.

Although metalaxyl and other phenylamide fungicides are the most highly effective of any that have been developed for control of late blight (see Chapter 7), the development of resistance within populations of *P. infestans* has become a limiting factor in the use of this fungicide. In the Netherlands, 11% of the isolates from community gardens where metalaxyl had not been used were resistant, but 45% of the isolates were resistant in commercial fields where metalaxyl had been used (Fry et al. 1991). Development of resistance to metalaxyl has been alleviated to some extent by combining a broad-spectrum fungicide, such as mancozeb, with metalaxyl and by limiting the number of times fields are sprayed with metalaxyl. Use of metalaxyl on seed fields is not allowed in some countries because of the danger that metalaxyl-resistant biotypes might be disseminated with seed tubers. See Chapter 7 (Table 7.7 for references on resistance to metalaxyl) for a more complete discussion of resistance and Chapter 3 for a discussion of the genetics of metalaxyl resistance showing that resistance is associated with a single gene. Principles governing the use of phenylamide fungicides as described by Delp (1980, 1984, 1988) and Delp and Dekker (1985) are reviewed in Chapter 7.

The development and wide dissemination of the "new" *P. infestans* biotypes (e.g., US7 A2 and other isolates) (S. B. Goodwin et al. 1995) to many areas of the world during the past decade is one of the most serious threats to the control of late blight since the disease was first noted during the 1840s. Many of these new biotypes, which Fry and his colleagues (e.g., Fry and Goodwin 1995; see other citations in Chapters 3 and 7) have detected by allozyme and DNA fingerprinting technology, are of the previously unknown A2 mating type, are more aggressive, and (a more serious problem) are resistant to metalaxyl, the most effective late blight fungicide developed to date. These new biotypes are now present on both potatoes and tomatoes in California (S. B. Goodwin et al. 1995) and in other western states (Deahl et al. 1991, 1995) where late blight had not previously been considered a serious threat to production. Serious attention will have to be applied by regulatory and experiment station agencies to overcome this problem. Some fungicides, to which resistance in *P. infestans* has not developed, are discussed in Chapter 7. Gisi (1991) presents research data that indicate that application of two or three fungicides elicits a synergistic response to control late blight in Europe.

Since sporangia of *P. infestans* increase and spread rapidly and over long distances (Hirst 1953; Hirst and Moore 1957; Hirst and Steadman 1953), attention to the problem of control should be given not only by individual growers but by regulatory agencies in large geographical regions.

Breeding for race-specific resistance to late blight was once considered an efficacious approach to control but has since proved to be of only limited use because many pathologic (physiologic) races of *P. infestans* have the ability to attack new cultivars with single-gene resistance. The early breakthrough involving incorporation of single dominant genes for resistance to the various races of *P. infestans* into *Solanum tuberosum* from other solanaceous species such as *S. demissum* was initially highly successful, but new pathogenic races of the fungus developed in the field that rendered the new cultivar susceptible. As the population of the new race increases and becomes dominant in the following years, the new cultivar is no longer resistant (see Chapters 3 and 7 for a more detailed discussion of race-specific and horizontal resistance).

General (horizontal) resistance, which is more stable but does not confer as high a level of resistance as single-gene resistance, has been identified in many cultivars (Niederhauser 1961, 1962, 1991, 1993; Niederhauser and Mills 1953) and after the failure of single-gene-type vertical resistance, has become the best approach to genetic control (Umaerus 1983; Dowley et al. 1991). As explained by Vanderplank (1963) and MacKenzie et al. (1983), general resistance, also referred to as rate-limiting, multigenic, field, or horizontal resistance (Chapter 6), reduces the rate of the epidemic and is equally effective against all races. General resistance is more stable because usually it is conditioned by more than one gene and there is less probability that mutations in populations of *P. infestans* will simultaneously occur in nature in more that one gene at a time. Since plants with general resistance are not immune, less selection pressure is placed on the population of *P. infestans* than by cultivars with a single gene for immunity. For instance, when the plant has immunity, the only isolates that will survive are the virulent mutants, because *P. infestans* does not sporulate on race-specific resistant plants. In most regions of the world, breeding programs now emphasize the development of general resistance instead of race-specific resistance. Methods of assessing general resistance in breeding lines have been described by Main and Gallegly (1964) and Malcolmson (1976). For detailed discussions of race-specific and multigenic or general resistance in relation to late blight of potato, see Gallegly and Niederhauser (1959), Vanderplank (1968), Day (1973), Umaerus (1969, 1970), and Umaerus et al. (1983) and Chapter 6, where more citations are given and the subject is discussed further. International cooperative ventures for the control of late blight of potato are discussed by Niederhauser (1993), a pioneer in the research effort to control late blight in Mexico and other regions around the world.

Many cultivars with general resistance (for example, Sebago) are available commercially in different regions of the world (O'Brien and Rich 1976; Rich 1983). Since some cultivars are better adapted to certain localities, such information should be obtained from local advisors before selecting resistant cultivars. Plants with general resistance require less fungicide than susceptible cultivars (Fry 1975, 1977a, 1982; Fry et al. 1983). After the crop matures, vines should be killed by an acceptable chemical method to prevent infection of tubers by inoculum in leaves and stems (Rich 1983).

Late Blight of *Lycopersicon esculentum* Mill. (Tomato)

Late blight of tomato and its control is described by J. B. Jones et al. (1991).

Leaf lesions are water soaked with definite margins and a gray green color. The infected tissue becomes brown and necrotic, and the leaf dies. Stems and petioles are also affected. Eventually, the plant will be killed if the disease is not controlled. The foliage symptoms are similar to those on potato. On tomato fruits, gray green, water-soaked spots enlarge rapidly. The lesions then turn dark brown, and although the diseased areas become wrinkled, the rotted tissue remains firm. The unaffected parts of the fruit ripen to a normal red color. Occasionally, the lesion may have rings in a zonate pattern that superficially resemble those of buckeye rot caused by *P. parasitica;* however, in fruit rot caused by *P. infestans,* the rings are much closer together. Seeds from infected fruit often give rise to infected tomato seedlings (Vartanian and Endo 1985a).

Disease Development. Disease development is similar to that of late blight on potato. Since rainfall during summer months is rare in California, late blight was seldom a problem before 1979. In certain areas, however, dew formation on staked tomatoes is sufficient to allow late blight to become limiting to production (Vartanian and Endo 1985a, b). Late blight became a problem after 1979 probably because of a series of relatively mild winters in which frost did not kill overwintering volunteer solanaceous host plants and because of intensive tomato-cropping practices (three crops per year in San Diego County). Late blight was found in the field throughout the year on volunteer tomato plants and on plants grown in home gardens. The prevalent tomato races were T0 and T1. Only the A1 mating type occurred in southern California. Race T1 grew and sporulated more rapidly than race T0 (Vartanian and Endo 1985b). In 1993, metalaxyl-resistant A2 isolates of *P. infestans* were found in California (M. D. Coffey, *personal communication*).

Control. Bordeaux mixture used as a foliar spray before infection is effective but is toxic to the foliage of tomato, so other, more insoluble copper-based compounds, pthalimide, dithiocarbamate, or phenylamide fungicides are more often used. The fungicides are applied on the basis of late blight forecasts similar to those employed for potato. Y. Cohen et al. (1979) found that the systemic phenylamide fungicide metalaxyl, used as a single soil drench, controlled late blight of potted tomato plants and gave protection from blight for at least 6 weeks. Many subsequent citations listed in Chapter 7 indicate that the phenylamide fungicides (e.g., metalaxyl) are effective for control of late blight.

Although metalaxyl is highly effective, resistance to metalaxyl within populations of *P. infestans* on potato has become a problem in Europe and now occurs widely in California (M. D. Coffey, *personal communication*). The addition of the broader spectrum fungicide mancozeb to metalaxyl or other phenylamide fungicides may at least partially alleviate this buildup of biotypes with resistance to *P. infestans.* This subject is discussed further in Chapters 3 and 7.

J. B. Jones et al. (1991) note that the disease-forecasting systems, such as the Hyre system, which predicts disease onset on the basis of temperature and rainfall, the Wallin system, which predicts disease on the basis of temperature and humidity, and BLITECAST, which integrates the two systems in a computer program, help to determine when application of a fungicide will be most effectual. Use of forecasting systems is discussed in Chapter 7.

Although cultivars with resistance to the known tomato races of *P. infestans* are commercially available, host resistance is not an important element of control in the United States (J. B. Jones et al. 1991). The early breeding work was complicated by the presence of potato and tomato races of *P. infestans* and the possible interrelationships between the two (Berg 1926). It is now known that some potato races behave as different tomato races when inoculated on tomato plants. Races T0 and T1 are recognized to be most prevalent in the

United States; T1 is the more aggressive. Race characteristics of a given isolate should be considered separately on the two hosts, since tomato genes for resistance to *P. infestans* differ from those known in the potato (Wilson and Gallegly 1955; Gallegly and Niederhauser 1959).

Some new biotypes of *P. infestans* can be aggressive on both tomato and potato. Legard et al. (1995) state, "Our results were consistent with the hypothesis that increased aggressiveness on tomato (*Lycopersicon* spp.) has evolved in isolates already pathogenic to *Solanum* spp." Their report also shows that some isolates from potato are relatively nonaggressive to tomato.