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Potato and Tomato Late Blight Caused by *Phytophthora infestans*:

Traced back to its Andean origin (47,58), *Phytophthora infestans* (Mont.) de Bary (Oomycota, Stramenopiles) caused the first recorded instance of late blight (LB) in Philadelphia and New York City potato (*Solanum tuberosum*) crops in 1843. Due to weather patterns, winds spread the dehiscent sporangia (131) to neighboring states. By 1845, LB was found from Illinois to Nova Scotia and from Virginia to Ontario. The disease then crossed the Atlantic Ocean with a shipment of infested seed potatoes from the United States to Europe, mainly to Belgian farmers, in 1845 (131). Once *P. infestans* reached Ireland, a country that was strongly dependent on potatoes as a main source of food and was predisposed to adverse political, social, and economic factors (94,117), wide infestation of potato by *P. infestans* resulted in a near-complete destruction of the crop. This led to the death of one million people and the displacement of another million refugees (135,167), many of whom emigrated to New York State. Continued spread of *P. infestans* in subsequent years resulted in a worldwide distribution of LB by the beginning of the twentieth century and global devastation of potato and tomato crops (77).

Once an unprotected potato field or tomato crop (field, greenhouse, and/or plastic-cover cultures) is infected by *P. infestans*, the whole crop can be devastated within 7 to 10 days (47; Fig. 1). Economic losses may be in the form of reduced yield, lower quality of the fruit (such as low specific gravity), diminished storability, and increased cost associated with fungicide applications (42; Fig. 1). Current potato yield losses caused by LB reach as much as \$6.7 billion annually (4,28,60), depending on the severity of disease, plant variety, and management practices. In 2009, in the United States, the total potato losses approximated \$3.5 billion, while the total yield losses in fresh and processing tomatoes reached \$46 million and \$66 million, respectively (http://www.nass.usda.gov/Data_and_Statistics/Quick_Stats/). Of these, up to half could be

attributed to losses due to LB (72). These represent approximately 7% yield losses of the U.S. tomato production (fresh market and processing), and up to 3.5% yield losses in potato over the last two decades (http://www.nass.usda.gov/Data_and_Statistics/Quick_Stats/). In tomato, fruit infection may range from 41 to 100% in unprotected fields and from 12 to 65% in plots protected with systemic fungicides (42). Polish tomato production has suffered significantly from rampant outbreaks of LB over the past several years, often causing yield losses of up to 100% (113,181; Fig. 1). Social impact of LB is illustrated by the significant media and internet coverage, for instance by using Google Trends to track the usage of the word “blight” as an internet search-word. Web searches peaked in 2005, 2007, and 2009, respectively, during the LB epidemics (170). These were accompanied by higher fungicide applications worldwide, indicating the need for crop protection-related expenditures, and environmental safety concerns. Predictions that fungicide applications needed to suppress LB in 2040 to 2065 would increase by 20 to 25%, compared to that required in 1977 to 2008 (36), further add to the fiscal and environmental costs related to LB control.

The success of *P. infestans* as a pathogen originates from its effective reproduction in both asexual and sexual forms. In the asexual form, *P. infestans* produces thousands of sporangia per lesion on sporangiophores, which are indeterminate structures that aid in air dispersal of the sporangia using passive movement of wind, rain, or wind-blown rain (34,145). The sporangium can germinate directly at temperatures above 15°C and quickly develops mycelial growth and subsequent sporangia on leaf, stem, and fruit tissues (109,136). At lower temperatures, sporangia forgo mycelia growth by directly forming and releasing zoospores (asexual spores), which then germinate and cause new infections at an even more rapid pace (172). Depending on environmental conditions, regeneration time can be short and the entire cycle repeated in 5 to 7 days (<http://www.biology.ed.ac.uk/research/groups/jdeacon/microbes/blight.htm>).

P. infestans is a heterothallic organism with two known mating types, A1 and A2 (22). When the mycelia of the two mating types interact, sexual reproduction may occur by means of oospore formation (34). Oospores are large, thick-walled spores that enable the organism to survive for long periods in plant debris or soil outside the living host plant (83), a trait not shared by the asexual spores. Until recently, only the A1 mating type was detected in

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potato growing areas outside Mexico, suggesting that sexual reproduction did not play a significant role in the disease cycle. Researchers have observed both mating types in many production areas in the United States and around the world (48,150). The presence of both mating types allows for sexual recombination and production of new and possibly more aggressive isolates. This has also made LB disease management efforts even more challenging.

Control of LB is therefore indispensable. The common methods employed to counter infestation include cultural practices, fungicide sprays, and use of resistant cultivars (15; Fig. 1). LB control via cultural practices alone can be very challenging, and *P. infestans* could be particularly destructive in areas where both tomatoes and potatoes are grown year-round, as for example in the highland tropics of Africa, South America, Asia (29), and Europe

(3,26,131). Chemical control has increased in popularity in recent years (47), and two main groups of compounds have been used routinely, including protectant (e.g., chlorothalonil, dithiocarbamates, and triphenyl tin hydroxide) and systemic fungicides (e.g., phenylamides, such as metalaxyl/mefenoxam, aliphatic nitrogen fungicides such as cymoxanil, and morpholine fungicides such as dimethomorph). Chemical treatments also can be ineffective, in particular when the environmental conditions are highly conducive to disease development (57,183). Moreover, improper applications of phenylamides have created a selective pressure on the pathogen, leading to the establishment of phenylamide-resistant isolates of *P. infestans* in many countries (56,57). These issues, as well as economic and environmental safety considerations, necessitate careful adoption of effective and sustainable disease control measures

Fig. 1. *Phytophthora infestans* affecting a tomato field stresses the need for late blight (LB) control. Tomatoes grown in field (Research Institute of Horticulture, Skierniewice, Poland, 2008) in alternating rows of susceptible (*Solanum lycopersicum* var. Rumba) and resistant (*S. lycopersicum* LB11 developed at the Institute of Horticulture, Skierniewice, Poland) accessions show clear difference in reaction to LB. Inset: A close-up showing yield (tomato fruit) losses due to LB.

(56), including the development and integration of commercially acceptable cultivars with genetic resistance against LB (71,98; Fig. 1).

Many recent reviews of LB addressed the oomycetal effectors and their roles in pathogenesis (2,11,67,70,170). This review aims to summarize the recent advances in cellular and molecular understanding of and breeding for resistance against LB in potato and tomato. Tremendous progress has recently been made in these areas through the availability of the second generation sequencing of *Phytophthora* genomes (60,128) and the sequencing of the host plant genomes: The potato genome of ~844 Mbp (~39,000 genes, mostly located within the ~570 Mbp of euchromatin; 152) and the tomato genome of ~950 Mbp (~40,000 genes, >90% of which lie within the ~220 Mbp of euchromatin; 115,177). With these tools in hand, it is expected that the molecular basis of potato and tomato resistance against LB will soon be discerned and the information will be utilized in breeding programs (41,78).

P. infestans Pathogenesis: A Glance

Basic details of pathogen infection and defense response during *P. infestans* attack have been known for some time (13,33,145). Still, the more recent integration of cellular and molecular approaches has revolutionized our understanding of oomycete–host plant interactions. In particular, the immunocytochemistry and green-fluorescence protein (GFP)-tagging studies have revealed the location of plant receptors involved in pathogen detection and mechanisms underlying the basal defense (33,34). Severe LB epidemics may occur when *P. infestans* grows and reproduces rapidly on the host plant, in particular when tomatoes are part of the agroecosystem (34). *P. infestans* adopts a two-step infection mode, typical of hemibiotrophs. An early phase of infection, in which the pathogen requires living host cells and obtains nutrients through development of haustoria, is followed by extensive necrosis of host tissue resulting in colonization and sporulation. The life cycle of

P. infestans involves differentiation into as many as 11 different cell types. These cell types are highly specialized for life cycle stages involved in sexual and asexual reproduction, propagule dispersal, spore germination, host penetration, and biotrophic or necrotrophic phases of infection (9).

The early stages of disease symptoms are easily missed due to not all plants being affected simultaneously. Initial infection symptoms, including small lesions on leaf tips and plant stems, are visible only after 3 to 4 days, and in some cases reach only 1 to 2 mm in diameter. As the pathogen penetrates the plant tissue, lesions enlarge in size and white mold appears on the leaf underside (abaxial surface). The entire plant may collapse in 5 to 10 days.

P. infestans Pathogenesis: Zoom-in to Cellular Level

Formation of oospores, the sexual spores originating from mating between *P. infestans* A1 and A2 mating types, may lead to the generation of more aggressive clonal lineages (53). First observed in Central Mexico (51), the oospores may play a key role in the epidemiology of LB (53,95) and serve as a persistent source of inoculum in the field, similar to potato tuber-borne inoculum (183). This has been demonstrated by the ability of *P. infestans* oospores to initiate the disease *in situ* or in detached leaves in the laboratory (95).

On the other hand, the asexual sporangia serve as the major vehicle driving epidemics during the season. Sporangia release zoospores under favorable weather conditions, known as the Smith period, which is defined as tissue wetness with relative humidity greater than 90% lasting for at least two consecutive days, and on each day at least 11 hours with moderate temperatures of 10°C or above (34,87,95). Sporangia are capable of sensing the environment and regulating germination. Germination occurs either through the direct extension of germ tubes or by zoosporogenesis. The latter is stimulated by cool and moist conditions, and it is important because it extends the range in which infections could occur. Sporangia develop many organelles that are absent in hyphae, such as large peripheral vesicles, encystment vesicles, kinetosomes, and flagella (31,68). Zoosporogenesis involves cleavage of the multinucleate sporangial cytoplasm by nucleus-enveloping membranes, assembly of flagella, dissolution of the sporangial papilla, and expulsion of uninucleate zoospores (65,68). Several of the early-induced genes encode participants in vesicle movement that may aid in assembling these structures. Other early-induced genes encode components of the spore-specific vesicles or organelles themselves including over 70 flagella-associated, as well as a thrombospondin-like encystment protein (134). Under favorable field conditions, sporangia can maintain viability for as long as a week, although levels of mRNAs enabling germination (66,82) and the germination dynamics may change during this period (137,173).

P. infestans preferentially sporulates in planta during dark periods. Studies have demonstrated that continuous light inhibits sporulation (33), although varying results have been reported for other members of the genus (132). As there is no evidence for a light-regulated clock in *Phytophthora*, this suggests that a plant-derived signal generated by the day–night cycle influences sporulation. Similar phenomena are reported for other oomycetes (66,82,130). At the plant surface, *P. infestans* zoospores maneuver so that their ventral surface faces the plant before the flagella are detached and proteins from three different cortical vesicles are secreted onto the zoospore surface (63). By doing so, the spores form walled cysts (Fig. 2, A: ii), which germinate (Fig. 2, A: iii) from the center of the ventral surface, allowing the hyphae to grow along the anticlinal walls between epidermal cells to form appressoria for breaching plant cells (Fig. 2, A: iv; 39,64). Occasionally, the multinucleate sporangia can also initiate infections (65). Mature sporangia may preform the proteins needed for zoosporogenesis and encystment (82,127).

During the early biotrophic growth phase in *P. infestans*, nutrients are obtained from living plant cells. This is achieved by forming a penetration peg, which pierces the cuticle and enters an epi-

Fig. 2. Schematic view of the infection cycle of *Phytophthora infestans* on **A**, susceptible, and **B and C**, resistant plants. **A**, (i) zoospore, (ii) cyst, (iii) germinating cyst, (iv) germinated cyst with appressorium, (v) germinated cyst with appressorium and infection vesicle, (vi) haustorium, (vii) sporangium. **B and C**, Similar to panel A except that plant cells undergoing hypersensitive death are shown in red. The hypersensitive response (HR) can include groups of plant cells (**B**) or 1 to 2 cells (**C**) depending on the genotypes of plant and pathogen. After: (87).

dermal cell to form an infection vesicle (Fig. 2, A: v). Formation of digit-like haustoria (i.e., appressoria-like structures) by the branching hyphae, and the subsequent secretion of enzymes that degrade components of the plant cell wall (Fig. 2, A: vi; see below) further facilitate the nutrient access. Haustoria develop within the boundary of the plant cell wall, although they remain outside the host cell plasma membrane (66). The haustoria are specialized for nutrient uptake from the host cell. During necrotrophic growth, nutrients required for pathogen growth and reproduction are obtained from dead and dying cells in the necrotic lesions that develop as the pathogen colonizes the plant. As the infected tissue necrotizes, the mycelium develops sporangioophores that emerge through the stomata to produce numerous asexual sporangia (Fig. 2, A: vii; 66).

P. infestans–Host Plant Interplay

With over 240 Mbp encoding a mere 18,155 genes in *P. infestans* (60,82,128,129), its genome exhibits extremely discontinuous distribution of gene density. Hundreds of putative disease-effector genes localize to the expanded, repeat-rich and gene-sparse regions of the genome, which constitute ~74% of the genome size. In contrast, the housekeeping “core ortholog” genes occupy the repeat-poor and gene-dense regions of the genome (60,128). Additionally, both seem to have evolved at different paces, with the gene-sparse regions experiencing accelerated rates of evolution. Distribution patterns of the pathogen genes, which are induced during preinfection and infection stages, indicate enrichment in genes located in these gene-sparse regions (9,128,129). In marked contrast, the slowly evolving gene-dense regions are enriched in genes induced in sporangia (60,128,129). This distinct genomic environment is thought to contribute to *P. infestans*’ evolutionary potential by promoting genome plasticity, thus enhancing genetic variation of effector genes leading to host adaptation (128,129).

Similar to other pathogen effectors, the effectors secreted by the haustoria of the invading *P. infestans* can be differentiated into two main categories, depending on their site of action: Apoplastic effectors (extracellular) and cytoplasmic effectors (translocated across the plant cell plasma membrane). Extracellular effectors remain in the apoplastic space where they interact with three groups of extracellular host molecules: (i) Cell walls, (ii) host plant–derived proteases, and (iii) defense-response network.

(i) Cell-wall degrading enzymes enable the initial penetration of the plant surface and the subsequent colonization of plant tissues. *P. infestans* genome carries more than 20 genes encoding endopolygalacturonases or β -glucanases that degrade pectin and β -glucans, respectively (111). Cell-wall degrading enzymes are released as a temporal cascade, with pectinases often being the first to be secreted. Digestion of pectins leads to plant cell separation, tissue maceration, and exposure of other cell wall components for degradation (67,82).

(ii) *P. infestans* effector proteins that inhibit enzymatic activity enable protection against plant-derived hydrolytic enzymes (including glucanases, chitinases, and proteases; 149). For example, glucanase and chitinase inhibitors arrest the production of potent elicitors (1,70). In order to inhibit the action of plant proteases synthesized in response to pathogen attack, there are 14 members in the extracellular protease inhibitor (*EPI*) gene family in *P. infestans*. Two of these genes (*EPI1*, *EPI10*) are up-regulated during tomato infection and inhibit the tomato protease P69B (154,155). Four members of cystatin-like protease inhibitors (*EPIC*; 157) family have been identified in *P. infestans*, two of which (*epiC3*, *epiC4*) have orthologs in *P. sojae* and *P. ramorum*. Both *epiC1* and *epiC2* are up-regulated during tomato infection, and *EPIC2B* inhibits a tomato papain-like cysteine protease, PIP1, an enzyme that is related to the tomato protein Rcr3 (147,157).

(iii) Elicitors and toxins are apoplastic *P. infestans* effectors of a diverse range of small cysteine-rich proteins, where the disulfide bonds are likely to help stabilize the proteins in the apoplastic environment. Elicitins induce hypersensitive response (HR)-dependent cell death and other biochemical changes associated with defense

responses in *Nicotiana* spp. For example, strains of *P. infestans* deficient in the INF1 elicitor are pathogenic on the nonhost plant *Nicotiana benthamiana*, which suggests that the INF1 protein conditions avirulence at the species level (70,88). INF1 is specifically expressed during early biotrophic growth in tomato and suppresses the action of necrosis-inducing effectors (secreted by the oomycetes during necrotrophic growth) as well as programmed cell death (90,139). Although elicitors trigger plant defense responses in *Nicotiana* spp., they play an important role in oomycete pathogenicity through their ability to bind plant sterols and act as sterol carrier proteins (120). As *Phytophthora* cannot synthesize sterols and must assimilate them from external sources, elicitors are believed to serve an essential role in *Phytophthora* growth and development (74).

Cytoplasmic effectors, on the other hand, get across the host cell’s plasma membrane and function inside the plant cell, where they orchestrate metabolic changes that favor pathogen development (13,89). Out of 1,415 proteins making up the total secretome (129), the 563 genes putatively encoding the cytoplasmic effectors in *P. infestans* (60,128) constitute a highly versatile and dynamic arsenal of proteins, which are employed by the pathogen to manipulate its hosts, target plant molecules, and alter its processes (119,129). Proteins predicted to serve as the cytoplasmic effectors can be differentiated in two protein families: (i) RXLR (amino acid motif within the respective proteins) and (ii) CRN (crinkling and necrosis).

(i) In *P. infestans*, an N-terminal RXLR motif and its functional variants facilitate transport of effectors belonging to this family into the infected host cell (67,70,162,178,179) by binding to phospholipid, phosphatidylinositol-3-phosphate (PI3P), abundant on the outer surface of plant cell and some animal cell plasma membranes. PI3P-mediated effector entry may be widespread in plant, animal, and human pathogenesis. Entry into both plant and animal cells involves lipid raft-mediated endocytosis, while blocking the PI3P binding inhibits effector entry, suggesting new therapeutic avenues (85). On the other hand, the remaining highly diverse C-terminal region of the RXLR effectors is responsible for their function inside the host cells (170). Studies suggest that the genes encoding the RXLR-containing effectors underwent relatively rapid birth and death evolution and that positive selection acted mainly on the C-terminal domain during this evolutionary process (128,180).

(ii) CRN protein family is the second class of *P. infestans* effector proteins that are translocated into the plant cytoplasm. CRN1 and CRN2 were first identified following an *in planta* functional expression screen in which *P. infestans* proteins were synthesized within the plant cytoplasm following introduction of genes by a vector derived from *Potato virus X* (158). Expression of *crn1* and *crn2* in tobacco and tomato led to a leaf-crinkling and necrosis phenotype and induction of defense response genes. Subsequent analysis revealed that the CRNs form a complex family of relatively large proteins (400 to 850 amino acids) in *Phytophthora* (179); however, none of these proteins carries an RXLR motif; instead, they do contain a conserved N-terminal LXLFLAK motif, which clearly shows some similarity to the RXLR sequence (86). *P. infestans crn1* and *crn2* genes are expressed during colonization of tomato host plants, and *crn8* encodes a secreted protein with a predicted kinase domain. It has been proposed that after transport into the host cytoplasm, the CRN effectors manipulate host cellular processes to cause macroscopic phenotypes such as cell death, chlorosis, and tissue browning (158). The CRN deletion mutants identify a C-terminal domain that is sufficient to induce cell death when expressed in the plant cytoplasm (60,67,119). Recent sequencing of the *P. infestans* genome has revealed the existence of 196 CRN effectors (60,128). The large repertoires of predicted cytoplasmic effectors are indicative of their important roles during host infection.

A list of the *P. infestans* effectors whose genes have been cloned and characterized is shown in Table 1 (11,67). Additional information on roles of *P. infestans* cytoplasmic effectors in virulence

through suppression of plant defense response and influence on plant defense signaling pathways can be found in numerous recent reviews related to this topic.

Host Plant Defense

Plants are finely tuned to detect the presence of pathogens on their surface, perceiving both chemical and physical signals of pathogen origin, and react rapidly to the attempted infection (66,69). Possible plant responses to oomycete invasion have been reported, varying from invaded cells with no visible response to more localized reactions preventing formation of pathogen intracellular structures, to HR and programmed cell death, the latter occurring only after pathogen growth had proceeded to the formation of a clearly identifiable haustorium (76,79).

The primary line of induced defense in tomato plants against *P. infestans* is resistance against penetration at the leaf surface; this is a highly effective defense strategy rapidly mobilized by host plants to attempted penetration (69). Recent studies have demonstrated the direct participation of plasma membrane-bound receptor proteins in the recognition of apoplastic pathogen elicitors and activation of host defenses (11,66). Additionally, physical pressure, such as that exerted by the invading penetration peg, may constitute an important signal as part of their surface detection system, which normally leads to reversible formation of microfilaments beneath the pressure point within minutes of the acting stimulus. Both stimuli may trigger the downstream signaling pathways, including salicylic acid (SA)-regulated induction of cell death, ethylene-dependent production of phytoalexin, and reactive oxygen species (ROS)-regulated expression of defense genes and induction of HR (66,139).

Early localized epidermal cell reactions include major rearrangements within the cytoplasm leading to its aggregation beneath the pathogen site, synthesis of ROS, and accumulation of callose beneath the appressoria within 14 h after inoculation or natural pathogen attack (66,69). The rapidly occurring site-directed cytoplasmic streaming is achieved by reorganization of cortical and vascular actin arrays, such that actin microfilaments and cables form a ra-

dial array focused on the infection site. Subsequently, due to the actin-dependent focus of cellular secretory apparatus (endoplasmic reticulum, Golgi bodies, and peroxisomes) beneath the infection site, a wide variety of molecules are delivered to the developing cell wall apposition. Callose, a major component of the cell wall apposition, alongside other cell wall apposition components, such as phytoalexins, phenolics, silicon, H₂O₂, and enzymes like peroxidases and enzyme inhibitors, is likely to inhibit pathogen hyphal penetration. Subsequent defense responses include changes in gene expression and production of phytoalexins and pathogenesis-related (PR) proteins (66,139).

Only after the fall of primary host-plant defenses and successful hyphae penetration and formation of haustorium, the plant invokes the second system of resistance against the pathogen, which involves recognition of specific pathogen molecules and, as a consequence, induction of programmed cell death. HR constitutes a highly effective means of restricting pathogen growth and its containment before transition to the necrotrophic phase occurs (66,171). There are two main groups of molecules that induce HR, elicitors and effectors. Upon their recognition by host plants, elicitors induce HR. Such is the case for the *P. infestans* INF1 elicitor, triggering cell death upon interaction with lectin-like receptor kinase NbLRK1 in *N. benthamiana* (66,90). Thus far, eight *Phytophthora* avirulence effector proteins have been cloned (Table 1). All of them are recognized by cytoplasmic nucleotide-binding site/leucine-rich repeat (NBS-LRR) receptors (11,170). Race-specific and broad-spectrum resistance genes of the host plants induce a similar set of defense reactions in response to corresponding avirulence factors, the effector proteins (66,139).

Genetics and Breeding of Late Blight Resistance

Since the occurrence of the Irish famine in the 1840s, there has been a great deal of interest in developing LB-resistant potato and tomato cultivars. In the course of this process, it has been observed that resistance against *P. infestans* could be classified into race-specific resistance (a.k.a. pathotype-specific or vertical) and race-non-specific resistance (a.k.a. field, horizontal, or partial), with

Table 1. Characterized effectors of *Phytophthora infestans*

Effector (putative; function)	Biological activity	Reference
Apoplastic effectors		
<i>Enzymes degrading cell wall</i>		
Endo-polygalacturonase (PIGP1)	Plant cell wall pectin degradation	159
Endo-glucanases	1,3-β-glucans hydrolysis	110
Exo-glucanases	1,3-β-glucans hydrolysis	110
<i>Inhibitors of enzymes</i>		
Cystatin-like protease inhibitors	Inhibition of plant cystein proteases and potentially suppression of plant defense response	157
Kazal-like extracellular protease inhibitors	Inhibition of plant proteases and potentially suppression of plant defense response	154–156
<i>Pathogen-Associate Molecular Patterns (PAMPs) and small cysteine-rich proteins</i>		
Elicitins (INF1 and others)	Sterol carrier proteins; defense response trigger	80, 90
PcF-like (scr74)	Phytotoxin-like; hypersensitive response (HR) trigger	102
Transglutaminase (Pep13)	Oxidative burst and HR trigger	25, 61
Effectors translocated to cytoplasm		
<i>RXLR type</i>		
AVR1	HR induction upon interaction with R1	161, 170
AVR2	HR induction upon interaction with R2, R2-like, Rpi-abpt, Rpi-blb3	105, 170
AVR3a	HR induction upon interaction with R3a; suppression of INF1-derived HR	5, 17–19
AVR3b	Specific HR induction upon interaction with R3b; no interaction with R3a	101
AVR4	HR induction upon interaction with R4	166, 170
AVRblb1	Class I and II induce HR upon interaction with Rpi-blb1, but class III can suppress Rpi-blb1-mediated HR	27, 170
AVRblb2	HR induction upon interaction with Rpi-blb2	118, 170
AVRvnt1	HR induction upon interaction with Rpi-vnt1	126, 170
<i>Other types</i>		
Crinkling / necrosis inducing proteins	HR suppression / induction	60, 119, 158
SNE1	Suppression of HR induced by various avirulence proteins	91, 99

rices defined by the disease interaction with different host plant genotypes. Developing plants that display disease resistance conferred by major genes (i.e., vertical resistance) are initially effective at preventing infection and protecting the crop from LB. Due to the rapid evolution of pathogen effectors and sexual reproduction of *P. infestans* leading to more aggressive lineages (95,150), however, the vertical resistance against the pathogen could ultimately fail (95,96). In contrast, race-non-specific resistance is often controlled by several genes or qualitative trait loci (QTLs), and potentially could be more durable (23,153). In both potato and tomato, vertical as well as horizontal resistances against LB have been reported, as discussed below. Race-specific major resistance genes, however, have been practically more useful in breeding for resistance against LB, and in fact against most other potato and tomato diseases, in particular when multiple resistance genes are pyramided to increase resistance strength and durability. Thus, it is prudent to identify additional major resistance genes against LB, a task that is underway in many potato and tomato breeding programs around the world (112).

Genetics and Breeding of Late Blight Resistance in Potato

At least 11 race-specific LB resistance genes (*R1* to *R11*) have been identified in potato wild species *S. demissum*, several of which have been mapped (Fig. 3) or cloned (21,106,170) and incorporated into various potato cultivars (163). Currently, most new cultivars of potato carry one or more LB resistance genes and demonstrate some good level of field resistance (21,54). All of these *R* genes confer race-specific, hypersensitive resistance responses. Nevertheless, such race-specific resistance genes may lose their effectiveness as new and more aggressive *P. infestans* strains appear that could overcome the resistance. For example, isolates of *P. infestans* have been identified that overcome all 11 *R* genes in potato (21). Nonetheless, pyramiding the race-specific resistance genes often results in stronger and more durable resistance, as has been observed in many plant species, including potato (35,116) and tomato (52,121) (see below).

From among the 11 major LB resistance (*R*) genes identified in *S. demissum*, four, *R1*, *R2*, *R3a*, and *R3b*, have been cloned (73,170). *R1*, which confers foliar as well as tuber LB resistance, was the first LB resistance gene that was cloned and characterized (10). *R1* has a coiled-coil (CC) domain and putative NBS and LRR domains. Effector-based studies with Avr2 (J. G. Morales, unpublished results) have shown that *R2* and *R2-like*, located on linkage group IV near marker TG339 in a major LB resistance gene cluster (123), are functionally equivalent to *Rpi-blb3* and *Rpi-abpt* (73,170) and share the NBS-LRR structure (170). Using fine mapping and precise disease screening procedures, it was determined that *R3* consisted of two tightly linked genes, *R3a* and *R3b*, which were located on the distal end of chromosome 11, approximately 0.4 cM apart (75). While *R3a* confers foliage resistance, *R3b* confers full foliar and tuber resistance against incompatible *P. infestans* isolates (101). In subsequent studies, it has been determined that *R3a* encodes a CC-NBS-LRR protein and exhibits 82% nucleotide identity with *R3b* located in the same *R3* cluster (101). *R3b*-encoded protein specifically recognizes Avr3b, a newly identified avirulence factor from *P. infestans*. *R3b* does not recognize Avr3a encoded by the corresponding avirulence gene, *Avr3a*, indicating that, despite their high sequence similarity, *R3a* and *R3b* have clearly distinct recognition specificities (101).

At least three resistance genes have been reported in *S. bulbocastanum*, which seem to confer broad-spectrum resistance against many *P. infestans* isolates (165,170). The first gene (*RB*) was mapped to potato chromosome 8 and subsequently cloned and characterized (Fig. 3; 146). The same gene was also mapped and cloned by another group, which named it *Rpi-blb1* (164). Unfortunately, *S. bulbocastanum* cannot be directly crossed with the cultivated potato (*S. tuberosum*), making breeding for this gene tedious and time-consuming. Luckily, more recently, *RB/Rpi-blb1* homologues were discovered in *S. stoloniferum*, a species that can be

crossed with *S. tuberosum*, though with low efficiency, creating the possibility that this resistance could be transferred to the cultivated potato via sexual hybridization (174). Additional *RB/Rpi-blb1* homologues with 83.5% nucleotide identity and conferring broad-spectrum resistance against potato LB were cloned from *S. verrucosum* accession PI275260, a species that also can be crossed with the cultivated potato. Interestingly, this functional ortholog named *RBver* contains an insertion of a complete leucine rich repeat compared to *RB/Rpi-blb1*, and differs from its allele in a susceptible accession at only four amino acid residues (103). Obviously, because *RB/Rpi-blb1* genes have been cloned, they can be transferred to both potato and tomato via genetic transformation (see below).

A second source of LB resistance in *S. bulbocastanum* led to the identification and mapping of *Rpi-blb2* gene on potato chromosome 6 (165). This gene, which has been fine-mapped and cloned, exhibits 82% sequence identity at the amino acid level with the *Mi-1* gene, which is responsible for resistance against root knot nematode, and is mapped to the same region on chromosome 6 (165). A third source of LB resistance in *S. bulbocastanum* led to the identification and fine mapping of *Rpi-blb3* to a 0.93-cM region of potato chromosome 4 (123). In addition to the major LB resistance genes from *S. demissum* and *S. bulbocastanum*, a few other LB resistance genes have been identified in other wild species of potato, including *S. berthaultii* Hawkes (40), *S. mochi-quense* Ochoa (144), *S. phureja* Juz. & Bukasov (141), *S. pinatisectum* Dunal (97), and *S. ruiz-ceballosii* and *S. kurtzianum* (141).

Besides the major LB resistance genes in potato, a number of resistance QTLs, which confer quantitative/field resistance against LB, have been identified in potato. Briefly, the resistance QTLs have been identified based on different types of populations, including (i) intraspecific crosses within *S. tuberosum* (35,100,142), (ii) crosses between cultivated species *S. phureja* and *S. tuberosum* (21,55), (iii) crosses between *S. tuberosum* and various potato wild species, including *S. berthaultii* (40), *S. microdontum* (12), *S. paucisectum* (168), and *S. vernei* (148), and (iv) crosses between cultivated species *S. phureja* and potato wild species *S. stenotomum* (38,140). In many cases, the LB resistance QTLs have been associated with undesirable characteristics, including lateness in maturity, thus reducing their potential utility. Also, as is the case for many other complex disease resistance traits, often QTLs are not consistent across populations and may vary with the species and pathogen isolate used, method of disease evaluation, mapping analysis, and environmental conditions of the experiments. Nonetheless, across the various QTL mapping studies, one major resistance QTL, located on potato chromosome 5 in the vicinity of *R1*, seems to be consistent across several studies, suggesting its potential significance. Unfortunately, this QTL also is associated with lateness in maturity (169), again reducing its potential value. It is currently unknown whether there is a single gene at this location with pleiotropic effects on both LB resistance and plant maturity or if there are tightly linked independent genes affecting the two traits.

Genetics and Breeding of Late Blight Resistance in Tomato

Genetic resistance against tomato LB has been of interest for many years, and three major resistance genes have been identified in the red-fruited tomato wild species *S. pimpinellifolium*. These genes have been mapped to tomato chromosomes 7 (*Ph-1*), 10 (*Ph-2*), and 9 (*Ph-3*) (Fig. 3). *Ph-1* is a single dominant gene providing resistance against race T-0 (16,125), but it has been rapidly overcome by new races of the pathogen (37,49,125). The resistance conditioned by *Ph-2*, a single incomplete-dominant gene mapped to the long arm of chromosome 10 (114), provides only partial resistance against several pathogen isolates (50,125,160). *Ph-2* provides only a reduction in the rate of disease development rather than blocking the disease, and it often fails in the presence of more aggressive isolates (M. R. Foolad, personal observation; 14,150). A much stronger resistance gene, *Ph-3*, was found in *S. pimpinelli-*

Fig. 3. A genetic map of tomato (after http://solgenomics.net/cview/map.pl?map_version_id=55) showing the chromosomal locations of major genes and qualitative trait loci (QTLs) for late blight resistance in potato and tomato. Open horizontal bars denote the approximate location of major *R* genes (*R1*, *R2*, *R2-like*, *R3a*, *R3b*, *R5*, *R6*, *R7*, *R8*, *R9*, *R10*, *R11*: 124) and other identified *R* genes (*Rpi-abpt*, *Rpi-blb3*, *Rpi1*, *RB/Rpi-blb1*, *Rpi-mcq*, *Rber*: 73,124); and the open vertical bars indicate the approximate location of resistance QTLs in potato (*Pi*: 73,124). The solid horizontal bars denote the approximate location of the major *R* genes (*Ph-1*, *Ph-2*, *Ph-3*: 32,84,114,124), and the solid vertical bars indicate the approximate location of resistance QTLs in tomato (*Ribh2*, *Ribh4a*, *Ribh4b*, *Ribh4*, *Ribh7*, *Ribh8*, *Ribh9*, *Ribh12*: 84; *LB-1*, *LB-2*: 46; *lb1a-c*, *lb2a-b*, *lb3*, *lb4*, *lb5a-b*, *lb6a-b*, *lb7a-b*, *lb8a-b*, *lb9a-b*, *lb10*, *lb11a-b*, *lb12a-b*: 23).

folium accession L3708 (a.k.a. LA1269 or PI365957) by L. Black at the Asian Vegetable Research and Development Center in Taiwan (6). This gene confers incomplete-dominant resistance against a rather wide range of *P. infestans* isolates of tomato (29,32,122). To our knowledge, several tomato-breeding programs around the world, including NC State Tomato Breeding Program (R. G. Gardner and D. R. Panthee, *personal communication*), Cornell Tomato Breeding Program (M. Mutschler, *personal communication*), Penn State Tomato Breeding Program (M. R. Foolad, *unpublished data*), AVRDC, the World Vegetable Center (P. Hanson, *personal communication*), have succeeded in transferring this resistance gene to fresh-market or processing tomato breeding lines and commercial cultivars. For example, several fresh-market tomato breeding lines (e.g., NC1 CELBR [*Ph-2* + *Ph-3*] and NC2 CELBR [*Ph-2* + *Ph-3*]) and hybrid cultivars (Plum Regal [*Ph-3*], Mountain Magic [*Ph-2* + *Ph-3*] and Mountain Merit [*Ph-2* + *Ph-3*]), have been released, most recently by the North Carolina State Tomato Breeding Program, USA (52,121) (<http://www.ces.ncsu.edu/fletcher/staff/rgardner/>). Also, more breeding lines and experimental hybrid cultivars are in the pipeline from this and other public tomato breeding programs. Unfortunately, the *Ph-3*-conferred resistance is also race-specific and seems to have been overcome by new pathogen isolates (R. G. Gardner, *personal communication*; 29,43).

The breakdown or reduced effectiveness of the known resistance genes in tomato and the appearance of new and more aggressive isolates of *P. infestans* necessitated the identification, characterization, and utilization of new sources of resistance. Recently, in an intensive search to identify new sources of LB resistance in tomato, a few highly resistant accessions were identified within the tomato wild species *S. pimpinellifolium* (43,44). The identified accessions exhibited resistance against seven different isolates of *P. infestans*, which were of US8, US13, US14, or US15 clonal lineage. Among these, one accession, PSLP153, was chosen for genetic characterization as well as identification and mapping of new LB resistance gene(s). A parent-offspring correlation analysis, using F₂ and F₃ populations of a cross between PSLP153 and a tomato breeding line with susceptibility to LB, indicated a heritability of approximately 0.86 for the LB resistance conferred by PSLP153 (112). A selective genotyping approach led to the identification of two genomic regions on tomato chromosomes 1 (tentatively named *Ph-5-1*) and 10 (tentatively named *Ph-5-2*) conferring resistance against tomato LB (M. R. Foolad, *unpublished data*). Efforts are underway to further delineate the two resistance regions and fine map new resistance genes. Simultaneous breeding efforts to transfer *Ph-5-1* and *Ph-5-2* resistance genes from PSLP153 have led to the development of several new fresh-market and processing tomato lines with improved LB resistance. Furthermore, efforts are underway to develop commercial breeding lines and hybrid cultivars containing these resistance genes in combination with previously known resistance genes, namely *Ph-2* and *Ph-3*. As most useful LB resistance genes thus far identified in tomato are considered race-specific major genes, it is prudent to either pyramid multiple major resistance genes or identify and incorporate useful horizontal resistance (discussed below).

In addition to the above major LB resistance genes in tomato, several QTLs conferring race-non-specific resistance have been reported in different accessions of the green-fruited tomato wild species *S. habrochaites* (8,23,29,84). For example, in accession LA2099, QTLs conferring LB resistance were identified basically on all 12 tomato chromosomes (Fig. 3; 23). Subsequently, with the goal of transferring selected resistance QTLs to the cultivated tomato genetic backgrounds, three nearly isogenic lines (NILs) were developed, each containing one resistance QTL (24). Nevertheless, severe linkage drag prevented the NILs from being useful for breeding purposes. Further inspection of the NILs determined that they also contained genes/QTLs for other characteristics, including plant type, canopy density, maturity, and fruit size and yield in the same introgressed regions. Similar to that in potato (discussed above), QTLs for LB resistance in tomato were generally associated with late maturity and large plant size (23,24).

LB resistance QTLs have also been identified in *S. habrochaites* accession LA1777 (84). In this study, five major QTLs (*Rlbhq4a*, *Rlbhq4b*, *Rlbhq7*, *Rlbhq8*, *Rlbhq12*) were identified on four tomato chromosomes using *S. lycopersicum* × *S. habrochaites* introgression lines. This study also identified a novel QTL for LB resistance in LA2099 (Fig. 3; 84). A different *S. habrochaites* accession with LB resistance that has been used for inheritance and mapping studies is LA1033 (8,29,104,107). In this accession, an inheritance study indicated the presence of at least two LB resistance genes (107), and mapping studies indicated the presence of several QTLs contributing to LB defense response.

A major limitation in the tomato LB resistance breeding research is the lack of clear standards for *P. infestans* race assessment. Researchers have tried to circumvent this difficulty by determining various characteristics of the pathogen isolates, including mating type, fungicide sensitivity, isozyme genotype, and mitochondrial and nuclear DNA (e.g., restriction fragment length polymorphism [RFLP] and simple sequence repeat [SSR]) fingerprints (30,59,143,150). Also, some researchers have tried to characterize/classify *P. infestans* isolates based on their pathogenicity on established resistant lines carrying known LB resistance genes, which to some extent is analogous to the Black's standards in potato (108). Tomato researchers at the AVRDC started analyzing their *P. infestans* isolate collections in this manner in the early 1990s, when *S. pimpinellifolium* accession L3708 was believed to carry a single resistance gene, *Ph-3*, and *S. habrochaites* accession LA1033 was designated as the source of *Ph-4* resistance gene (7). Follow-up investigations revealed complex resistance involving multi-genes in L3708 (92,93,175) or multi-QTLs in LA1033 (107). Classifications such as these constituted a mere first step toward development of a more comprehensive and useful classification system of the races of tomato-virulent *P. infestans* strains. It is expected, however, that with the availability of *P. infestans* genomic sequence data and identification of additional sources of LB resistance in tomato, more information regarding the various races of the pathogen will soon become available.

Speeding Up the Race: Transgenic Approaches to Breeding for Late Blight Resistance

Two novel approaches to developing potatoes and tomatoes with LB resistance have been considered, pyramiding major resistance genes via traditional breeding or marker-assisted selection (MAS), and genetic transformation. Resistance gene pyramiding has been practiced for various diseases in several crop species, including powdery mildew resistance in wheat (176), LB resistance in potato (54,151), and combining resistance against different diseases and insects in tomato (43,133,138). It is expected that, similar to potato, pyramiding the existing as well as new sources of major LB resistance genes in tomato will provide a sustainable control of LB in tomato. Pyramiding of the resistance genes using conventional breeding approaches, however, may not always be possible, or it may be laborious and time-consuming. An alternative approach to introducing single or multiple LB resistance genes is genetic transformation (42,77). This approach, however, requires prior knowledge of the gene(s), including identification, cloning, and characterization.

The utility of the transgenic approach to developing plants with resistance against LB has been substantiated by demonstrating improvement in resistance via increasing copy number of the transgene(s). In general, the *RB* gene, like many other disease resistance genes, is constitutively transcribed at low levels. In transgenic potatoes carrying the *RB* gene, disease resistance was enhanced as copy numbers and transcript levels increased (20). It seems that the resistance-gene transcript may have to surpass a threshold level before triggering innate plant RNA silencing (20). Two years of field trials demonstrated that transgenic lines of cultivated potato carrying the *RB* transgene(s) did not exhibit any significant reduction in tuber size or yield compared to nontransgenic plants (62). The same *RB*-containing transgenic plants, despite their foliar resistance, did not exhibit any increased resistance against LB in tubers (62).

A recent attempt with the transgenic approach using potato *R1* gene heterologously expressed in tomato indicated the activation of the transgene in the *R1* incompatible reaction during *P. infestans* attack and its expression at a very early stage of infection (41). Similarly, upon heterologous expression of the coding sequence of the *R3a* gene from the wild potato species *S. demissum* in the cultivated potato, the transgenic plants challenged with *P. infestans* displayed strong resistance response (182). Based on these results, the authors postulated in their patent application that the *R*-gene could be used as a plant selectable marker, upon a compatible interaction between pathogens and host plants (182).

A similar transgenic approach, employing novel LB *R* genes from the potato wild species *S. okadae* (*Rpi-oka1*) and *S. mochi-quense* (*Rpi-mcq1.1*, *Rpi-cmq1.2*), was used to enhance LB resistance in potato and tomato cultivars (81). In this study, eight out of nine recovered transgenic potato plants of the susceptible cultivar Desiree exhibited resistance against the tested *P. infestans* isolates. Furthermore, when cloned under their native promoter and terminator sequences and stably transformed into potato or tomato cultivars, expression of all three genes induced resistance against a wide spectrum of tested *P. infestans* isolates. Interestingly, the spectrum of LB resistance exhibited for each construct was in agreement with that exhibited by the original wild potato accessions harboring these genes. In particular, *Rpi-oka1*-derived LB resistance was retained in the transgenic plants, and the resistance phenotype was not due to constitutive activation of defense pathways by the transgene (81). Similarly, LB susceptible potato and tomato cultivars exhibited resistance when transformed with resistance gene *Rpi-vnt1.1* from *S. venturii* (45). In this study, the transgenic potato and tomato plants exhibited resistance against 10 of the 11 isolates tested; only the isolate EC1 from Ecuador was able to overcome *Rpi-vnt1.1* and cause disease on the inoculated plants (45).

Conclusions and Future Prospects

P. infestans is one of the most devastating plant pathogens of all time. Under the right conditions, it is able to spread at an alarming pace, threatening crop production on a vast scale. Recently, research on the interactions between host plants and *P. infestans* has contributed a wealth of knowledge to our understanding of the cellular and molecular biology of the pathogen infection strategies and key components of these interactions. Immunocytochemistry studies and GFP-tagging of plant proteins have provided detailed cell-level knowledge of the dynamic changes during initiation of infection, including the production, structure, and behavior of the *P. infestans* spores. Studies of oomycete effectors increase our understanding of how such agents inhibit plant defense and enhance pathogen virulence. Sequencing the *P. infestans* genome has opened a new chapter in the pathogenicity studies, enabling a high-throughput classification and analyses of the putative effector proteins, leading to the discovery of previously overlooked genes involved in virulence.

Simultaneously, considerable efforts have been made in both potato and tomato to identify genetic sources of LB resistance, map and clone resistance genes or QTLs, and transfer resistance to modern breeding lines and cultivars. Although transfer of race-specific major resistance genes in potato has significantly contributed to a better control of LB and considerable reduction in fungicide application, such resistance is often not durable due to high temporal and spatial variation in *P. infestans* populations. Fortunately, pyramiding the major LB resistance genes in potato has proven to enhance the utility of the genetic approach to control potato LB. Furthermore, the identification and characterization of major LB resistance genes in *S. bulbocastanum* that significantly reduce disease progression when potato plants are exposed to different isolates of *P. infestans* is very promising. Moreover, efforts in identifying QTLs conferring field resistance against potato LB have been rewarding, and several major resistance QTLs with potential utility in breeding programs have been identified. A noticeable drawback here, however, has been an undesirable asso-

ciation of field resistance with plant physiological maturity. Despite this disappointment, a few LB resistance QTLs have been identified which seem to be independent of plant maturity, and thus may be useful in developing early to midseason potato cultivars with sufficient LB resistance. Overall, it is expected that the knowledge of different LB resistance genes and QTLs in potato will translate into development of new cultivars with improved and durable resistance and a reduction in the use of fungicides for disease control.

Comparatively, much less work has been done on the genetics, genomics, and breeding of LB resistance in tomato. This has been in part due to a greater significance of LB in potato than in tomato, at least before the 1990s, when many of the potato isolates were not pathogenic to tomato. Nevertheless, due to the recent increased significance of tomato LB, currently more efforts are being devoted to the identification, characterization, and introduction of resistance genes into new tomato breeding lines and hybrid cultivars. In addition, the ongoing advances in sequencing the tomato genome will undoubtedly contribute to further characterization of the host plant resistance in tomato. It is expected that in tomato more resistance genes or QTLs will be identified and characterized, and future commercial releases will have genes and/or QTLs for LB resistance. Also, a greater effort will be placed on developing tomato breeding lines and hybrid cultivars with multiple LB resistance genes and QTLs. Pyramiding of the existing as well as new sources of resistance genes or QTLs in tomato will provide a more sustainable control of tomato LB, similar to the pyramiding of LB resistance genes in potato.

As more resistance genes and QTLs are identified and characterized in potato and tomato, and with the recent advances in molecular biology and genome sequencing of both the host and the pathogen, commercial availability of transgenic potato and tomato cultivars with LB resistance will eventually be a reality. Recent attempts with transgenic approach using the potato LB resistance genes heterologously expressed in potato and tomato have been promising. In particular, the initial attempts in transferring the broad-spectrum major resistance gene(s) from *S. bulbocastanum* have been successful and very promising.

Future research should not only seek employment of particular genes conferring LB resistance, but also patterns of defense genes activated, and thus molecular and cellular defenses raised, during attacks of compatible/incompatible races of *P. infestans*. Studies aiming at discovery of the pathogen signal molecules and effectors have entered into a new era of high-throughput research, supported by the genetic information regarding *Phytophthora*. Employment of genetic engineering into LB resistance breeding should speed up in the arms races against *P. infestans*, the *R* gene destroyer (47).

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