Physical Education, Health and Social Sciences

https://journal-of-social-education.org E-ISSN: 2958-5996

P-ISSN: 2958-5988

Late Blight of Potato: Lessons from Phytophthora infestans on Pathogen Evolution and Resistance Breakdown

Aqib Shafiq[1], Sayed Muhammad Ukkashah Iftikhar[2], Abdullah Bin Talat[3], Nabeel Ahmad*[4], Muhammad Haroon[5], Ameer Jan[6]

[1] Department: Plant Pathology, University of Agriculture Faisalabad. Islamic Chamber of Commerce and Development. aqibuaf.edu.pk@gmail.com aqibshahan@gmail.com

[2] Department of Plant Pathology, University of Agriculture Faisalabad. ukkashahiftikhar18@gmail.com

[3] Department of Plant Pathology University of Agriculture Faisalabad. mianabdullahbintalat@gmail.com

[4] National Institute for Biotechnology and Genetic Engineering (NIBGE). *Corresponding Author nabeel8700@gmail.com

[5] Texas Tech university. muhharoo@ttu.edu

[6] Department of Botany University of Makran Panjgur. Ameerjan@uomp.edu.pk

DOI: https://doi.org/10.63163/jpehss.v4i1.1228

Abstract

Late blight, caused by the oomycete pathogen Phytophthora infestans, remains one of the most destructive diseases affecting potato and tomato production worldwide, inflicting annual economic losses estimated at $6.7–12 billion and driving extensive fungicide use. This review explores the evolutionary success of P. infestans through its historical impact including the Irish Potato Famine (1845–1852) and modern biological mechanisms. Key features include the pathogen's hemibiotrophic lifestyle, rapid asexual and sexual reproduction, and a distinctive "two-speed" genome architecture characterized by conserved gene-dense regions and highly plastic, repeat-rich gene-sparse regions enriched in transposable elements and effector genes. The extensive RXLR effector superfamily enables sophisticated suppression of host immunity via mechanisms such as hypersensitive response inhibition, vesicle trafficking disruption, and autophagy hijacking. Plant resistance relies on nucleotide-binding leucine-rich repeat (NLR) proteins, including sensor NLRs networked with NRC helpers, but resistance genes frequently break down due to effector mutations, silencing, or deletions. Emerging clonal lineages like EU_41_A2 highlight ongoing adaptation, while fungicide resistance and climate change further complicate management. Biotechnological advances, including cisgenic R-gene stacking and CRISPR/Cas9-mediated editing of susceptibility (S) genes (StDMR6-1), offer promising paths toward durable resistance. Integrated pest management combining sanitation, decision support systems, and durable host resistance is emphasized for sustainable control.

Keywords: Phytophthora Infestans, Late Blight, Potato, Two-Speed Genome, RXLR Effectors, Effector-Triggered Immunity, Resistance Breakdown, NLR Proteins, CRISPR/Cas9, Integrated Pest Management

1. Introduction

The devastating plant disease known as potato late blight remains the most significant threat to global potato and tomato production, characterized by its historical role in human catastrophe and its modern status as a primary driver of agricultural pesticide use (Chakrabarti et al., 2022). Caused by the hemibiotrophic oomycete *Phytophthora infestans*, the disease is defined by its rapid onset and its nearly unparalleled capacity to overcome host resistance and chemical controls (Haas et al., 2009). The pathogen's ability to evolve new virulence and functional traits highlights a sophisticated evolutionary-ecological framework that challenges the sustainability of current agricultural systems (Nowicki et al., 2012). Understanding the molecular interplay between *P. infestans* and its Solanaceous hosts requires a deep exploration of the pathogen's unique "two-speed" genome, the diverse repertoire of effector proteins it secretes, and the complex intracellular immune networks that plants deploy in defense (Wu et al., 2017).

2. Historical and Socio-Economic Trajectories of Late Blight

The history of *P. infestans* is inextricably linked to the Irish Potato Famine of 1845–1852, an event that serves as a foundational case study in plant pathology and humanitarian disaster (Shakour, 2020). While the pathogen likely originated in the Andean region of South America, with Mexico serving as a secondary center of diversity, its introduction to the United States occurred in 1843, appearing first in Philadelphia and New York City (Lona, 2021). From there, the pathogen crossed the Atlantic in 1845, likely via infested seed potatoes shipped to Belgium, subsequently spreading across Europe with catastrophic speed (Cottyn et al., 2023).

In Ireland, the socio-economic conditions of the mid-nineteenth century created a uniquely vulnerable society. The Irish population was the most rapidly growing in Europe in the early 1840s, and at least two-thirds of the population was entirely dependent on the potato as their primary source of food (Haverkort et al., 2009). This dependence was compounded by the use of a single potato variety, the 'Lumper,' which lacked genetic diversity and was highly susceptible to the blight (Afzal, 2021). When the disease struck, it triggered a near-complete destruction of the harvest, resulting in at least one million deaths and the displacement of another million people who emigrated to North America and elsewhere (Angmo et al., 2023).

Historical analysis reveals that the severity of the Irish Famine was not merely a biological phenomenon but a consequence of political and economic structures. Despite the total failure of the potato crop, Ireland continued to export massive quantities of grain, livestock, and dairy products to England (Blum et al., 2026). In 1847, often called "Black '47," while hundreds of thousands were dying of starvation, nearly 4,000 vessels carried food from Irish ports to British cities like Bristol, Liverpool, and London (Ziegler et al., 2023). This tension between "money crops" for export and "food crops" for local subsistence illustrates a critical lesson in global food security: the presence of food does not guarantee access if the socio-political will to redistribute resources is absent (Chege, 2025).

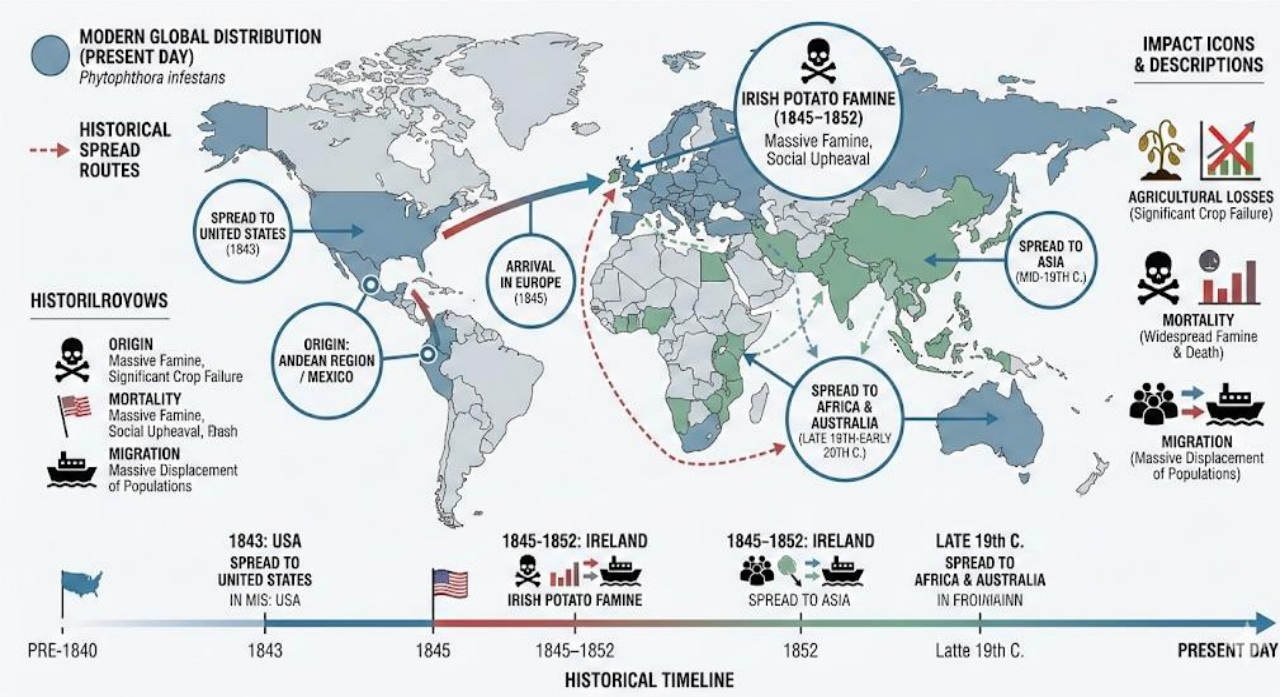
Table 1: Historical and Economic Impact Metrics of Late Blight

Metric	Estimated Magnitude	Reference
Global Annual Economic Losses	$6.7 - $12 Billion USD	(Nowicki et al., 2012; Haas et al., 2009)
Mortality During Irish Famine (1845-1852)	approx. 1,000,000 People	(Haas et al., 2009; Haverkort et al., 2009)
Emigration Following the Famine	approx. 1,500,000 - 2,000,000 People	(Haverkort et al., 2009; McGrath, 2010)
Irish Population Reduction (1845-1850)	approx. 33%	(Powderly, 2019)

U.S. Potato Yield Losses (Recent Decades)	approx. 3.5%	(Nowicki et al., 2012)

In modern contexts, *P. infestans* continues to exert a heavy economic burden. Conservative estimates place global annual losses at $6.7 billion, though more recent evaluations suggest the figure may reach $12 billion when including the costs of fungicide application and post-harvest spoilage (Lamichhane et al., 2024). In developing countries, where effective chemical control is often cost-prohibitive, late blight frequently results in yield losses exceeding 60% (Chakrabarti et al., 2022). Furthermore, the disease's social impact remains high, as evidenced by surges in internet searches for "blight" during regional epidemics, such as those occurring in the United States and Europe in 2005, 2007, and 2009 (Wang et al., 2025). The global importance of late blight is deeply rooted in its historical spread and devastating socio-economic consequences. The geographic dissemination of *Phytophthora infestans* and its historical milestones are illustrated in figure 1.

FIGURE 1. GLOBAL IMPACT AND HISTORICAL SPREAD OF POTATO LATE BLIGHT



3. Biological Architecture and Pathogenesis

Phytophthora infestans belongs to the Oomycota, a group of filamentous organisms that are morphologically similar to true fungi but phylogenetically related to heterokont algae, such as brown algae and diatoms (Chaithra et al., 2025). This evolutionary distinction is reflected in several fundamental biological differences, including cell walls composed of beta-1,3 and beta-1,6 glucans and cellulose rather than chitin, and a diploid rather than haploid or dikaryotic vegetative state (Manoharachary, 2019). The pathogen is a near-obligate hemibiotroph, meaning its infection process begins with a biotrophic phase that maintains host cell viability followed by a destructive necrotrophic phase (Al-Abedi, et al., 2025).

The asexual life cycle of *P. infestans* is designed for rapid population expansion. It produces sporangia on specialized structures called sporangiophores that emerge through host stomata or necrotic tissue. These sporangia are readily dehiscent and can be aerially dispersed over several kilometers (Sundar, 2025). Upon landing on a susceptible host in the presence of free water or high humidity, the sporangia follow one of two developmental paths depending on the ambient

temperature. In warmer conditions (20–25 degrees C), the sporangia germinate directly via a germ tube to penetrate plant tissues (Brus-Szkalej, 2019). In cooler conditions (10–15 degrees C), sporangia undergo zoosporogenesis, releasing up to eight wall-less, biflagellated zoospores (Subhani, 2016).

The sexual cycle requires the presence of two mating types, designated A1 and A2. When these mating types encounter each other, they produce oospores, which are thick-walled, hardy survival structures (Singh, 2024). Oospores can persist in the soil for several years without a host, serving as a primary source of inoculum that can initiate epidemics earlier in the season than sporangia originating from infected tubers (Suffert et al., 2021). The global spread of the A2 mating type, which was historically confined to Mexico until the late twentieth century, has introduced sexual recombination into populations that were previously clonal, leading to increased genetic diversity and the emergence of more aggressive lineages (Mahaffee et al., 2023).

3.1 Cellular Mechanics of Infection

The transition from a motile zoospore to an intracellular pathogen involves a precise sequence of events. Encystment is triggered by contact with the host surface, during which dorsal and ventral vesicles are secreted to adhere the cyst to the plant (Flieger et al., 2018). Germination occurs within 20 to 30 minutes, producing a germ tube that typically forms an appressorium (Nowicki et al., 2012). Once inside the host, *P. infestans* develops specialized infection structures called haustoria. These structures invaginate the host plasma membrane but do not penetrate it, creating a large surface area for the secretion of effector proteins and the uptake of host nutrients (Amponsah et al., 2021).

4. The Two-Speed Genome: A Framework for Adaptation

The most striking feature of the *P. infestans* genome is its extreme plasticity, which has led to the coining of the "two-speed genome" concept. The genome, which is unusually large at approximately 240 Mbp, is divided into two distinct compartments based on gene density and repetitive content (Knaus et al., 2020).

The core genomic compartment consists of gene-dense regions (GDRs) that harbor essential housekeeping genes. These regions are characterized by low levels of repetitive DNA and are subject to purifying selection, resulting in high levels of conservation and stability (Sánchez-Vallet et al., 2018). In contrast, the second compartment consists of gene-sparse regions (GSRs) that are highly enriched in transposable elements (TEs) and repetitive DNA (Chen et al., 2022). The GSRs evolve at a much faster rate than the GDRs, driven by positive selection and the destabilizing influence of TEs (Rech et al., 2019).

Table 2: Genomic Compartmentalization in Phytophthora infestans

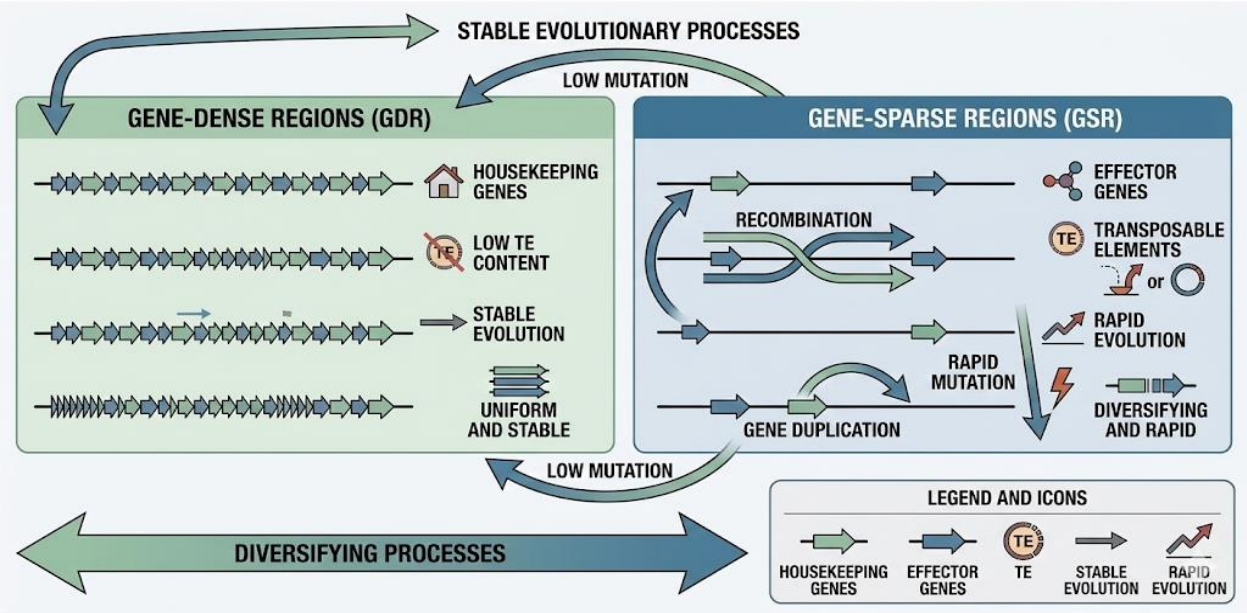
Genomic Region	Repeat Density	Evolutionary Pressure	Primary Gene Functions
Gene-Dense (GDR)	Low (less than 25%)	Purifying Selection	Housekeeping, Basic Metabolism
Gene-Sparse (GSR)	High (more than 75%)	Positive Selection	Pathogenicity, Effectors, Virulence

4.1 Role of Transposable Elements in Diversification

Transposable elements make up nearly 74% of the *P. infestans* genome. These elements actively and passively contribute to the evolution of virulence traits. Actively, TEs can cause gene duplications or deletions when they move within the genome (Mat Razali et al., 2019). Passively,

the high concentration of repetitive sequences facilitates erroneous double-strand break repair and non-homologous recombination, leading to chromosomal rearrangements and gene family expansions (Ranjha et al., 2018). One of the defining genomic features of *P. infestans* is its compartmentalized genome organization known as the two-speed genome. This genomic architecture is illustrated in figure 2.

FIGURE 2: THE TWO-SPEED GENOME ARCHITECTURE OF *PHYTOPHTHORA INFESTANS*



5. Molecular Weaponry: Effector Biology and Virulence

The pathogenicity of *P. infestans* is mediated by an extensive repertoire of secreted proteins, termed effectors, which are categorized by their site of action. Apoplastic effectors are secreted into the host extracellular matrix and include protease inhibitors, lectins, and cell-wall degrading enzymes designed to neutralize antimicrobial compounds and facilitate tissue invasion (Pinto et al., 2025). Cytoplasmic effectors, however, are translocated into the host cell and function as the primary manipulators of plant immunity (Bozkurt et al., 2015).

5.1 The RXLR Effector Superfamily

The most prominent class of cytoplasmic effectors is defined by the RXLR motif (Arginine-any amino acid-Leucine-Arginine), which is situated at the N-terminus of the protein and is required for translocation into the host cytoplasm (Chepsergon et al., 2022). *P. infestans* is predicted to encode approximately 550 to 563 RXLR effectors. These effectors employ a multi-pronged strategy to disrupt plant immunity:

1. **Immunosuppression:** Effectors like *AVR3a* target essential host defense proteins. Specifically, *AVR3a* binds to and stabilizes the host E3 ligase *CMPG1*, preventing its degradation and thereby suppressing defense-triggered cell death (Du et al., 2018).
2. **Vesicle Trafficking Interference:** Some effectors, such as *AVR1* and *AVR3*, interfere with the plant's vesicle trafficking systems, blocking the delivery of defense-related compounds (Akram 2023).
3. **Autophagy Hijacking:** The effector *PexRD54* competitively binds to host *ATG8* proteins, repurposing the cellular clearance system for the benefit of the pathogen (Bozkurt et al., 2015).

Table 3: Functional Roles of Key RXLR Effectors in Immunity Suppression

Representative Effector	Functional Target	Impact on Host Immunity	Reference
AVR3a	Host E3 ligase CMPG1	Suppresses HR and stabilizes host cells	(Bozkurt et al., 2015)
AVR1	Callose/Vesicle Trafficking	Suppresses basal defense and cell death	(Nowicki et al., 2012; Bozkurt et al., 2015)
PexRD54	Host ATG8 Protein	Hijacks autophagy for nutrient redirection	(Nowicki et al., 2012; Bozkurt et al., 2015)
SFI Effectors	PTI Signaling Components	Neutralizes early immune recognition	(Bozkurt et al., 2015; Bozkurt et al., 2017)
IPI-O	Rpi-blb1 Receptor	Recognized by Rpi-blb1 but can be modified	(Haas et al., 2009; Van der Vossen et al., 2005)

6. Host Immunity: The NLR Recognition Landscape

The Solanaceous host plants respond to *P. infestans* through a two-tiered innate immune system. Pattern-Triggered Immunity (PTI) is initiated by cell-surface receptors that detect conserved microbial molecules (Yu et al., 2021). To overcome PTI, the pathogen secretes effectors, which are monitored by intracellular receptors known as Nucleotide-binding Leucine-rich Repeat (NLR) proteins, leading to Effector-Triggered Immunity (ETI) (Song et al., 2024).

6.1 Recognition Models: Guard, Decoy, and Integrated Decoys

NLRs recognize effectors through three primary evolutionary models:

- **Guard Model:** The NLR "guards" a functional host protein (the guardee) that is the target of a pathogen effector.
- **Decoy Model:** The plant evolves a non-functional mimic of a guardee, known as a decoy, solely to trap the effector.
- **Integrated Decoy Model:** NLR proteins contain additional integrated domains (ID) that mediate direct recognition by mimicking protein targets (Wu et al., 2017).

6.2 The NRC Helper NLR Network

In Solanaceous plants, a complex genetic network of "helper" NLRs, termed the NRC family, acts as a signaling hub for multiple "sensor" NLRs. Sensor NLRs like *Rpi-blb2* and *R1* require specific helpers like *NRC4* (DeFalco et al., 2021).

Table 4: NRC Network Pairings and Resistance Components

Resistance Protein (Sensor)	Source Species	Cognate Effector (Avr)	Helper NLR Required
R1	Solanum demissum	AVR1	NRC4
Rpi-blb2	Solanum bulbocastanum	AVRblb2	NRC4
Prf / Pto	Solanum lycopersicum	AvrPto / AvrPtoB	NRC2 / NRC3
Mi-1.2	Solanum lycopersicum	(Nematode Effectors)	NRC4
Rpi-vnt1	Solanum venturii	AVRvnt1	(NRC Network)

7. Dynamics of Resistance Breakdown

The fundamental challenge in managing late blight is the rapid breakdown of host resistance genes. Historically, *R* genes introgressed from wild species (such as *R1* to *R11*) were widely deployed in the 20th century (Berindean et al., 2024). However, *P. infestans* populations possess a high rate of diversity at effector loci, allowing them to quickly select for "resistance-breaking" biotypes (Angmo et al., 2023).

7.1 Molecular Mechanisms of Evasion

Resistance breakdown occurs through several distinct genetic modifications:

1. **Amino Acid Substitution:** Minor genetic shifts can prevent recognition. For example, the difference between avirulent and virulent alleles of *Avr3a* is only three amino acids (Nowicki et al., 2012).
2. **Gene Silencing:** Strains can overcome resistance by downregulating or stopping the expression of an *Avr* gene (Manoharachary, 2019).
3. **Deletion:** GSRs facilitate the physical loss of effector genes through unequal recombination (Haas et al., 2009).

8. Contemporary Population Shifts: The Case of EU_41_A2

The evolution of *P. infestans* is vividly illustrated by the recent emergence and expansion of the *EU_41_A2* clonal lineage in Northern Europe. Since its first detection in Denmark in 2013, *EU_41_A2* has expanded its distribution to Sweden, Norway, Poland, Germany, and Finland (Corbière et al., 2019). This is the first documented case of a clonal lineage persistently expanding within a sexually recombining population (Pereyra et al., 2023).

9. Chemical Management and Evolutionary Resilience

Fungicides remain a crucial component of integrated disease management (IPM). However, the pathogen's ability to develop resistance to these chemicals mirrors its ability to overcome host genes (Suffert et al., 2021).

9.1 Fungicide Resistance and Reversibility

While single-site fungicides are highly prone to resistance, multi-site fungicides like mancozeb have traditionally been considered low-risk. However, experimental studies have demonstrated that *P. infestans* can develop resistance to mancozeb after approximately 200 days of continuous acclimation. Interestingly, this resistance appears to be reversible when the selective pressure is removed (Knaus et al., 2020).

9.2 The Impact of Global Warming

Climate change significantly drives the genetic variation of fungicide resistance. Research on the cytochrome b (*Cyt-b*) gene indicates that temperature is a key driver of genetic diversity. Warmer climates may enhance mutational diversity, pre-adapting pathogens to evade chemical controls while also generating genetic load that constrains fitness (Ranjha et al., 2018).

10. Biotechnological Frontiers: Stacking and S-Gene Editing

The limitations of traditional breeding have spurred the development of advanced strategies, including cisgenic *R* gene stacking and *S* gene knockout using CRISPR/Cas9 (Kieu et al., 2021).

10.1 Cisgenics and R-Gene Pyramiding

Cisgenesis involves transferring genes between crossable species without using foreign DNA. By "stacking" multiple *R* genes from wild relatives like *S. bulbocastanum*, breeders create varieties with more durable resistance. Trials in Bangladesh during 2023-24 showed that potatoes carrying a 3R-gene stack exhibited complete resistance in sprayed and non-sprayed plots (Ghislain et al., 2019).

10.2 S-Gene Modification via CRISPR/Cas9

Modifying Susceptibility (*S*) genes host genes the pathogen hijacks for infection can provide broad-spectrum resistance. The *StDMR6-1* gene is a primary target. CRISPR/Cas9-mediated knockouts of *StDMR6-1* have shown significantly increased resistance over four years of field trials without yield penalties or tuber quality trade-offs (Van der Vossen et al., 2005).

Table 5: CRISPR/Cas9 S-Gene Modifications and Physiological Outcomes

S-Gene Target	CRISPR Modification Type	Resistance Effect	Physiological Trade-offs	Reference
StDMR6-1	Tetra-allelic Deletion	High LB Resistance, Early Blight, Common Scab	None reported in 4-year trial	(Karlsson et al., 2024)
StSR4	RNP-mediated Knockout	Enhanced LB Resistance	Dwarf phenotype, growth inhibition	(Sun et al., 2022)
StDND1	Functional Knockout	Enhanced LB Resistance	Auto-necrotic spots, occasional dwarfing	(Kieu et al., 2021)
StCHL1	Functional Knockout	Enhanced LB Resistance	Minimal trade-offs reported	(Kieu et al., 2021)
StMLO1	CRISPR Mutagenesis	Potential Resistance	Still under assessment	(Kieu et al., 2021)

11. Integrated Management and Decision Support

Sustainable control requires an Integrated Pest Management (IPM) framework:

- **Sanitation:** Eliminating inoculum sources by removing cull piles (
- **Host Resistance:** Utilizing stacked *R* genes and *S* gene
- **Decision Support Systems (DSS):** Using platforms like Simphyt and EuroBlight to optimize fungicide timing based on weather models (Haverkort et al., 2009).

12. Conclusions

Phytophthora infestans exemplifies a highly adaptive plant pathogen whose evolutionary toolkit encompassing a compartmentalized "two-speed" genome, massive effector repertoires, rapid reproduction, and sexual recombination continues to outpace conventional control strategies nearly two centuries after triggering the Irish Potato Famine. The recurrent breakdown of major R genes, emergence of aggressive clonal lineages such as EU_41_A2, and developing resistance to fungicides (including multi-site compounds under prolonged selection) underscore the limitations of single-tactic approaches in the face of ongoing pathogen evolution, exacerbated by global warming and agricultural intensification. Nevertheless, recent biotechnological innovations, particularly the pyramiding of multiple wild-derived R genes via cisgenesis and precise CRISPR/Cas9 editing of host susceptibility factors like StDMR6-1, demonstrate substantial potential for engineering broad-spectrum, durable resistance with minimal fitness costs.

Sustainable management of late blight demands a holistic integrated pest management framework that integrates these advanced genetic tools with cultural practices, precision fungicide application guided by forecasting models (EuroBlight, Simphyt), and inoculum reduction strategies. By addressing both the molecular arms race between pathogen and host and the socio-economic drivers of disease vulnerability, it is possible to mitigate the persistent threat of late blight and enhance global food security in potato-dependent regions.

References

Bozkurt, T. O., Belhaj, K., Maqbool, A., Chaparro-Garcia, A., Pandey, P., Petre, B., Tabassum, N., Cruz-Mireles, N., Hughes, R. K., Sklenar, J., Win, J., Menke, F., Findlay, K., Banfield, M. J., & Kamoun, S. (2015). An effector of the Irish potato famine pathogen antagonizes a host autophagy cargo receptor. *eLife*, *4*, e10856. https://doi.org/10.7554/eLife.10856

Corbière, R., Hansen, J. G., Runno-Paurson, E., Nielsen, B. J., Cooke, D. E. L., & EuroBlight Consortium. (2019). Examining phenotypic traits contributing to the spread in northern European potato crops of EU_41_A2, a new clonal lineage of Phytophthora infestans. *Phytopathology*, *109*(1), 10–10.

Ghislain, M., Rivera, C., Roman, M. L., Tovar, J., Gamboa, S., Forbes, G. A., & Kreuze, J. F. (2019). Stacking three late blight resistance genes from wild species directly into African highland potato varieties confers complete field resistance to local blight races. *Scientific Reports*, *9*(1), 4242. https://doi.org/10.1038/s41598-019-40837-2

Haas, B. J., Kamoun, S., Zody, M. C., Jiang, R. H., Handsaker, R. E., Cano, L. M., Grabherr, M., Kodira, C. D., Rafidei, S., Mauceli, E., Bozkurt, T. O., Akimoto, M. A., Huitema, E., Saunders, D. G., Tatnell, J., Studholme, D. J., Terry, S. J., Thines, M., Rougon-Cardoso, A.,... Nusbaum, C. (2009). Genome sequence and analysis of the Irish potato famine pathogen Phytophthora infestans. *Nature*, *461*(7262), 393–398. https://doi.org/10.1038/nature08358

Haverkort, A. J., Boonekamp, P. M., Hutten, R., Jacobsen, E., Lotz, L. A., Kessel, G. J., Visser, R. G., & van der Vossen, E. A. (2009). Societal costs of late blight in potato and prospects of durable resistance through cisgenic modification. *Potato Research*, *52*(3), 249–257. https://doi.org/10.1007/s11540-009-9137-x

Kieu, N. P., Lenman, M., Wang, E. S., Petersen, B. L., & Andreasson, E. (2021). Mutations introduced in susceptibility genes through CRISPR/Cas9 genome editing confer increased late blight resistance in potatoes. *Scientific Reports*, *11*(1), 4487. https://doi.org/10.1038/s41598-021-83972-w

Nowicki, M., Foolad, M. R., Nowakowska, M., & Kozik, E. U. (2012). Potato and tomato late blight caused by Phytophthora infestans: An overview of pathology and resistance breeding. *Plant Disease*, *96*(1), 4–17. https://doi.org/10.1094/PDIS-05-11-0458

Sun, K. B., Park, S. J., Park, J. S., Lee, H. J., Shin, S. Y., Lee, S. M., Choi, G. J., Kim, S. G., Cho, H. S., Jeon, J. H., Kim, Y. S., Park, Y. I., & Kim, H. S. (2022). Editing of StSR4 by Cas9-RNPs confers resistance to Phytophthora infestans in potato. *Frontiers in Plant Science*, *13*, 997888. https://doi.org/10.3389/fpls.2022.997888

Van der Vossen, E. A. G., Gros, J., Sikkema, A., Muskens, M., Wouters, D., Wolters, P., Pereira, A., & Allefs, S. (2005). The Rpi-blb2 gene from Solanum bulbocastanum is an Mi-1 gene homolog conferring broad-spectrum late blight resistance in potato. *The Plant Journal*, *44*(2), 208–222. https://doi.org/10.1111/j.1365-313X.2005.02527.x

Wu, C. H., Abd-El-Haliem, A., Bozkurt, T. O., Belhaj, K., Terauchi, Japan R., Vleeshouwers, V. G., & Kamoun, S. (2017). NLR network mediates immunity to diverse plant pathogens.

Proceedings of the National Academy of Sciences, *114*(30), 8113–8118. https://doi.org/10.1073/pnas.1702041114

Chakrabarti, S. K., Sharma, S., & Shah, M. A. (2022). Potato pests and diseases: a global perspective. In Sustainable management of potato pests and diseases (pp. 1-23). Singapore: Springer Singapore.

Shakour, K. E. (2020). From colonial legacy to difficult heritage: responding to and remembering An Gorta Mór, Ireland's great hunger. University of South Florida.

Lona, A. (2021). Epidemic Louse-Borne Typhus, and biological and socio-economic factors: a review of disease events during the Irish potato famine and recent history (Doctoral dissertation, Kansas State University).

Cottyn, H., Beeckaert, E., & Bruneel, D. (2023). The Voyages of a Potato Companion: Phytophthora Infestans. Anthropocenes–Human, Inhuman, Posthuman, 4(1).

Afzal, A. (2021). Potato variety improvement through marker assisted selection (Doctoral dissertation, Brac University).

Angmo, D., Sharma, S. P., & Kalia, A. (2023). Breeding strategies for late blight resistance in potato crop: recent developments. Molecular biology reports, 50(9), 7879-7891.

Blum, M., Colvin, C. L., & McLaughlin, E. (2026). Scarring and selection in the Great Irish Famine. The Economic History Review, 79(1), 189-220.

Ziegler, L. (2023). The Third Horseman: Preventability Versus Apocalypse in the Great Famine of 1315 and the Irish Potato Famine.

Chege, M. (2025). Farming From Afar: Translocal Farmers and Agrarian Change in Kenya (Doctoral dissertation, Clark University).

Lamichhane, S., Neupane, S., Timsina, S., Chapagain, B., Paudel, P. P., & Rimal, A. (2024). Potato Late Blight Caused by Phytophthora Infestans; an Overview on Pathology, Integrated Disease Management Approaches, and Forecasting Models. Integrated Disease Management Approaches, and Forecasting Models.

Chakrabarti, S. K., Sharma, S., & Shah, M. A. (2022). Potato pests and diseases: a global perspective. In Sustainable management of potato pests and diseases (pp. 1-23). Singapore: Springer Singapore.

Wang, W. J., Li, J., Zhao, G., Wang, Y. P., Fan, S., Dong, Y. Q., ... & Zhan, J. (2025). Pathogenicity and virulence of Phytophthora infestans: The ever-evolving threat to food security and its sustainable management strategies. Virulence, 16(1), 2586882.

Chaithra, M., Shivakumar, K. V., Mahesha, H. S., & Nayak, A. M. (2025). Phytophthora. In Compendium of Phytopathogenic Microbes in Agro-Ecology: Vol. 1 Fungi (pp. 571-607). Cham: Springer Nature Switzerland.

Manoharachary, C. (2019). Diversity and Taxonomy of Fungi. Division of Plant Pathology, 1.

Al-Abedi, H. F., & Al-Abedi, S. F. (2025). Chromistian Fungal Analogous from Consolidation to Evolutionary and Genomic Exploration.

Sundar, J. S. (2025). From pathogen to prey: new insights into the role of effectors from Phytophthora infestans. Acta Universitatis Agriculturae Sueciae, (2025: 7).

Brus-Szkalej, M. (2019). The biology and ecology of Phytophthora infestans: the role of cell wall proteins in development, pathogenicity and potato defence activation. Acta Universitatis Agriculturae Sueciae, (2019: 81).

Subhani, M. N. (2016). Phytophthora infestans (Mont.) de Bary (Peronosporales: Pythiaceae): A review of life history, evolutionary status of pathogen, host range, mating types, symptomology, disease cycle, means of dispersal, extent of losses, and management. Journal of Global Agriculture and Ecology, 5(2), 108-126.

Singh, K., & Gurjar, H. REPRODUCTION IN FUNGI. Pathways of Plant Pathology, 24.

Suffert, F., Sache, I., & Lannou, C. (2011). Early stages of septoria tritici blotch epidemics of winter wheat: build-up, overseasoning, and release of primary inoculum. Plant pathology, 60(2), 166-177.

Mahaffee, W. F., Margairaz, F., Ulmer, L., Bailey, B. N., & Stoll, R. (2023). Catching spores: Linking epidemiology, pathogen biology, and physics to ground-based airborne inoculum monitoring. Plant Disease, 107(1), 13-33.

Flieger, A., Frischknecht, F., Häcker, G., Hornef, M. W., & Pradel, G. (2018). Pathways of host cell exit by intracellular pathogens. Microbial cell, 5(12), 525.

Amponsah, J., Tegg, R. S., Thangavel, T., & Wilson, C. R. (2021). Moments of weaknesses–exploiting vulnerabilities between germination and encystment in the P hytomyxea. Biological Reviews, 96(4), 1603-1615.

Knaus, B. J., Tabima, J. F., Shakya, S. K., Judelson, H. S., & Gruenwald, N. J. (2020). Genome-wide increased copy number is associated with emergence of dominant clones of the Irish potato famine pathogen Phytophthora infestans. MBio, 11(3), 10-1128.

Sánchez-Vallet, A., Fouché, S., Fudal, I., Hartmann, F. E., Soyer, J. L., Tellier, A., & Croll, D. (2018). The genome biology of effector gene evolution in filamentous plant pathogens. Annual review of phytopathology, 56(1), 21-40.

Rech, G. E., Bogaerts-Márquez, M., Barrón, M. G., Merenciano, M., Villanueva-Cañas, J. L., Horváth, V., ... & González, J. (2019). Stress response, behavior, and development are shaped by transposable element-induced mutations in Drosophila. PLoS Genetics, 15(2), e1007900.

Chen, H., Shu, H., Fang, Y., Song, W., Li, Z., Fang, Y., ... & Dong, S. (2022). Genome-wide analyses of histone modifications and chromatin accessibility reveal the distinct genomic compartments in the Irish potato famine pathogen Phytophthora infestans. Biorxiv, 2022-02.

Mat Razali, N., Cheah, B. H., & Nadarajah, K. (2019). Transposable elements adaptive role in genome plasticity, pathogenicity and evolution in fungal phytopathogens. International journal of molecular sciences, 20(14), 3597.

Ranjha, L., Howard, S. M., & Cejka, P. (2018). Main steps in DNA double-strand break repair: an introduction to homologous recombination and related processes. Chromosoma, 127(2), 187-214.

Pinto, L., Soler-López, L., Serrano, A., & Sánchez-Rodríguez, C. (2025). Between host and invaders: the subcellular cell wall dynamics at the plant–pathogen interface. Annual Review of Plant Biology, 76(1), 255-284.

Chepsergon, J., Nxumalo, C. I., Salasini, B. S., Kanzi, A. M., & Moleleki, L. N. (2022). Short linear motifs (SLiMs) in “Core” RxLR effectors of Phytophthora parasitica var. nicotianae: a case of PpRxLR1 effector. Microbiology Spectrum, 10(2), e01774-21.

Du, Y., Weide, R., Zhao, Z., Msimuko, P., Govers, F., & Bouwmeester, K. (2018). RXLR effector diversity in Phytophthora infestans isolates determines recognition by potato resistance proteins; the case study AVR1 and R1. Studies in mycology, 89(1), 85-93.

Akram, A. (2023). Functional characterisation of the flax rust AvrP effector protein.

Yu, T. Y., Sun, M. K., & Liang, L. K. (2021). Receptors in the induction of the plant innate immunity. Molecular Plant-Microbe Interactions, 34(6), 587-601.

Song, Y. (2024). Exploration of plant recognition receptors as tools to engineer crop immunity (Doctoral dissertation, Dissertation, Tübingen, Universität Tübingen, 2024).

DeFalco, T. A., & Zipfel, C. (2021). Molecular mechanisms of early plant pattern-triggered immune signaling. Molecular cell, 81(17), 3449-3467.

Berindean, I. V., Taoutaou, A., Rida, S., Ona, A. D., Stefan, M. F., Costin, A., ... & Muntean, L. (2024). Modern breeding strategies and tools for durable late blight resistance in potato. Plants, 13(12), 1711.

Angmo, D., Sharma, S. P., & Kalia, A. (2023). Breeding strategies for late blight resistance in potato crop: recent developments. Molecular biology reports, 50(9), 7879-7891.

Pereyra, R. T., Rafajlović, M., De Wit, P., Pinder, M., Kinnby, A., Töpel, M., & Johannesson, K. (2023). Clones on the run: The genomics of a recently expanded partially clonal species. Molecular Ecology, 32(15), 4209-4223.