TECHNICAL MANUAL

Potato late blight

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Potato **late blight**

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Preface

The development of new technologies has given specialists a better understanding of the biology of *Phytophthora infestans* (Mont.) de Bary. However, much of this information is presented piecemeal in specific publications that at times are difficult to understand. Integrating all the new information can be even more difficult. This guide is an attempt to present some of the new information in a summarized form that gives a broad view of the problem. The CIP (International Potato Center) Project for Integrated Management of Late Blight has developed a wide variety of training activities for professionals, technicians and community leaders, as well as participatory research programs using the Farmer Field School methodology to facilitate knowledge transfer. Furthermore, integrated management strategies have been developed together with national researchers and rural development institutions, considering agro-ecosystem care, farmer and environmental health, all of which are particularly affected by inappropriate and inefficient use of fungicides.

We hope this guide will be useful for researchers, teachers, students, technicians and farmers interested in this topic. Chapters on the pathogen, the disease and integrated management attempt to cover as much information as possible. For this reason, special care has been taken in analyzing some of the specialized and recent bibliography in order to present the information in a didactic and objective way. The chapter on evaluation of disease resistance has been laid out based on specialists' experience and aims to be a useful guide for those who need to evaluate resistance of new varieties or evaluate new control strategies.

The authors

"He who loves practice without theory is like the sailor who boards ship without a rudder and compass and never knows where he may cast." *Leonardo da Vinci*

Introduction

Potato late blight, caused by *Phytophthora infestans* (Mont.) de Bary, is one of the most devastating potato diseases worldwide. In Ireland in 1845, it caused the total destruction of the potato crop, which was the main staple food in that country, causing the deaths of thousands of people and the migration of many of the survivors to North America and other places in Europe. Since then, numerous studies in etiology, epidemiology and disease control have been made, which have also increased since the A2 mating type was found in Europe in 1984. In addition, the development of biochemical and molecular techniques has led to the improvement in genetic studies of pathogen populations. These studies sounded the alarm on the risks to potato production after the pathogen population had changed in many locations leading to the appearance of strains with resistance to certain systemic fungicides, as well as strains that are more virulent and difficult to manage. Another consequence of population change has been the coexistence of two mating types leading to the presence of oospores as a consequence of sexual reproduction of the pathogen.

TAXONOMY:

THE NAME *PHYTOPHTHORA INFESTANS* COMES FROM GREEK WORDS *PHYTO* = PLANT, AND *PHTHORA* = DESTROYER. THIS PATHOGEN, MEMBER OF THE OOMYCETES CLASS, BELONGS TO THE KINGDOM CHROMISTA AND IS PHYLOGENETICALLY RELATED TO DIATOMS AND BROWN ALGAE (FIG.1).

THE CELL WALL OF OOMYCETES IS MAINLY COMPOSED OF CELLULOSE AND ß-GLUCANS RATHER THAN CHITIN AND IS NOT ABLE TO SYNTHESIZE STEROLS. THESE FEATURES LET US ASSUME THAT OOMYCETES HAD A EVOLUTION DIFFERENT FROM FUNGI SUCH AS ASCOMYCETES AND BASIDIOMYCETES.

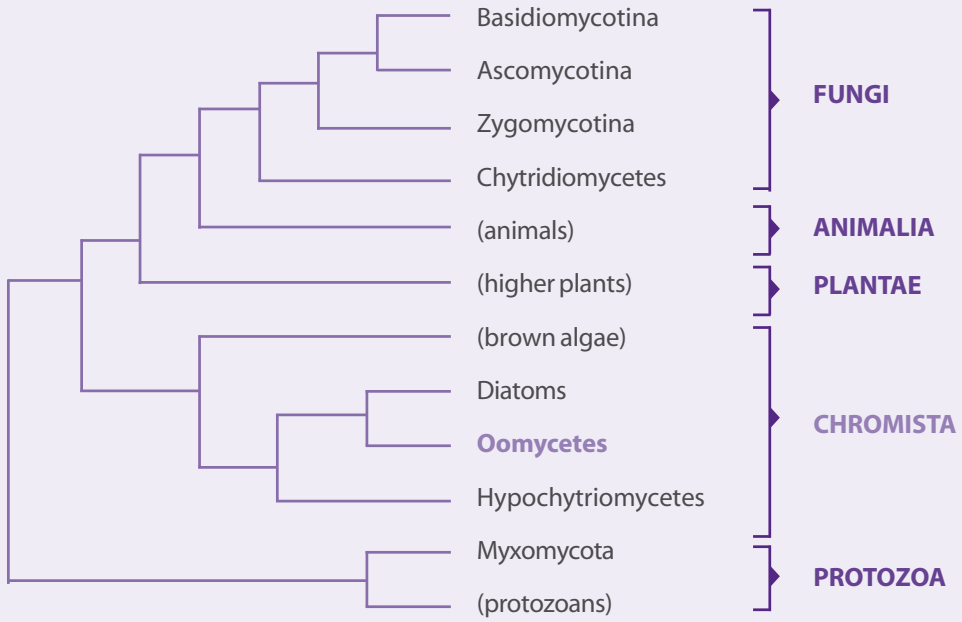


Figure 1. Schematic diagram showing phylogenetic relationships between the five Eukaryota kingdoms with emphasis in the position of Oomycetes [Adapted from Cavalier-Smith, 1987 cited by Llácer *et al.*, Eds. (1996) and adapted from Förster *et al.*, 1990 and Illingworth, *et al.*, 1991 cited by H. Judelson (1997)]

Morphology:

The mycelium is coenocytic, which means that it not divided into sections by septa. Sporangia are oval-shaped, ellipsoidal to lemon-shaped, spindle-like in the base, caducous, with less than 3 mm pedicel and semipapillate. Sporangia size varies from 36 x 22 µm to 29 x 19 µm (Fig. 2 and 3). Successively-born sporangiophores show a small swelling just below the sporangium (Fig. 4).

Figure 2. Mycelium with no septa (m); lemon-shaped and ellipsoidal sporangia (Photo: W. Pérez).

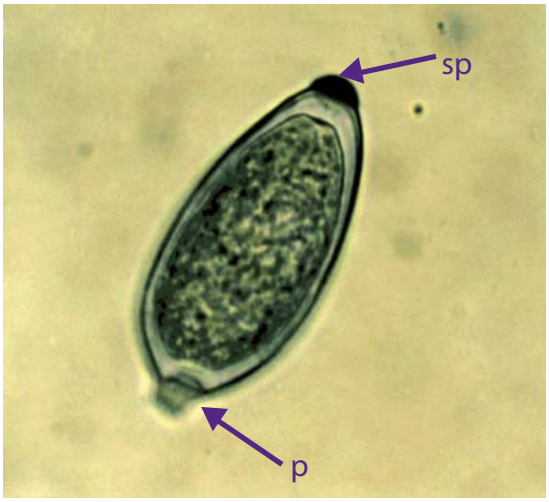


Figure 3. Lemon-shaped sporangium showing pedicel (p) and semipapilla (sp) (Photo: W. Pérez).

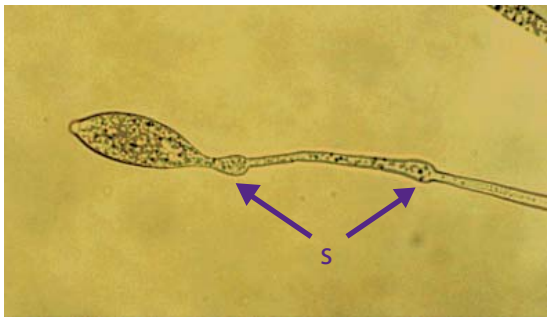


Figure 4. Successively-born sporangiophore showing swellings (s) formed just below the sporangium (Photo: W. Pérez).

P. infestans is heterothallic, with two mating types, A1 and A2. These are actually compatibility types and differ in hormone production and response rather than in sexual dimorphism. It was suggested that hormones A1 and A2, produced by A1 and A2 compatibility groups, respectively, stimulate the opposite mating type to form both male (antheridia) and female (oogonia) structures in differing degrees. Thus, isolates strongly "male" will form more antheridia than oogonia and those strongly "female" will form more oogonia than antheridia, whereas some isolates tend to be well-balanced. *P. infestans* is generally self-incompatible although some degree of selfing can occur.
Oospores formed on leaves have 30 µm (24 – 35 µm) diameter on average and the diameter of those formed on culture media ranges between 24 to 56 µm (Fig. 5).

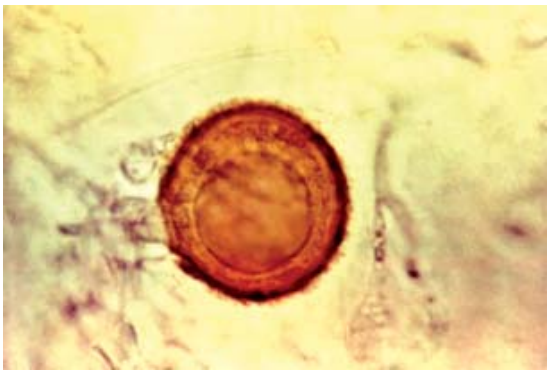


Figure 5. Typical oospore showing characteristic color and thickened wall (Photo CIP).

Hyphal swellings or chlamidospores have not been reported in this pathogen. Only in one article published in Russia, chlamidospores were reported after a 4 to 9 months incubation period in culture media at 9 - 10°C (Patrikeyeva (1979), cited by Erwin and Ribero, 1996).

Life cycle

Asexual

In free water at low temperatures, sporangia germinate indirectly producing about 8 - 12 mononucleated and biflagellate zoospores. Zoospores are formed inside the sporangium and are released when the

sporangial wall is broken at the papilla level, which allows zoospores to swim freely. Zoospores encyst on solid surfaces, which means they are brought to a halt, become round in shape and form a cell wall. Then, when humidity is present, they can develop a germ tube and penetrate leaves through stomata or form an apresorium, so the penetrating hypha enters directly through the cuticle. Once in the plant, the mycelium develops intercellularly, forming haustoria inside the cells. Extra-cell haustoria are occasionally formed (Figure 6).

With temperatures above 15° C, sporangia can germinate directly, forming a germ tube that penetrates the leaf epidermis, thus infecting the host.

Sexual

Gametangia are formed in two separate hyphae and the union of gametes occurs when the oogonium goes through the antheridium and plasmogamy takes place. This leads to fertilization and formation of one oospore with a thickened wall. The oospore is strong and is able to survive in plant debris. Under favorable conditions, the oospore produces a germ tube that forms an apical sporangium which can release zoospores or form a germ tube that serves as primary inoculum (Fig. 6)

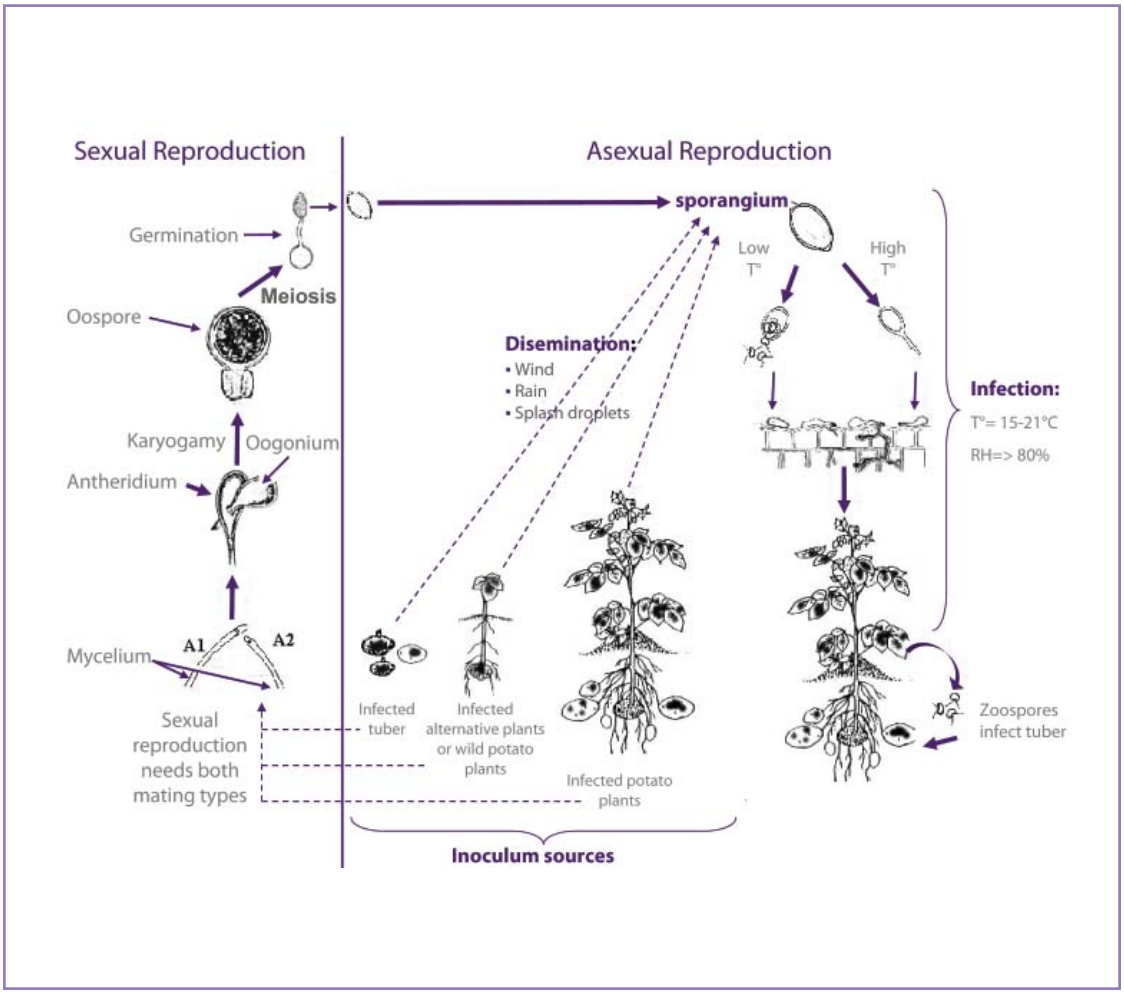


Fig. 6
Life of cycle *Phytophthora infestans* (designed by W. Pérez).

Genetic variability

The possible sources of genetic variation in *P. infestans* are sexual reproduction, mutation, mitotic recombination, parasexualism, migration, and selection.

The markers most used to characterize populations of this pathogen have been virulence, mating type, isozymes, mitochondrial haplotypes, restriction fragment length polymorphism (RFLP) and microsatellites (also known as single sequence repeats or SSR). Furthermore, an increasing number of studies based on sequencing of various nuclear or organelle genes have been developed and the full genomes of a number of isolates have been sequenced.

Unfortunately, there is no common usage of vocabulary in plant pathology. Within the literature related to *P. infestans,* and a number of other pathogens as well, the term **"virulence"** has been used as the genetic ability of a *P. infestans* race (a particular strain) to overcome host resistance, causing a compatibility reaction, that is, the disease occurs. In many other areas of biology, the term virulence refers to the amount of disease a strain causes (i.e., a quantitative phenomenon). In this paper we will use the former definition to maintain consistency with the earlier literature on this disease. When virulence is used to define the ability to cause disease, the term "aggressiveness" is commonly used to describe the ability of a pathogen isolate to cause more serious disease, i.e., two isolates may be virulent (cause disease) on a potato genotype but one causes more serious disease and is thus more aggressive.
Resistance genes (R genes) encode products that identify other products in a specific way, especially other products encoded by pathogen avirulence genes. If the R gene product in a plant recognizes the avirulence gene product of a pathogen, rapid death of plant cells near the infection point occurs and the infection is stopped, i.e., there is no disease. Loss or change of avirulence genes leads to what is often called a compatible reaction and disease occurs. The term **race** groups isolates based on virulence related to R-genes in different potato genotypes. These plants are referred to as "differentials" because they are used to identify the race of a pathogen isolate.

Using virulence phenotypes to infer genetic variation in the pathogen population has many constraints because the inference is based on the phenotypic reaction of a pathogen and host without knowing the genetic makeup of either. Other genes in both the host or pathogen may also influence the reaction. Environmental conditions may also influence the reaction.

As noted, two **mating types** are needed to initiate sexual reproduction in heterothallic species. The discovery of the A2 mating type out of the Toluca valley of Mexico, considered by most researchers as the pathogen's center of origin, was the first evidence of major change in the population of *P. infestans* worldwide, which until then had only reproduced asexually outside Mexico. Since then, the A2 mating type has been reported world wide.

Pathogen **resistance to fungicides** occurs when certain strains have lower sensitivity than normal to a particular product or class of products. This resistance is the result of stable and hereditary mutations. Resistance to the active ingredient metalaxyl and other phenylamides has been reported in *P. infestans* populations worldwide, becoming a limiting factor when using this type of fungicides. Temporary reduction in sensitivity to a fungicide is an adaptation trait of the pathogen; however, because it is not hereditary, it can be reverted by changing chemical control strategies.

Isozymes are variants of an enzyme with the same or similar catalytic activity. **Allozymes** are a special type of isozymes in which variants are codified by the same locus. Therefore, they are allelic to one another

and constitute good markers for studying population genetics when allelic frequencies need to be known. More than 50 isozymes have been characterized in *P. infestans*, the most useful for markers being Glucose-6-phosphate isomerase (GPI) and Peptidase (Pep), because of the polymorphism present between genotypes (Fig. 7 and Table 1). Nonetheless, accurate detection of these enzymes is somewhat work intensive and one notes a reduction in their use in the recent literature.

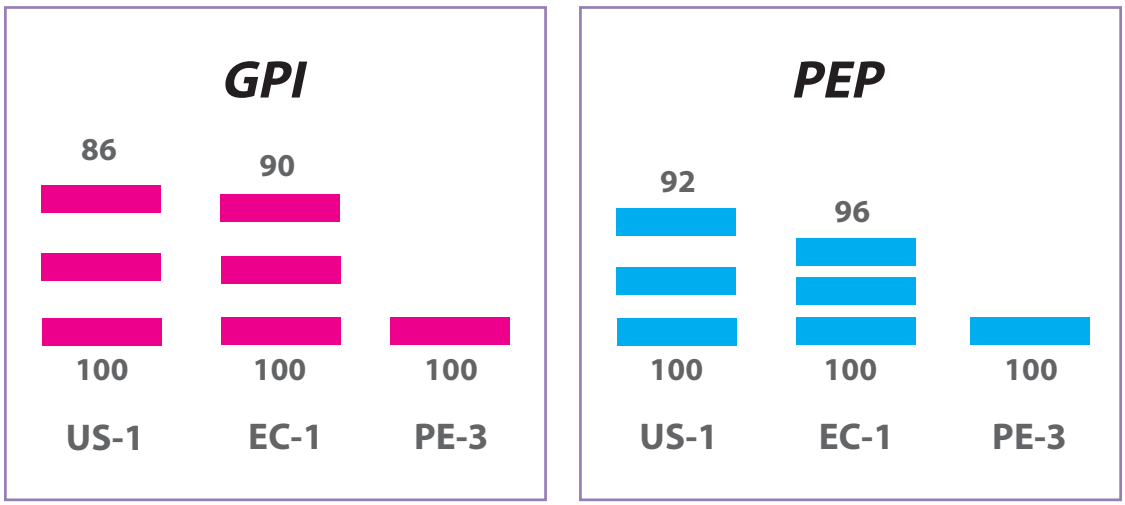


Figure 7.
Diagram of electrophoretic migration of Glucose-6-phosphate isomerase (GPI) and Peptidase (Pep) isozymes on cellulose acetate gels (CAE), from three lineages of *Phytophthora infestans*.

	ISOZYMES	
***Phytophthora infestans* Lineage**	**Glucose-6-phosphate isomerase (GPI)**	**Peptidase (PEP)**
US – 1	86/100	92/100
PE – 3	100/100	100/100
EC – 1	90/100[a]	96/100[b]
US – 6	100/100	92/100
US –7	100/111	100/100
US – 8	100/111/112	100/100

Table 1.
Electrophoretic migration values for GPI and PEP isozymes on cellulose acetate gels, reported for some lineages of *Phytophthora infestans*.
[a] Electrophoretic migration on starch gel.
[b] Electrophoretic migration on poliacrylamide gel.

Genetic information of the pathogen stored in chromosomal DNA and certain organelles (for example, mitochondria) may be studied by using molecular markers such as RFLP and SSR, which identify various lineages. The term "**clonal lineage**" involves all those isolates that descend asexually from a marker-identified genotype. DNA fragments amplified and visualized by electrophoretic techniques, and hybridized DNA fragments detected by chemiluminescence in autoradiograhies, are the "DNA fingerprints" of each pathogen isolate which allow its characterization and differentiation among reported clonal lineages. The RG57 probe, frequently used in RFLP to characterize *P. infestans* isolates, has led to the identification of 25 different bands, many of which have been shown to be polymorphic among clonal lineages (Fig. 8).

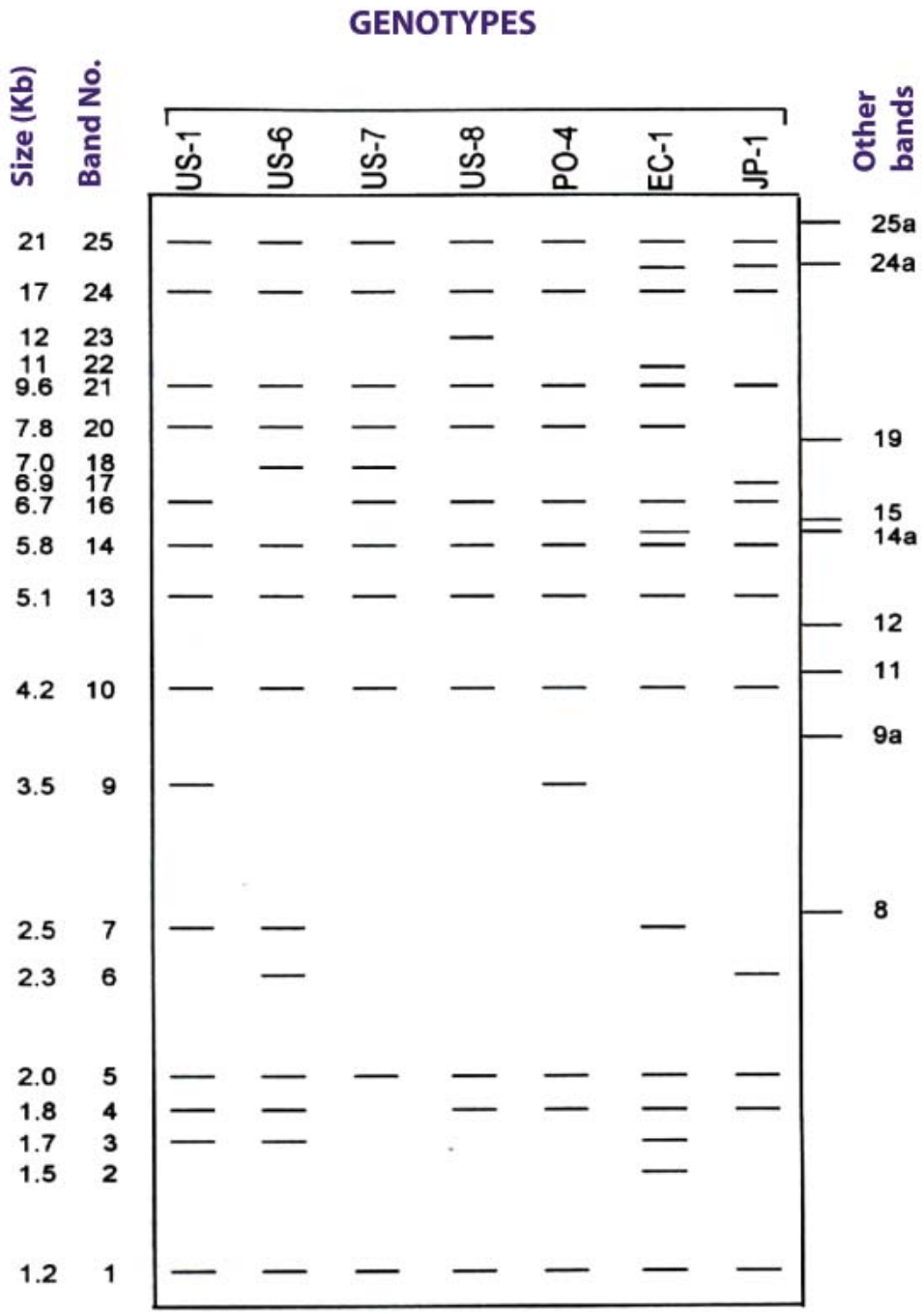


Figure 8. Graphic representation of electrophoretic migration of DNA fragments using the RG57 probe, reported for some *Phytophthora infestans* genotypes. Each band represents a different genetic locus that segregates for the presence or absence of bands (Forbes, 1998).

Analysis of mitochondrial loci helps to detect migration events. Apparently, mitochondrial DNA is uniparentally transmitted and each genotype contains a unique mitochondrial haplotype. If an unknown genotype is introduced to a new area, mitochondrial DNA will help as a marker for the clonal progeny. Until now, haplotypes Ia, Ib, IIa and IIb have been reported (Fig. 9 and Table 2).

Some published *P. infestans* lineages	Mitochondrial haplotype
PE-3 , US – 7, US – 8	Ia
EC – 1	IIa
US – 1	Ib
US – 6	IIb

Table 2.
Mitochondrial haplotypes reported for some lineages of *P. infestans*.

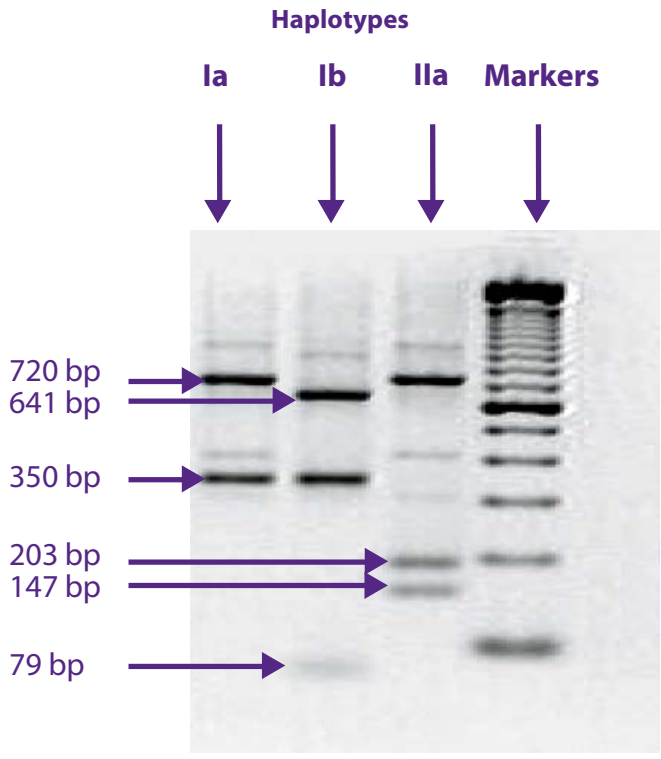


Figure 9.
Electrophoretic migration of Ia, Ib and IIa haplotypes on 2% agarose gels. (Photo: S. Gamboa)

Recently, many researchers have been using SSR markers because they are highly specific, single locus, codominant, polymorphic, reproducible and a smaller amount of pathogen DNA is needed (Cooke and Lees, 2004).

The Disease

LATE BLIGHT CAUSED BY *PHYTOPHTHORA INFESTANS* IS THE MOST IMPORTANT DISEASE OF POTATO (*SOLANUM TUBEROSUM* L.) AND A VERY SERIOUS DISEASE OF TOMATO (*S. LYCOPERSICUM*), PEAR MELON (*S. MURICATUM* AIT.) AND SEVERAL OTHER SOLANACEAE ARE ALSO AFFECTED BY THIS PATHOGEN.

Symptoms on potato:

Leaves: Spots are light to dark brown in color, water-soaked, irregularly shaped, sometimes surrounded by a yellow halo and not limited by leaf veins (Fig. 10). Symptoms begin to develop near the edges or leaf tips (Fig. 11). Under high humidity conditions, a white mildew growth is formed on the underside of the leaves, which represents the pathogen structures (sporangiophores and sporangia) (Fig. 12). Lesions expand rapidly, turn dark brown, develop necrosis and kill tissue. In the field, severely affected plants have a distinctive odor, due to rapid decomposition of foliar tissue (Fig. 13).

Figure 10. Necrotic spots with yellow border, caused by *P. infestans* (Photo: W. Pérez).

Figure 11. Initial lesions on edges and leaf tips (Photo: W. Pérez).

Figure 12. Whitish mycelium present on the leaves underside (Photo: W. Pérez).

Figure 13. Plants severely affected by late blight (Photo: W. Pérez).

Stems and petioles: Lesions are necrotic, elongated, of 5 – 10 cm of length, brown to black color, usually located from the middle third to the higher part of a plant, showing vitreous consistency (Fig. 14 and 15). When the disease reaches the stem, it is easily broken when people or field machines pass by, or when there is a strong wind (Fig. 16). With high humidity, there is also sporulation on these lesions but not as profuse as in leaves.

Figure 14. Characteristic lesions at the tip and stem of the plant (Photo: W. Pérez).

Figure 15. Elongated and dark brown lesions present on the stem (Photo: W. Pérez).

Figure 16. Affected stem breaks easily (Photo: W. Pérez).

Tubers: Affected tubers show irregular areas, slightly depressed. The skin turns a reddish-brown (Fig. 17). In a cross-section, finger-like extensions can be seen from the external surface to the tuber medulla. In advanced stages of the disease, a granular chestnut-brown to brown rot can be found (Fig. 18). Under these conditions, secondary rot may occur, caused by other fungi (*Fusarium* spp.) and bacteria (*Erwinia* spp, *Clostridium* sp, etc.), causing tuber disintegration and making diagnosis difficult.

Figure 17. Irregular reddish-brown colored lesions on tubers surface (Photo: Collection CIP).

Figure 18. Necrotic stretch marks from surface to the inner part of tuber (Photo: W. Pérez).

Epidemiology

In the absence of a sexual cycle, the pathogen survives as mycelium in tubers of volunteer plants, seed tubers (Fig. 19) or discarded tubers near crop fields. Sporangia can also survive several days and even weeks in humid soil; however they do not survive freezing temperatures. Sprouts developed from infected tubers

constitute the initial inoculum; mycelium grows through the stem and reaches the soil surface. When mycelium reaches aerial parts of the plant, sporangia are formed and dispersed by the wind or are splashed to neighbor plants. Sporangia are produced during wet nights and are dispersed to new leaves in the mornings, to reinitiate the cycle. The germ tubes of sporangia or zoospores form appressoria and penetrate through cells adjacent to the occlusive cells of stomata. They can also penetrate the periclinal wall of epidermal cells and form an intercellular mycelium. In a couple of days (4 days in optimal conditions: moderate temperatures and high humidity), after infection has started, new sporangiophores emerge through stomata and produce numerous sporangia that will infect other plants. In just one season, many asexual generations can be produced. Under humid conditions, sporangia located in leaves and stems are washed off and pulled down to the soil where they can produce zoospores and infect tubers near the soil surface. Infection takes place through wounds or lenticels. Once inside the tuber cells, haustoria are formed, in the same way they are formed in leaves, and use cell content as food. Infections during the season may occur when tubers are exposed to contaminated foliage or sporangia still present in the soil. Most of these tubers get infected with rot in the soil through secondary infections produced by other microorganisms, induce infections under inappropriate storage conditions, or mycelium survives on seed tubers until the next season.

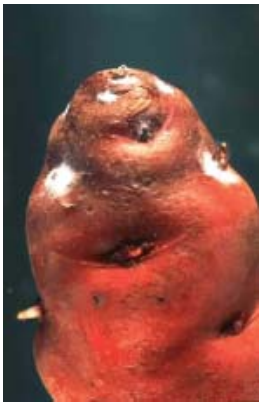


Figure 19. Mycelium of *P. infestans* developing on seed tubers, after inappropriate storage (Photo: W. Pérez).

Figure 20. Conditions of cloudiness and drizzling that favor late blight epidemics (Photo: O. Ortiz).

Management of Late Blight

INTEGRATED MANAGEMENT IS THE USE OF DIFFERENT DISEASE CONTROL METHODS (FIG. 21). IT IS APPLIED TO REDUCE OR AVOID LOSSES, SO THAT FARMERS ACHIEVE A BETTER PROFIT AS WELL AS AVOID RISKS FOR ENVIRONMENT AND HUMAN HEALTH. ONE NEEDS TO CONSIDER THAT THESE CONTROL METHODS DO NOT EXCLUDE EACH OTHER.
THE MAIN COMPONENTS OF LATE BLIGHT MANAGEMENT INCLUDE GENETIC, CHEMICAL, CULTURAL AND BIOLOGICAL CONTROL.

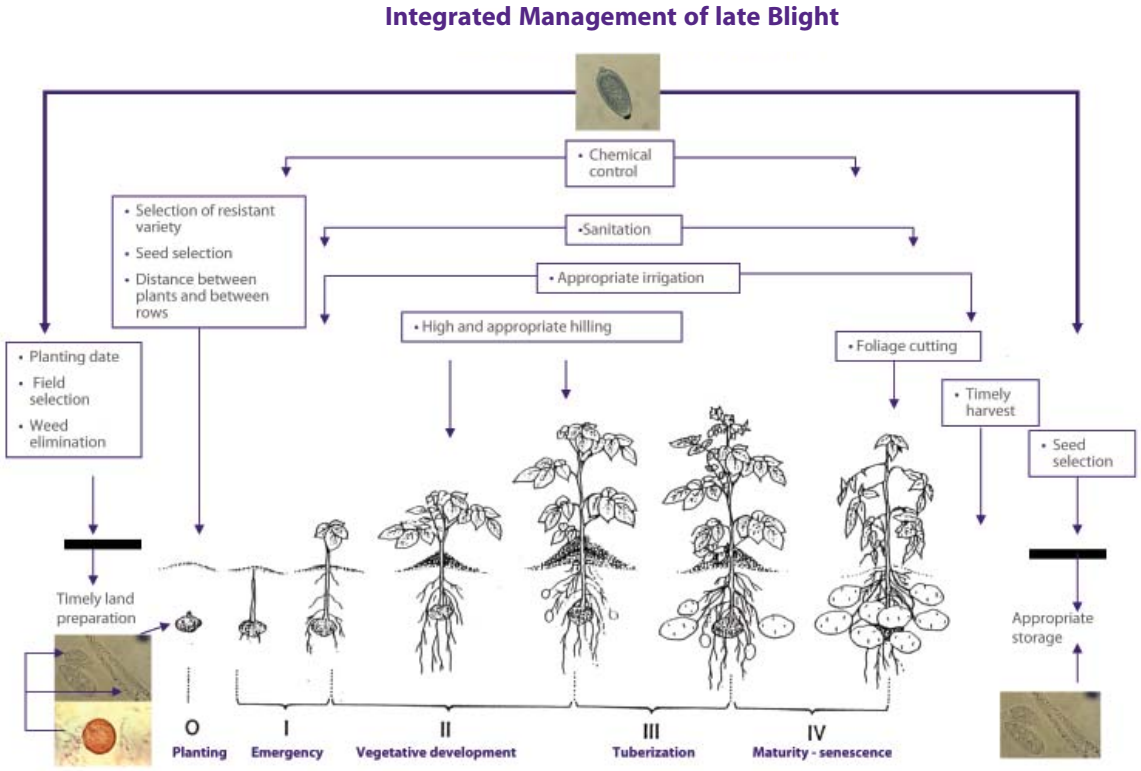


Figure 21. Scheme of integrated management of potato late blight (Designed by W. Pérez).

Genetic Control

Genetic control refers to the use of varieties or species of the host that have resistance to the pathogen which acts to stop or slow down disease development. There are two ways that resistance to *P. infestans* is expressed in the potato plant. The first one is characterized by triggering a hypersensitivity response (HR) as small necrotic lesions and is called race-specific resistance, vertical resistance, qualitative resistance, unstable resistance or complete resistance. It is governed by R genes with a strong effects that produce products which in turn interact with products of avirulence genes (Avr) of the pathogen. Most major genes known until now mainly come from *S. demissum*, however a number of new genes have recently been detected in *S. bulbocastanum* and other *Solanum* spp. This resistance is race-specific; its inheritance is qualitative and in the past never had long duration. The exact way in which products of R genes and Avr

genes interact is unknown; however, diverse models have been proposed.

The second type of resistance is governed by minor genes of additive effect and is called general resistance, quantitative resistance, polygenic resistance, non-specific resistance, partial resistance, horizontal or field resistance. Inheritance is quantitative and because it is governed by several or many genes, it is theoretically more stable and effective against all the pathogen races.

Some authors hypothesized that there is also some degree of race specificity for this type of resistance. Furthermore, adaptation for greater pathogen aggressiveness in host genotypes with general resistance has been identified. Integrating genetic resistance and chemical control helps, in reducing the use of fungicides, decreases production costs and reduces damage to human health and the environment.

The International Potato Center at present has improved material with putative horizontal resistance to late blight, high yield, earliness and good culinary quality (Table 3). These clones are available for distribution and interested parties should contact the International Potato Center.

Table 3. Improved clones with putative horizontal resistance to late blight and good culinary quality, available at the International Potato Center.

CIP Clone	Skin color	Flesh color
392634.52	Purple	White
393339.242	Purple	Cream
393382.44	Red	Yellow
393280.64	Red	Cream
393385.39	Red	Cream
393385.57	Red	Cream
393280.57	Red	Cream
391585.5	Pink /cream	White
393083.2	Pink /cream	Cream
391011.17	Cream	Yellow
391058.175	Cream	Yellow
391580.3	Cream	Yellow
392633.64	Cream	Yellow
393248.55	Cream	White
391583.25	Cream	Cream
392633.54	Cream	Cream
392657.171	Cream	Cream
393075.54	Cream	Cream
393079.24	Cream	Cream
393079.4	Cream	Cream
393242.5	Cream	Cream
393077.54	Cream / pink	White
392617.54	Cream / pink	White
392657.8	Cream / pink	White
393371.58	Cream / pink	White
391585.167	Cream / pink	Cream
392637.1	Cream / pink	Cream
392637.27	Cream / pink	Cream
393077.159	Cream / pink	Cream
393084.31	Cream / pink	Cream
393371.157	Cream / pink	Cream
393371.164	Cream / pink	Cream
393385.47	Cream / pink	Cream
393220.54	Cream / pink	Cream
393073.197	Cream / russet	Cream
393085.5	Cream / russet	Cream

Source: Breeding Program for Late Blight Resistance Database. 2006. International Potato Center. Lima – Peru.

Chemical Control

Chemical control involves the use of chemical products capable of preventing infection or of slowing down the disease once it has started. Products used to control late blight are classified as contact, systemic and translaminar (Table 4).

Contact

They act on plant surface and stop germination and/or penetration of the pathogen, reducing primary sources of the disease. They are also known as protectant, residual or contact fungicides. Copper fungicides and dithiocarbamates are among the most important (Table 4). They only protect the area where fungicide is applied; leaves formed after application of the product will not be protected against the pathogen.

Systemic

These products are absorbed through the foliage or roots. Translocation takes place from bottom to top, sometimes the other way round, internally through xylem and phloem. They are able to protect leaves formed after the application. They inhibit some or various specific phases of pathogen development. The constant use of certain products has caused the appearance of pathogen strains resistant to these fungicides.

Translaminars

These are products capable of moving through the leaf but not from leaf to leaf. For this reason, leaves formed after the product has been sprayed will not be protected against the pathogen.

The use of chemicals to control late blight started almost 140 years ago. At the beginning, products such as sodium chloride, lime and sulfur were used but, they were not efficient. The first effective compound was the Bordeaux mixture, discovered in the 1880s, composed of copper sulfate and lime. The Bordeaux mixture was widely used in potato until other cupric compounds proved to be more efficient. One of them, copper oxychloride, is still used to control blight.

In the 1940s, ethylenebisdithiocarbamates (EBDCs) were introduced to the market. Some of these products, such as zineb, maneb, metiran, mancozeb and propineb, increased the group of fungicides used to fight late blight.

Systemic fungicides were introduced to the agricultural market in the 1970s. Metalaxyl, ofurace, oxadyxil and benalaxyl, belonging to phenylamides, are the most effective products because they have curative effects, meaning, they can kill the pathogen even after plant infection. The main disadvantage of this group is that the pathogen population quickly develops resistance to them.

The most common method of preventing blight in tubers is spraying the foliage. Foliar application can reduce disease in tubers due to: i) reducing sporulation, ii) reducing the viability of sporangia on leaves, and iii) product residues falling from leaves may inhibit motility of zoospores in the soil. As may be supposed, not all the fungicides applied to the foliage will be equally effective in controlling blight on tubers.

Due to the wide range of products offered in the market, farmers have difficulty deciding when and what to apply. The decision involves many factors. However, some general principles can be useful for the producer. In general, the treatment for late blight is preventive, that is, applications are made before symptoms appear. The goal is to keep the field free of blight but in many cases this is very difficult. In some cases, producers have reported beginning applications after symptoms appear, but there is no clear evidence of the efficiency of this nor how it should be done.

Systemic products are more efficient in young plants, where new tissue grows fast. Because resistance to the product could appear, the use of systemic or translaminar products after infection is not recommendable but, it is in fact done. If a farmer thinks his crop is not well protected during the period favorable to infection, he should consider using a systemic or translaminar product.

Contact products do not protect new tissue (grown after application) and are washed away by rain. The amount of fungicide remaining on the leaf depends on the product and the amount and nature of the rain.

Table 4. Fungicides used to control late blight [1].

Group of Fungicides	Chemical Group	Active ingredients	Mode of action	Mobility[2]
Phenylamides	Acylalaninas	Benalaxyl	Interferes with ribosomal RNA synthesis	Systemic
		Furalaxyl		
		Metalaxyl		
		Metalaxyl-M (mefenoxam)		
	Butyrolactone	Ofurace		
	Oxazolidine	Oxadixyl		
Benzamides	Metilbenzamides	Zoxamide	Inhibits ß-tubuline assembly during mitosis	Contact
Fungicides QoI [3]	Imidazolinona	Fenamidone	Inhibits respiration at Qo	Translaminar
	Strobilurin	Azoyxystrobin		
Fungicides QiI [4]	Cyanoimidazol	Cyazofamid	Inhibits respiration at Qi	Contact
Fungicides C5[5]	Diarylamine	Fluazinam	Stops cellular energy production	Contact
Carbamates	Carbamate	Propamocarb	Affects cell membrane permeability	Systemic
Fungicides CAA [6]	Cinnamic acid amide	Dimetomorph	Affects cell wall synthesis	Translaminar
	Valinamide carbamate	Iprovalicarb		Systemic
Phosphorus-thiolates & Dithiolanes	Dithiolane	Isoprothiolane	Affects cell membrane and phospholipids synthesis	Systemic
Inorganic copper	Copper	Bordeaux mixture	Multi-site inhibitors	Contact
		Copper hydroxide		
		Copper oxychloride		
		Copper oxide		
Dithiocarbamates	Dithiocarbamate	Ferbam		
		Mancozeb		
		Maneb		
		Propineb		
		Zineb		
		Ziram		
Phthalimides	Phthalimide	Captan		
		Folpet		
Chloronitriles	Phthalonitrile	Chlorothalonil		
Sulphamidas	Phenyl sulphamide	Tolylfluanid		
Cyano-acetamide oximes	Acetamides	Cymoxanil	Unknown	Translaminar
Phosphonates	Organophosphate	Fosetyl-Al	Multi-site activity	Systemic
U 9	Acylpicolide	Fluopicolide	Modifies cell location of spectrin-like proteins	Translaminar

[1] Group of fungicides, chemical group, active ingredient and mode of action, according to FRAC, 2006.
[2] Classification of fungicides in systemic, translaminar and contact.
[3] Fungicides QoI = (Quinone outside Inhibitors)
[4] Fungicides QiI = (Quinone inside Inhibitors)
[5] Fungicides C5 = (Uncouplers of oxidatives phosphorylation)
[6] Fungicides CAA = (Carboxylic acid amides)

Resistance to fungicides

Resistance to fungicides means less than normal sensitivity to those products in a pathogen population. This kind of resistance is the consequence of stable and hereditary mutations. Resistance to active ingredient metalaxyl is one of the best examples reported among *P. infestans* populations worldwide, becoming a limiting factor for using this fungicide. A temporary decrease of sensitivity to a particular fungicide would be an adaptation of the pathogen, however, as it is not inherited, it may be reverted by changes in chemical control strategies.

Two types of risk of resistance to fungicides have been reported: fungicide inherent risk and pathogen inherent risk. Chemical features of the active ingredient and its mode of action on the pathogen are determinant factors for the fungicide inherent risk. Therefore, there are fungicides of high, medium and low risk for the development of resistance. Characteristics of pathogen life cycle, its reproductive rate, mode of dispersal and its mutation potential are associated with the pathogen inherent risk. Selection pressure of resistant isolates of the pathogen to a particular fungicide in large crop areas is related to the pathogen inherent risk. Therefore, there are pathogens of high, medium and low risk for the development of resistance.

The combination of both types of risk shows the real risk for the appearance of resistance to fungicides. A particular case is that of *Phytophthora infestans* which quickly developed resistance to phenylamide fungicides (metalaxyl, metalaxyl-M (mefenoxam), furalaxyl, oxadixyl, benalaxyl and ofurace) but not to dimethomorph , iprovalicarb, fluazinam, cymoxanil, azoxistrobin and fenamidone (fungicides QoI), propamocarb and organotins. That is why the Fungicide Resistance Action Committee (FRAC) has classified *P. infestans* as a high risk pathogen for phenylamide-type fungicides and only a medium risk pathogen for fungicides with other modes of action.

Antiresistance Management Strategies

- Restrict the number of high-risk fungicide applications.
- Mix a high-risk fungicide with a low-risk fungicide to be sure spores will not survive.
- Alternate applications of high-risk fungicides with low-risk fungicides, including the use of fungicides with different modes of action.
- Add other integrated management practices, different from those of the chemical component in order to avoid disease development.

Cultural control

Cultural control involves all the activities carried out during agronomic management which alter the microclimate, host condition and pathogen behavior in such a way that they avoid or reduce pathogen activity.

Planting time

Schedule planting time, especially in places where planting is made under irrigation, to avoid the period of higher incidence of the disease. This is not always possible in continuous production areas.

Selection of crop fields

Soils must have good drainage and adequate aeration, in order to avoid moisture on foliage and ground. Those areas remaining wet due to excessive soil moisture or excessive shading are potential sources for incidence of late blight. Some traditional techniques as "huacho rozado" in Colombia and Ecuador, which apparently improve drainage and aeration, have been associated with reduction of late blight (Unpublished data).

Destruction of volunteer plants and weeds
Avoid potato monocropping to escape primary inoculum likely to be present in plants or tuber debris infected during the previous season. Destroy any other alternative host, not only for *P. infestans* but for other diseases and pests.

Selection of variety
It is advisable to use resistant varieties. Combining varieties should be avoided in order to achieve adequate agronomic management of the crop and better disease control. Nevertheless, some authors recommend the mixture of varieties to reduce disease severity and obtain adequate yields, particularly the combination of susceptible and resistant varieties.

Selection of seed
Use of healthy seed tubers for planting must be guaranteed. Sometimes seed can be infected with *P. infestans* without blight symptoms. So far, there is no evidence that infected seed can be "cleaned" or healed with fungicides. However, there is a risk that infected tubers will sporulate and contaminate more tubers during the storage process or transportation. This is particularly problematic in countries where seeds are cut. In case contamination is likely to occur, it is possible to avoid an increase by treating seed with an effective product against *P. infestans* (Table 4).

Distance between plants and between rows
Distance between plants and between planting rows must be appropriate to reduce moisture on the foliage. This practice should be related to the variety and purpose of the crop (seed or consumer potato). However, data generated about the effects of plant density on late blight incidence are not consistent and often farmers must make density decisions based on other demands.

Hilling
Make high, well-formed hills to avoid or reduce contact of tubers with sporangia or zoospores coming from infected foliage. Hilling high has also been related to less severity of blight on foliage, because it promotes better drainage and aeration of soil causing foliage to dry faster.

Plant nutrition
Some authors have reported that high doses of phosphorus and potassium reduce late blight whereas high doses of nitrogen increase its incidence. Phosphorus and nitrogen apparently have contrary effects on tuber blight. Nitrogen slows down tuber maturity, which favors blight appearance, while phosphorus reduces incidence, accelerating maturity. A recent investigation in the Andean region proved that fertilization effects on late blight were less than effects of fertilization on yield so farmers generally make fertilization decisions based on yield.

Foliage cutting
Fifteen days before harvesting, foliage should be cut and removed from the field. In some countries, desiccants (sulfuric acid) or an herbicide (for example, DIQUAT) is used. Nonetheless, sulfuric acid is very dangerous and Diquat may damage tubers under certain conditions. It seems that several tuber diseases are favored when stem and root tissues rot. Therefore, "green lifting" (or "green harvest") has been studied in the Netherlands; it consists of harvesting the tubers and putting them back in the soil, without any stem or roots. Based on this investigation, we could suppose that uprooting the plant (instead of cutting it at soil level), would be favorable but no studies on this topic are known. Because of its simplicity and efficacy, it

seems that cutting foliage with a machete is generally recommended for small farmers, because it reduces late blight incidence in tubers due to early removal or destruction of plant foliage prior to harvesting.

Irrigation
Avoid excessive furrow irrigation, especially in soils with deficient drainage, because it may create micro-climates that favor disease or tuber rot. In places where sprinkler irrigation is used, do not irrigate in the evening as leaves will remain wet for a longer period of time and will favor foliage infection, exposing tubers to a potential infection.

Sanitation
In those areas where the disease is sporadic or limited to few sources of infection, desiccants should be applied to eliminate initial sources of inoculum, in order to prevent pathogen spread. Infected leaves can be easily removed from small plots or gardens.

Timely harvest
Harvest in a timely fashion and avoid field work under humid conditions, which favor tuber infection and further disease spread.

Destruction of discarded tubers
After harvesting, it is recommended to pick up discarded tubers (rotting, damaged, etc.) and use them to feed pigs or, lacking that, they should be otherwise discarded (composting or burying) to avoid their becoming a source of primary inoculum or a reserve for other pests and diseases.

Appropriate storage
Healthy tubers must be stored in order to avoid infections during the storage period.The use of diffused light stores is recommended.The use of seed tubers with green sprouts coming from these stores may result in a more uniform crop which can be harvested earlier,thus reducing exposure time to late blight.

Biological control

Biological control consists of reducing disease through the interaction of one or more live organisms with the disease-causing pathogen. A wide variety of investigations have reported the antagonistic effect of several microorganisms against *P. infestans*. Among these microorganisms are: *Serratia* spp., *Streptomyces* spp., *Pseudomonas* spp., *Bacillus* spp., *Trichoderma* spp., *Fusarium* spp., *Aspergillus* spp., *Penicillium* spp., *Myrothecium* spp., etc. Biological control is not common and reports of successful control are rare.

The use of garlic or onion extracts or infusions, or some vegetable ferments as barley, wheat, rice, garlic, tara, etc., has also been successful under laboratory and greenhouse conditions but, there is no clear evidence for its efficiency in the field.

Several reports mention that sprinkling liquid compost on potato leaves and stems makes microorganisms present in this liquid compete with *Phytophthora infestans* for living space on the surface of both organs, making pathogen establishment and further infection difficult. However, success has not yet been proved on commercial fields. By the same , preventive application of commercial bio-fungicides formulated from *Bacillus subtilis,* which theoretically hinder pathogen establishment, interrupting its development and inducing plant-acquired resistance, is under investigation because results obtained under field conditions are sometimes contradictory and usually show low efficacy.

Evaluation of late blight resistance

LATE BLIGHT IS A POLYCYCLIC DISEASE BECAUSE THE CAUSAL AGENT IS ABLE TO REPRODUCE AND RE-INFECT OTHER PLANTS IN THE SAME CROP SEASON. TO EVALUATE RESISTANCE OF PARTICULAR GENETIC MATERIAL TO THE DISEASE, IT IS ADVISABLE TO USE THE PARAMETER KNOWN AS AREA UNDER DISEASE PROGRESSIVE CURVE (AUDPC). THE ASSESSMENT OF THIS PARAMETER IS BASED ON THE PERCENTAGE OF LEAF AREA AFFECTED BY LATE BLIGHT, WHICH IS DETERMINED VISUALLY AND REGISTERED SEVERAL TIMES DURING THE OCCURRENCE OF THE EPIDEMIC.

The AUDPC is simple to assess because it uses multiple evaluations and does not need data transformation. It is very useful to carry out comparative analysis among varieties, genotypes or treatments in the same experiment and in the same crop season.

One disadvantage of using AUDPC is that it cannot be used to compare results from different experiments; these values are not comparable for different reasons. Another inconvenience is that the difference between resistant and susceptible materials can be underestimated when evaluations are made after the susceptible cultivar has been destroyed, or when evaluations start after the disease has already severely affected susceptible genotypes.

Considerations in evaluating resistance

Evaluations of the percent of leaf area blighted must be initiated at the beginning of the epidemic.
Time intervals for registering the disease should not be long; it is advisable for them to be shorter (1 week) if climate conditions are favorable to disease development.
Evaluations should stop when susceptible genotypes are near total destruction. Since susceptible materials cannot get more infected, the more resistant ones will tend to catch up.
The date of each evaluation must be recorded to calculate the AUDPC.
The relative AUDPC (rAUDPC) should be used to compare data from different experiments. This value is better than AUDPC but it may introduce bias when comparing experiments.
To calculate the rAUDPC divide the AUDPC by N, where N'=(The total number of days between the first and last evaluation) multiply by100.

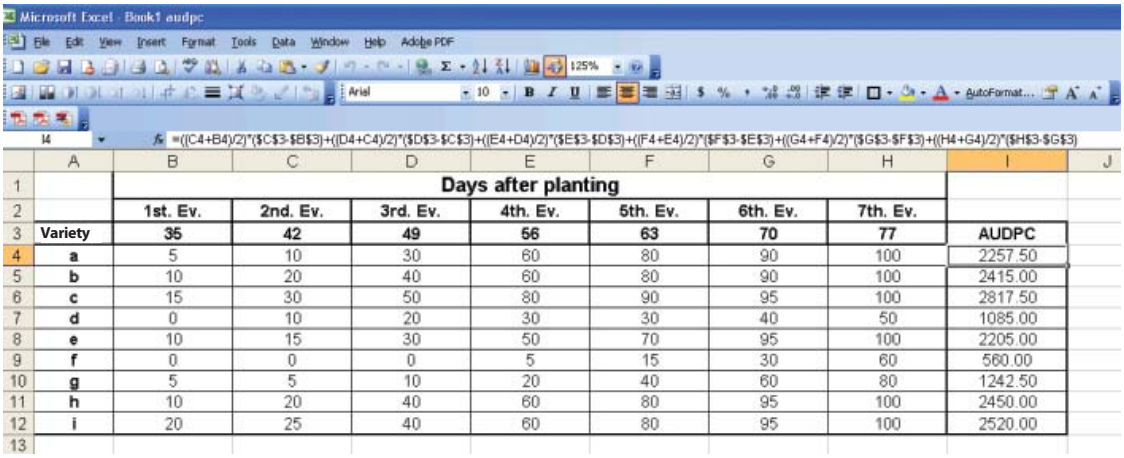
Assessment of AUDPC, a Microsoft Excel example

1. Evaluations of the percentage of diseased foliar area corresponding to each genotype according to the date of evaluation should be recorded. Number of days after planting in which evaluation was per formed must also be recorded (Figure 22). In the example, nine varieties (a, b, c, d, e, f, g, h, and i) were evaluated at 7, 14, 21, 28, 35, 42 and 49 days after planting.

2. Place the cursor over cell I4, which corresponds to clone "a" area, and calculate the AUDPC by using the following formula:

```
=       ((C4+B4)/2)*($C$3-$B$3)+((D4+C4)/2)*($D$3-$C$3)+((E4+D4)/
        2)*($E$3-$D$3)+((F4+E4)/2)*($F$3-$E$3)+((G4+F4)/2)*($G$3-
        $F$3)+((H4+G4)/2)*($H$3-$G$3)
```

3. Press **ENTER**, and the AUDPC value "2765.00" will be displayed in cell 14
4. Copy cell I4 to the other cells (I5 to I12) and press **Edit**. Select **PASTE SPECIAL**, select **FORMULAS** and click **OK**. Values of AUDPC will be displayed in every copied cell



	A	B	C	D	E	F	G	H	I	J
1		Days after planting								
2		1st. Ev.	2nd. Ev.	3rd. Ev.	4th. Ev.	5th. Ev.	6th. Ev.	7th. Ev.		
3	Variety	35	42	49	56	63	70	77	AUDPC	
4	a	5	10	30	60	80	90	100	2257.50	
5	b	10	20	40	60	80	90	100	2415.00	
6	c	15	30	50	80	90	95	100	2817.50	
7	d	0	10	20	30	30	40	50	1085.00	
8	e	10	15	30	50	70	95	100	2205.00	
9	f	0	0	0	5	15	30	60	560.00	
10	g	5	5	10	20	40	60	80	1242.50	
11	h	10	20	40	60	80	95	100	2450.00	
12	i	20	25	40	60	80	95	100	2520.00	
13										

Figure 22. EXCEL spreadsheet with necessary data to assess AUDPC.

Interpretation of results

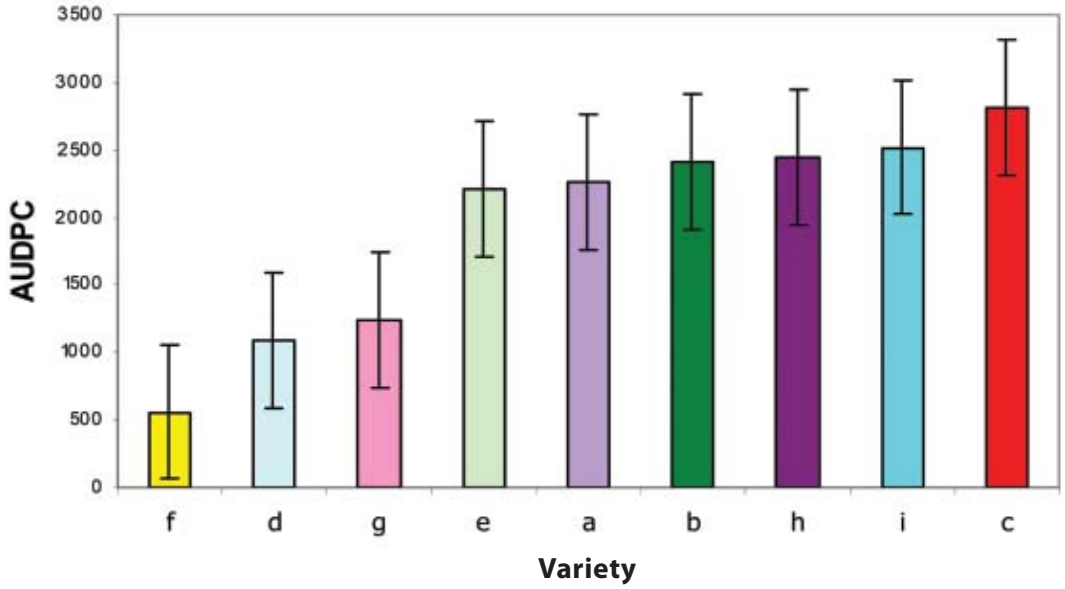
AUDPC estimates the area under the disease progress curve. This value is expressed as % - days, that is, the sum of non transformed daily percentage values of infection. Highest values will correspond to more susceptible varieties and lowest values will correspond to more resistant varieties (Table 5) .

Variety	AUDPC
f	560.00
d	1085.00
g	1242.50
e	2205.00
a	2257.50
b	2415.00
h	2450.00
i	2520.00
c	2817.50

Table 5. AUDPC calculated for potato varieties tested in the example.

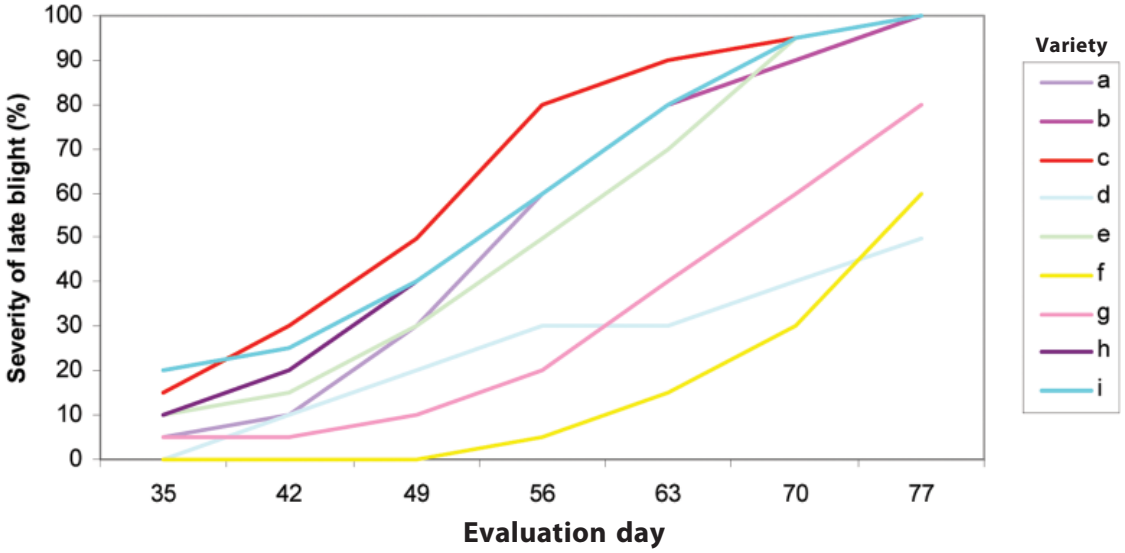
Results can be shown in graphics, where standard deviation values can be added if several repetitions have been made. It is recommended to have varieties with known resistance as indicators of susceptibility or resistance to late blight (Figure 23).

Figure 23. AUDPC values of potato varieties from Excel example



Since AUDPC is a numerical value, it alone does not explain the behavior of varieties during an epidemic, that is why it is necessary to plot the disease progress curve using data from evaluations of the diseased foliar area and evaluation day (Figure 24).

Figure 24. Disease progress curves of potato varieties from Excel example

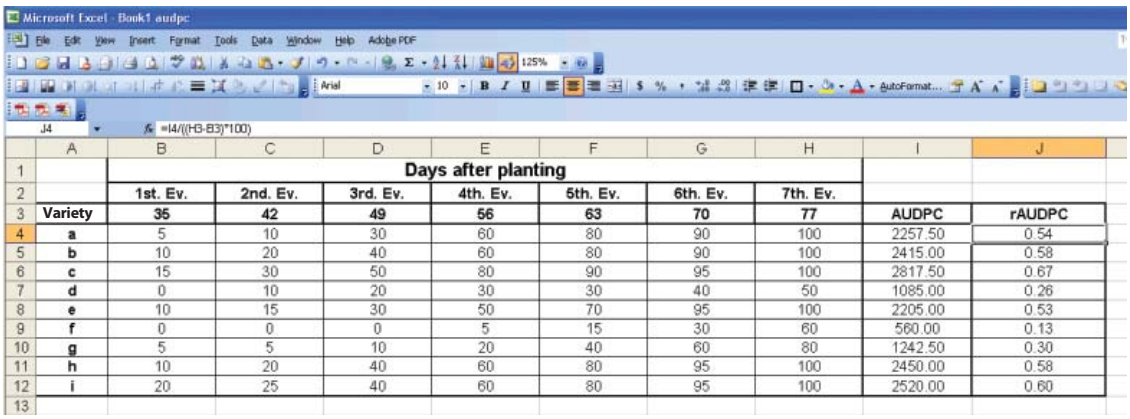


Microsoft Excel Assessment of rAUDPC

1. The same spreadsheet prepared to find AUDPC is used.
2. Place the cursor over cell J4, corresponding to clone "a" and calculate the rAUDPC by using the following formula:

```
= I4/((H3-B3)*100)
```

1. Press **ENTER** and rAUDPC value "0.18" will be displayed in cell J4 (Figure 25).
2. Copy cell J4 to the other cells (J5 to J12) and press **Edit**. Select **PASTE SPECIAL**, select **FORMULAS** and then **OK**. Values of rAUDPC will be displayed in every cell copied.



	A	B	C	D	E	F	G	H	I	J
1		Days after planting								
2		1st. Ev.	2nd. Ev.	3rd. Ev.	4th. Ev.	5th. Ev.	6th. Ev.	7th. Ev.		
3	Variety	35	42	49	56	63	70	77	AUDPC	rAUDPC
4	a	5	10	30	60	80	90	100	2257.50	0.54
5	b	10	20	40	60	80	90	100	2415.00	0.58
6	c	15	30	50	80	90	95	100	2817.50	0.67
7	d	0	10	20	30	30	40	50	1085.00	0.26
8	e	10	15	30	50	70	95	100	2205.00	0.53
9	f	0	0	0	5	15	30	60	560.00	0.13
10	g	5	5	10	20	40	60	80	1242.50	0.30
11	h	10	20	40	60	80	95	100	2450.00	0.58
12	i	20	25	40	60	80	95	100	2520.00	0.60
13										

Figure 25. Microsoft EXCEL Assessment of rAUDPC.

Interpretation of results

An evaluation of 100% of late blight-diseased foliar area for all evaluation dates would have a value of 1.0. All values of rAUDPC are expressed as the ratio of this value. Low values of rAUDPC will indicate low infection levels during the evaluation period; therefore, they will correspond to more resistant varieties.

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