Technical Information Bulletin 4

Late Blight of Potato

Phytophthora infestans

Jan W. Henfling

Late blight attack in the field

INTERNATIONAL POTATO CENTER (CIP)

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Jan W. Henfling

CIP
P.O. Box 5969
Lima - Peru

Location:
Av. La Universidad s/n
La Molina - Lima

Tel. 366920
Telex 25672 PE
Cable CIPAPA, Lima

Late Blight of Potato

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Objectives. Study of this bulletin enables you to:

- analyze the importance of late blight,
- describe the symptoms of the disease,
- describe the biology of the fungus,
- explain disease epidemiology,
- list control measures,
- distinguish between race specific resistance and general resistance,
- evaluate resistance in the field.

Study materials

- Infected plants and tubers.

Practicals

- Describe symptoms in the field and in stored tubers.
- Demonstrate sporulation as explained in Section 3.
- Observe and discuss in the field the types of resistance.
- Evaluate late blight severity in the field according to Section 7.

Questionnaire

1 What importance has late blight in your country?

2 What do farmers in your country know about the disease? What do they call it?

3 Describe symptoms on leaves.

4 How do symptoms of late blight differ from those caused by early blight?

5 What plant species does the fungus affect?

6 How does the mycelium develop?

7 Why is sporulation on the underside of leaves more abundant than on the upperside?

8 How are the zoosporangia spread?

9 Describe indirect spore germination.

10 What are *mating types*?

11 Name sources of infection.

12 How do tubers become infected?

13 How much time is required for production of zoosporangia, liberation of zoospores, and penetration?

14 Under what conditions does the disease develop and spread most rapidly?

15 At optimum conditions, how much time does a disease cycle take?

16 Describe the principles to forecast late blight.

17 Generally, what agronomic measure may be utilized to reduce the disease?

18 Why should affected foliage be destroyed at least one week before harvest?

19 How effective are preventive fungicides?

20 How effective are systemic fungicides?

21 What is the objective of integrated management of late blight?

22 How effective is general resistance?

23 Which type of resistance is generally preferable?

24 Why may it be necessary to evaluate the resistance under local conditions?

25 In a screening program where the breeding populations contain R genes, why is it important to select only clones or varieties that show at least some sporulating lesions?

Late Blight of Potato

Phytophthora infestans

Late blight is the most serious fungal disease of potatoes. It affects leaves, stems, and tubers, and can destroy a potato field within a few days. The disease is caused by the fungus *Phytophthora infestans* and develops most rapidly at low temperatures and high humidity. In developed agricultural regions, the disease can relatively easily be controlled by fungicides; this however, is hardly feasible for the small farmer in many developing areas. Other possibilities of control exist, but their application requires knowledge about the disease.

1 IMPORTANCE OF LATE BLIGHT

Late blight is not only the most serious fungal disease of potatoes, it also occurs almost everywhere where potatoes are grown and is especially important in the traditional potato growing areas.

On several occasions the disease has reached disastrous proportions. Well documented is the Irish Famine of around 1845 to 1850 when, as a result of a late blight epidemic, one million persons of a total of eight million inhabitants died and another 1.5 million left the country. The population of Ireland had completely depended on the potato that virtually formed its only food source. The biology of the disease and methods of its control were as yet entirely unknown. In other parts of Europe and in North America, the disease was as severe as in Ireland. In these areas, however, a famine was avoided due to a more varied food supply.

In spite of increased knowledge on the disease, late blight continues to be a major constraint to potato production worldwide. If not controlled, losses may reach 100 percent, and even lower infection levels may make the crop unfit for storage.

The disease can relatively easily be controlled by the use of fungicides. In many developing areas, however, chemical control is hardly feasible for the subsistence farmer due to the high cost of fungicide applications. In addition, in nearly all developing countries, the fungicides or their active ingredients are imported, making the potato an expensive vegetable and at the same time costing the country valuable foreign exchange.

2 SYMPTOMS

In an advanced stage of disease development, symptoms resemble those caused by frost attack. Plants severely affected with late blight produce a distinctive odor, which results from the breakdown of plant tissue. The disease affects leaves, stems, and tubers.

Leaves. The earliest symptoms of the disease are often present on lower leaves. They consist of small, pale to dark green spots that change into brown or black lesions, depending on the humidity of the air. Lesions begin frequently at leaf tips and margins. A pale green or yellow border, a few millimeters wide, often separates dead from healthy tissue. Under conditions of high humidity and cool temperatures, lesions expand rapidly. Sporulation may be visible at the lower surface of the leaves as a white mildew surrounding the lesions. It may become inconspicuous during the day, when the lesions dry and shrivel. In less than a week, the disease may spread from the first infected leaflets on a few plants to most plants in a field. Leaves may drop off.

Stems. Either by direct infection or extending from the leaves, lesions may develop on petioles and stems, where they expand lengthwise. Infected stems are weakened, and may collapse thereby causing death of plant parts above the lesion.

Tubers. Infected tubers show a superficial and irregular discoloration. Dry and brown necrotic lesions penetrate from the surface into the tuber tissue. Secondary pathogens (mainly bacteria) may convert the almost odorless (faint vinegar smell) dry rot typical for *P. infestans* into a badly smelling soft rot. Late blight normally does not spread during storage, but secondary infections may contaminate other tubers.

Note: Do not confuse the symptoms of late blight with those of early blight caused by *Alternaria solani*. Foliage lesions of early blight are brown and brittle, and generally show concentric rings. Sporulation does **not** appear as a white mildew. Tuber lesions caused by *A. solani* are clearly demarcated. Early blight also occurs under conditions that are too dry and warm for late blight (Technical Information Bulletin 17).

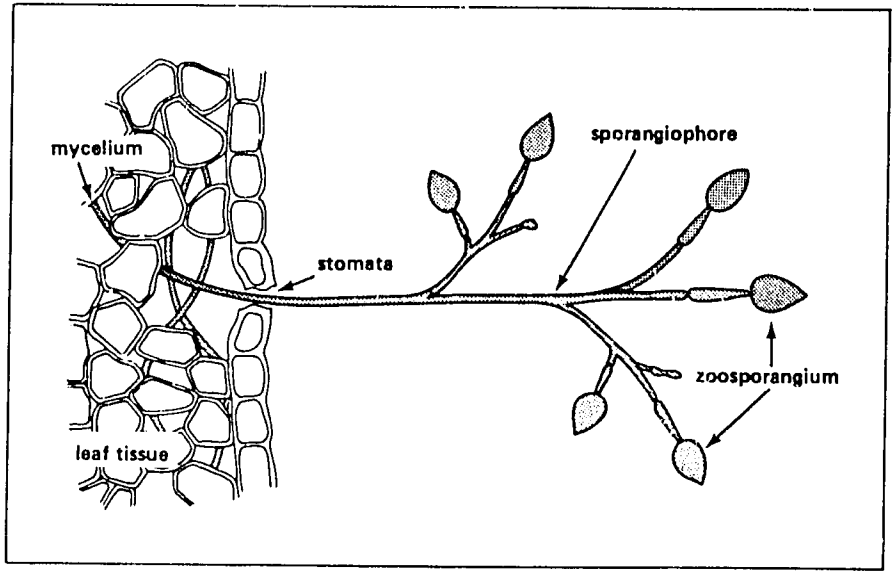
On leaves, pale to dark green spots change into brown or black lesions.
On tubers, dry and brown necrotic lesions penetrate from the surface into the tuber tissue.

3 BIOLOGY

Late blight is caused by the fungus *Phytophthora infestans* (Mont.) de Bary. The fungus affects potato, tomato, and some other plants of the *Solanaceae* family.

Most stages of the life cycle of *P. infestans* can only be seen with the aid of a microscope. In this way, fungal threads (*mycelium*) can easily be observed. They are characterized by the absence of cross walls (*septa*). The mycelium develops between cells (*intercellularly*) and only extensions of it (*haustoria*) enter the cells. Both asexual (*vegetative*) and sexual (*generative*) reproduction occurs.

Asexual reproduction. Between three and ten days after infection, depending on environmental conditions, spore-bearing organs (*sporangiophores*) emerge through openings (*stomata*) on the leaf surface. Stomata are more frequent on the underside than on the upperside of leaves. This explains why sporulation on the underside is more abundant than on the upperside. *Zoosporangia* (also called *sporangia* or *conidia*) develop at the end of these sporangiophores. When mature, the zoosporangia easily break off and are spread by the wind. Most spores are deposited after a few meters, however, travel distances of over 30 km have been reported.



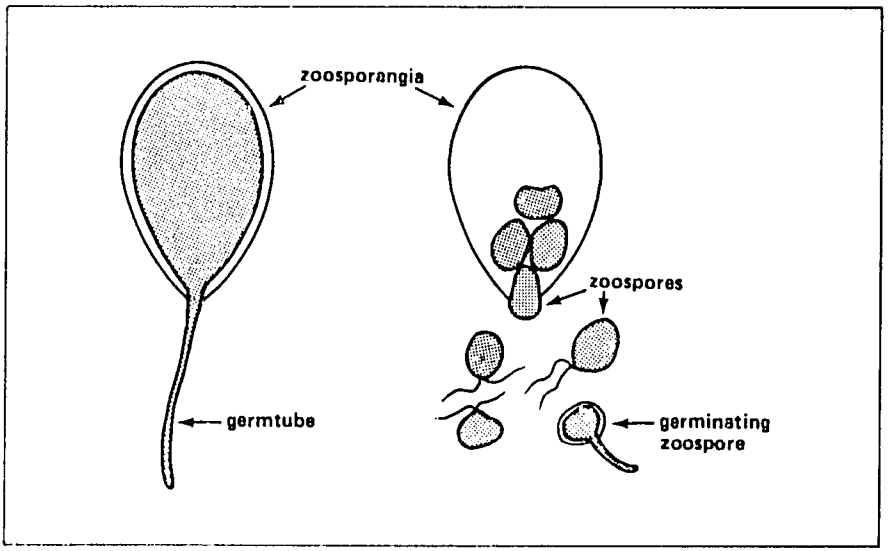
The mycelium develops between cells. Three to ten days after infection, sporangiophores emerge through stomata.

Sporulation can be demonstrated in a simple experiment: A stem with small lesions on the leaves is placed in a flask with water. Stem and flask are covered with a plastic bag. It is kept overnight at a temperature between 15 and 22 °C (room temperature). Next morning the white sporulation, especially on the underside of the leaves, can be observed.

The size of zoosporangia is just below the detection limit of the unaided eye. Their lemon-shaped form can be observed with a microscope. Zoosporangia may germinate **directly** or **indirectly**.

To a small degree, zoosporangia germinate **directly** at temperatures above 20 °C (optimum 24 °C). Zoosporangia behave as single spores. They form germtubes that penetrate into the plant tissue. In nature, direct germination seems of little importance.

Zoosporangia germinate **indirectly** at temperatures of 12 to 16 °C, each sporangium releasing from 10 to 20 swarm spores (*zoospores*). Activated by two flagellae, zoospores remain motile from a few minutes up to several hours. Under certain conditions they lose the flagellae, form a cell wall and subsequently a germtube.



In direct spore germination (left), the zoosporangium forms a germ tube. In indirect germination (right), each sporangium releases 10 to 20 zoospores.

On leaves and stems, germtubes may directly penetrate the plant epidermis (no stomata are required). On tubers, germtubes penetrate through lenticels or wounds. Since the fungus cannot survive for a prolonged period outside the host tissue zoosporangia or zoospores die, if no suitable host tissue is available.

Sexual reproduction. Until 1984, the sexual stage of *P. infestans* has only been reported in Mexico and parts of Central America. More recent reports inform about its occurrence in other parts of the world. When mycelia of different types of the fungus (called mating types A_1 and A_2) grow together, one of them may form male cells (*antheridia*) and the other female cells (*oogonia*). The oogonium grows through the antheridium, allowing fertilization. The fertilized oogonium develops into a thick-walled resting spore (*oospore*). Oospores, in contrast to zoosporangia and zoospores, can resist unfavorable conditions, such as droughts and low temperatures.

Formation of oospores helps *P. infestans* and related species to survive adverse conditions, such as the winter, dry periods, and absence of hosts. Possibly due to the absence of the A_2 mating type, oospore formation in *P. infestans* has not been observed outside Mexico and Central America.

Oospores of *P. infestans* germinate through formation of sporangia, similar to those described in asexual reproduction. After infection of a host, the resulting zoospores may start a new life cycle.

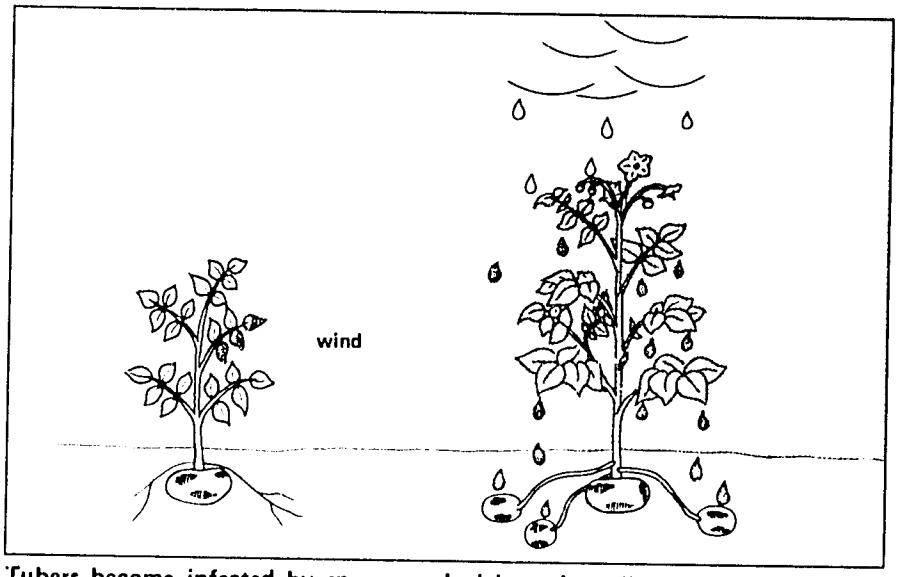
4 EPIDEMIOLOGY

With the exception of oospores which may survive in the soil, in nature the pathogen persists only in susceptible hosts. Sources of infection are:

- infected seed tubers,
- cull piles,
- neighboring potato fields,
- other host plants.

Infected seed tubers. In areas where potatoes are grown during defined seasons, diseased seed tubers are often the most important source of infection. Tubers become infected through lenticels and wounds when spores are washed into the soil by rain from infected leaves, especially when tubers are formed superficially or are not well covered by hilling. Also at harvest, tubers can become contaminated by contact with infected foliage.

Blight-infected tubers normally rot when planted in the field. However, few diseased tubers may form sprouts which then become primary sources of infection.



Tubers become infected by spores washed into the soil by rain from infected leaves.

Cull piles. Infected tubers are frequently found in cull piles. Also tubers from previous crops that have been left in the field may be infected and may form a source of primary infection for a new crop.

Neighboring potato fields. Neighboring potato fields are another source of infection especially in areas where potatoes are grown during the whole year.

Other host plants. Some other solanaceous plants may become affected by *P. infestans*. In many countries, the tomato is the most important alternative host. The pathogen also persists on wild species of potatoes that are widely distributed in the highlands of Mexico, Central and South America.

From primary infection sources, the wind disseminates the spores towards the fields. For development of the disease, temperature and humidity are of fundamental importance.

The most favorable temperature for development of the fungus (mycelium, sporangia) is around 21 °C, but the fungus remains alive in host tissues at air temperatures between 0 and at least 28 °C. Zoosporangia develop at temperatures between 9 and 22 °C.

Development of the fungus within the leaf is little affected by air humidity. However, sporangia are only formed when the relative humidity in the leaf canopy is above 95 percent. When the temperature is optimum, about eight hours of high humidity are required for production of zoosporangia, liberation of zoospores, and penetration. Water (dew, rain) must be on the leaf surface for at least two hours to allow zoospore formation, germination, and penetration.

In summary, the disease develops and spreads most rapidly at low temperatures and high humidity. Under these conditions, a disease cycle takes only three days. Unfavorable conditions may delay or temporarily interrupt the disease cycle.

For better disease management and the planning of a spray program, several methods of late blight forecasting have been developed, and are based on observation of temperature and humidity conditions. A severe late blight attack can be expected when the "leaf wetness period" exceeds 8 to 10 hours for several consecutive days, and temperatures range between 10 and 24 °C. Typical blight weather is characterized by cool, cloudy days with frequent rains.

Disease forecasting programs appear to function generally better in temperate climates, than in most of the tropical highlands where potatoes are mainly grown in the rainy season.

5 CONTROL

Development of late blight is favored when the potato variety is susceptible, and appropriate environmental conditions persist for sufficient time. Generally, any agronomic measure that affects this relationship may be utilized to reduce the disease.

Healthy seed. A basic condition for adequate crop production is the use of non-infected seed. This aliminates the primary infection source from the field.

Planting procedure. In regions with defined rainy seasons a change in planting time may help to reduce disease severity. This might however reduce the yield, as the potato requires abundant water during tuber formation.

Crop care. Any treatment that promotes rapid drying of foliage and reduces humidity within the crop helps to restrict disease development. These include wider planting distance and appropriate irrigation procedures. Sprinkler irrigation tends to increase disease severity.

Exposed tubers and those poorly covered by soil become readily infected by fungus spores washed down from the foliage. Proper hilling reduces the amount of spores reaching the tubers and may result in faster drying of the field after a rain.

Resistance. Varieties with the highest resistance to late blight should be used, if commercially acceptable. Two types of resistance, race specific resistance and general resistance, are described in more detail in Section 6.

Harvest. When the foliage has been affected by late blight, it should be destroyed mechanically or chemically at least one week before harvest. This practice reduces the chance of tuber infection by contact with infected leaves and stems and promotes skin suberization, making tubers less vulnerable to infection. In addition, this helps to reduce mechanical damage and infection by storage pathogens.

Tubers should be only harvested when they are mature (the skin should not be peeling). The soil should be dry to prevent infection through damaged skin or lenticels. Only disease-free tubers should be stored.

Crop residues, including infected tubers, should be removed from the field or plowed under. Cull piles should be covered with sufficient soil to prevent emergence of discarded tubers.

Fungicide applications. Preventive fungicides principally inhibit spore germination and penetration. Once the pathogen enters the leaves, most fungicides are ineffective. A preventive fungicide application program should start immediately after the first blight symptoms appear in a crop. Proper fungicide cover on the foliage should be maintained as long as favorable late blight conditions exist. In a susceptible crop, up to 15 applications per season may be required.

Proper dosage and application are as important as selecting an effective fungicide. Instructions given with the fungicide should be followed carefully. The spraying equipment should be kept in good condition to assure an even and fine spray application.

Observation of meteorological data may help determine when fungicide applications are necessary.

Newly developed systemic fungicides (acylalanines) are translocated within the plant. A constant and uniform coverage is less critical than with conventional fungicides, and frequency of spraying may be reduced. Granular formulations applied to the soil do not require spraying equipment. Systemic fungicides may be highly effective; however, development of resistance of the fungus to these fungicides has been reported recently.

No general recommendations for fungicide application can be given, since availability, effectivity, conditions, and regulations vary from country to country. Local experts should be consulted for recommendations in a particular area.

Integrated control. The best control is a combination of preventive measures, based on the use of resistant varieties. Costly fungicide applications can then be reduced. The objective of integrated management of late blight is not the eradication of the disease, but the most economical production of a potato crop.

6 TYPES OF RESISTANCE

Two types of resistance to late blight are generally distinguished:

- race specific resistance,
- general resistance.

Race specific resistance. As the name implies, race specific resistance is only effective against races of the fungus. It is controlled by dominant R genes (r = recessive) and is also called *R-gene resistance, major-gene resistance*, and *vertical resistance*. R genes of the potato are closely related to virulence genes of the fungus. For each resistance gene of the host, the fungus may possess a virulence gene that overcomes the resistance gene and makes the fungus compatible with its host.

Race specific resistance is a **qualitative** factor, that is, it is either effective or not, independent of the number of genes involved.

The resistance/virulence relationship may be visualized by the function of lock and key. With the help of R genes, a potato plant may be able to lock the fungus out – as long as the fungus does not possess the corresponding virulence keys. The plant may install several locks (R genes), and the fungus possess several keys. Whenever the fungus attacks with the right combination of keys, it may be able to enter the plant.

R genes cause a hypersensitive reaction in the plant tissue: the affected plant cells – and thus the invading fungal mycelium – die before the pathogen is able to reproduce itself.

R genes used in breeding have been mainly obtained from *Solanum demissum*. This potato species is native to Mexico, where the sexual stage of *P. infestans* also occurs. Coincidence of both facts led to the assumption that *S. demissum* and *P. infestans* evolved together (coevolution).

In the first half of this century, the incorporation of R genes from *S. demissum* into breeding materials resulted in spectacular success. Introduction of R genes, however, led to a change in the fungal population toward the predominance of compatible races, and each new variety was only resistant for a few years.

Relationship between some resistance genes of the potato and some virulence genes of the fungus *Phytophthora infestans* (examples)

Resistence genes of the potato	Virulence genes of the fungus 0 (none)	1	2	3	4	1.2	1.3	3.4	1.2.3	1.2.3.4
R_1		c				c	c		c	c
R_2			c			c			c	c
R_3				c			c	c	c	c
R_4					c			c		c
$R_{1.2}$						c			c	c
$R_{1.2.3.4}$										c
r	c	c	c	c	c	c	c	c	c	c

Specific resistance of the potato is controlled by dominant R genes (r = recessive). With the help of R genes, a potato plant may be able to lock the fungus out — as long as the fungus does not possess the corresponding virulence genes. The plant may combine several R genes. Whenever the fungus attacks with the right combination of virulence genes, it may be able to enter the plant.

The table shows only "compatible reactions" = c; all other relationships are "incompatible."

Presently 12 R genes are known, which in all their combinations theoretically correspond to $2^{12} = 4096$ possible races of the fungus. In view of the high variability of the fungus, breeders no longer rely on R genes, although these are widely distributed in breeding materials due to the frequent use of *S. demissum*.

General resistance. General resistance – also called *field resistance, horizontal resistance, minor gene resistance* – is not controlled by specific dominant resistance genes, as in race specific resistance. Many factors are expected to contribute to general resistance, such as the resistance of the cuticle and epidermal cells to mechanical force or enzymes, the activity of leaf substances to inhibit spore germination or penetration, the number of stomata, and many other factors.

By definition, a variety with general resistance is susceptible, but it is less susceptible than a less general resistant variety. The level of general resistance also depends on local factors, including growth conditions. However, it is effective independent of presence or absence of races of the fungus and does not break down rapidly, if at all.

General resistance is a **quantitative** factor and is controlled by many genes, each of which may contribute to a greater or lesser extent to the overall resistance. These genes can be found in many potato species, including the Mexican species, that at the same time may possess R genes.

Most breeders now concentrate on general resistance to improve varieties through a process called *recurrent selection*. General resistant progenies are intercrossed to accumulate genes of general resistance, evaluated for general resistance, and the most resistant progenies again intercrossed.

Since much of the present breeding material is derived from Mexican potato species, the two types of resistance – race specific resistance and general resistance – often occur simultaneously. This causes serious difficulties in evaluating progenies, as the two types of resistance are hard to distinguish in the field. For measuring exclusively general resistance, ideally a field should be inoculated with a race containing all virulence genes that are present in a host population.

7 EVALUATION OF RESISTANCE

For the farmer, use of resistant varieties is the most practical way to control late blight. In a national potato program, resistant material may be already available, or it can be obtained from other sources, such as CIP. Since general resistance to late blight depends on the specific environment of a potato growing region, evaluation of resistance under local conditions of the field may be necessary.

When taking the data on disease severity, carefully observe whether the symptoms are indeed due to late blight, or perhaps to other causes, such as early blight, frost, or herbicides. In bright sunlight it may be difficult to distinguish causes and level of damage. On sunny days, evaluations should be started at sunrise and completed before 11:00 a.m.

Intensity of late blight attack is commonly estimated visually on basis of the proportion (percent) of leaf area affected. At CIP, a scale has been developed where the percentages correspond to values 1 (no symptoms) to 9 (plants dead).

How often data are taken, depends on the objective of the work. As a rule, data should be taken weekly from the moment blight is first observed. To avoid that previous data influence the present evaluations, one person should take the data and another one write them down in the field book. Alternatively a small cassette recorder may be employed.

In a screening program where the breeding populations contain R genes, it is important to select only clones or varieties that show at least some sporulating lesions, as this indicates the presence of compatible races at the test site. This means that the general resistance of a clone or variety can be determined locally.

Various procedures to interprete the results and compare treatments (varieties, clones, etc.) include the *disease increase curve, infection rate (r),* and *area below the infection curve (A).* Obviously, the larger the values of r or A, the more susceptible a variety. See de following examples.

Example: Late blight experiment, Colombia, variety Tequendama, plot size 5 x 5 m, 3 replications. Late blight evaluation according to CIP scale values and the corresponding leaf areas affected (%).

Days after planting	CIP scale values in replication			Mean
	I	II	III	
28	1	1	1	1.0
36	2	4	3	3.0
42	3	4	4	3.7
48	3	5	5	4.3
57	5	6	6	5.7
63	5	7	6	6.0
69	6	7	7	6.7
77	7	8	8	7.7
83	8	8	8	8.0
89	9	9	9	9.0

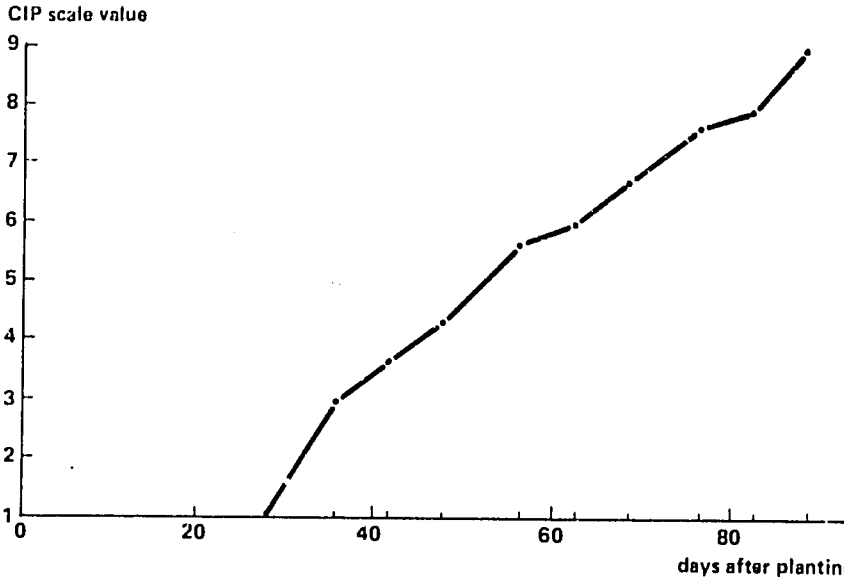
	Leaf area affected (%) in replication			
	I	II	III	
28	0.0	0.0	0.0	0.0
36	2.5	25.0	10.0	12.5
42	10.0	25.0	25.0	20.0
48	10.0	50.0	50.0	36.7
57	50.0	75.0	75.0	66.7
63	50.0	90.0	75.0	71.1
69	75.0	90.0	90.0	85.0
77	90.0	97.5	97.5	95.0
83	97.5	97.5	97.5	97.5
89	100.0	100.0	100.0	100.0

Field key for assessing potato late blight

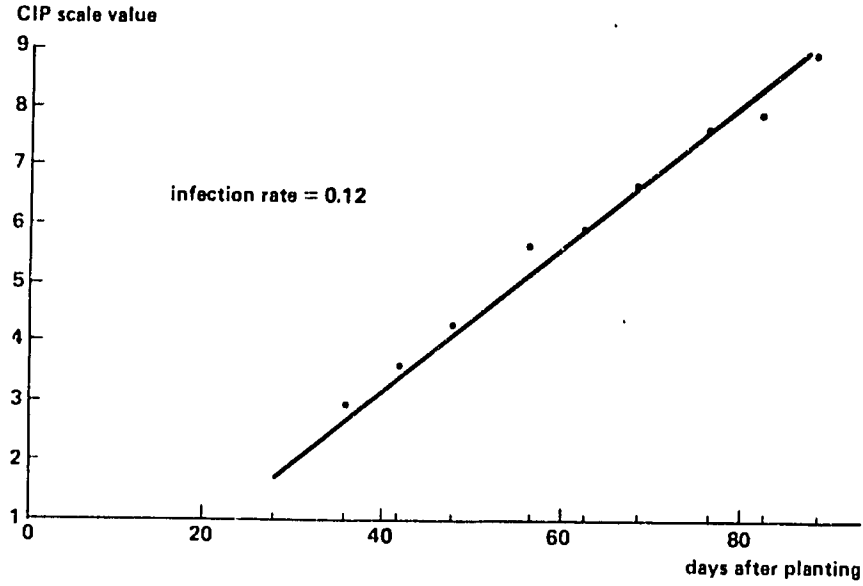
CIP scale value	Blight (%) mean	Blight (%) limits	Symptoms
1	0		No late blight observable.
2	2.5	traces - < 5	Late blight present. Maximum 10 lesions per plant.
3	10	5 - < 15	Plants look healthy, but lesions are easily seen at closer distance. Maximum foliage area affected by lesions or destroyed corresponds to no more than 20 leaflets.
4	25	15 - < 35	Late blight easily seen on most plants. About 25 % of foliage is covered with lesions or destroyed.
5	50	35 - < 65	Plot looks green; however, all plants are affected. Lower leaves are dead. About half the foliage area is destroyed.
6	75	65 - < 85	Plot looks green with brown flecks. About 75 % of each plant is affected. Leaves of the lower half of plants are destroyed.
7	90	85 - < 95	Plot neither predominantly green nor brown. Only top leaves are green. Many stems have large lesions.
8	97.5	95 - < 100	Plot is brown-colored. A few top leaves still have some green areas. Most stems have lesions or are dead.
9	100		All leaves and stems dead.

The description of symptoms is based on plants with 4 stems and 10 to 12 leaves per stem.

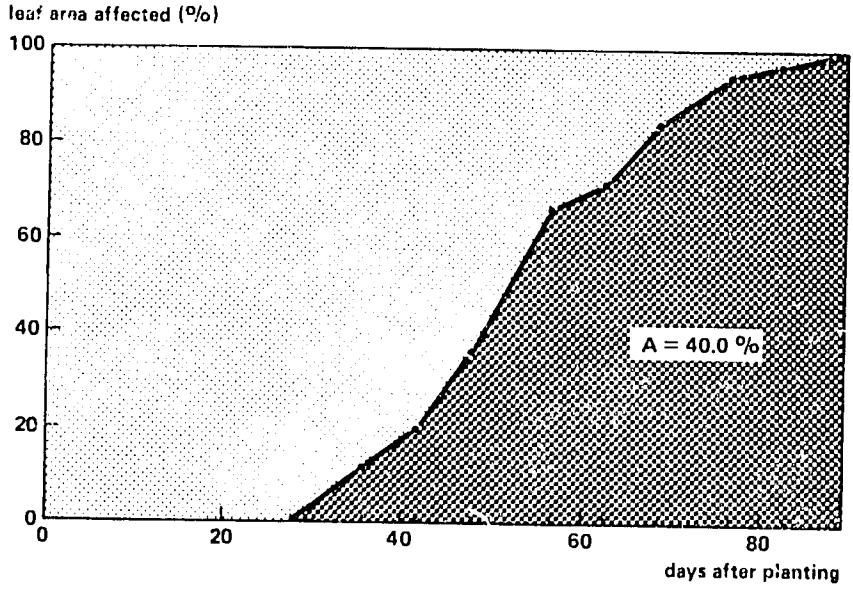
Disease increase curves (data plotted over time) of the treatments may be compared visually. Example: variety Tequendama, means of CIP scale values.



The *infection rate* (r) is the regression coefficient of the CIP scale values (means) over time (days after planting). Example: variety Tequendama, r = 0.12. r can also be calculated from the proportions of leaf area affected transformed into logits.



The *area below the infection curve* (A) is calculated from the (mean) percentages of leaf area affected drawn over time. "A" is usually expressed as proportion of the area below the infection curve (in %) in relation to the maximum area possible (leaf area 100 % affected from day of planting or day 0). Example: variety Tequendama, A = 40.0 %.



8 ADDITIONAL STUDY

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