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EDITORIAL

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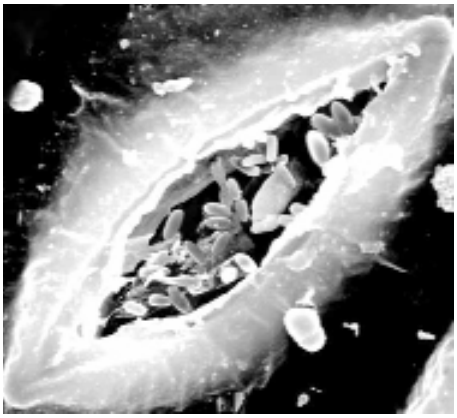
# Pathogenic plant–microbe interactions. What we know and how we benefit

Plants, like humans and other animals, also get sick, exhibit disease symptoms, and die. Plant diseases are caused by environmental stress, genetic or physiological disorders and infectious agents including viroids, viruses, bacteria and fungi. Plant pathology originated from the convergence of microbiology, botany and agronomy; its ultimate goal is the control of plant disease. Microbiologists have been attracted to this field of research because of the need for identification of the agents causing infectious diseases in economically important crops. In 1878—only two years after Pasteur and Koch had shown for the first time that anthrax in animals was caused by a bacteria—Burril, in the USA, discovered that the fire blight disease of apple and pear was also caused by a bacterium (nowadays known as *Erwinia amylovora*). In 1898, Beijerinck concluded that tobacco mosaic was caused by a "*contagium vivum fluidum*" which he called a virus. In 1971, Diener proved that a potato disease named potato spindle tuber was caused by infectious RNA which he called viroid.



Colonization of the inside of a pear-leaf stoma by the plant-beneficial bacteria *Pseudomonas fluorescens* EPS288 several days after inoculation. This bacterial strain inhibits infection by several plant-pathogenic fungi.

In 1933, Chester described the "hypersensitivity reaction" (HR) in plants [2], a type of blocking necrosis developed by non-host plants against many plant pathogens that invade their tissues. Later research proved that plants had evolved an immune response against microbes. The use of *Arabidopsis thaliana*—a crucifer plant of small size, rapid growth, and small genome—as a system model has brought about major advances in the knowledge of the genetic basis of plant–pathogen interactions. Plants, like animals, defend themselves by a combination of constitutive and inducible responses. In localized response, cell tissues react against pathogens by a type of programmed cell death consisting of electrolyte leakage from the cytoplasm and oxidative burst. In systemic defense, a signal spreads from the place of interaction, mediated by several molecules which have been identified as messengers in plants, such as salicylic or jasmonic acid, or even volatiles, such as nitric oxide or ethylene [1]. These messengers interact with specific binding proteins, which are involved in the transcriptional activation of pathogen-responsive genes, many of which are known as pathogenesis-related (PR) genes. Many products of these genes are enzymes involved in the flow of carbon from the primary to the secondary metabolism of plants, e.g. peroxydases, lypooxygenases, superoxyde dismutases, and phenylalanine-ammonia-lyase (PAL), a key enzyme in the synthesis of phenolic compounds with antimicrobial activity. Other products, such as phytoalexins, glucanases and chitinases, have also antifungal activity.

The existence of groups of host-range pathogenicity among pathogens at the subspecies level led plant pathologists to introduce the concept of races and patovars. Also, many races and pathovars show host-range specificity within cultivars—commercial or cultivated "varieties" of species of plants. The genetic basis of this strong specificity was first explained by the "gene-for-gene" theory of Flor [3], which was complemented with the elicitor-receptor model [1]. These models introduced the concept of avirulence (*avr*) genes in the pathogen, which are homologous of the resistance (R) genes in the host plant. A complementary combination of these genes results in an incompatible plant–pathogen interaction (rejection), which triggers host-cell defense mechanisms, whereas the non-complementary combination (compatible) results in infection.

Most evidences in favor of the gene-for-gene theory have been obtained from research performed with plant-pathogenic bacteria of the genera *Pseudomonas*, *Erwinia* and *Xanthomonas*. A group of genes involved are the hypersensitivity reaction and pathogenicity (*hrp*) genes, which control the capacity of bacteria to develop HR in non-host plants. The first confirmation of the role of *hrp* genes was provided by the discovery of harpins in *Pseudomonas syringae* and *Erwinia amylovora*, a type of proteinaceous elicitors of the HR. The transcription of *hrp* genes is controlled by a contact-dependent signal transduction cascade, constituting a type III secretion system, which is homologous and has features common with animal pathogenic bacteria such as *Yersinia*, *Shigella*, *Salmonella* and *Escherichia*. However, most of the proteins introduced into the host cell are extracellular virulence factors contributing to the pathogenicity of bacteria, and are coded by avirulence (*avr*) genes, which trigger programmed plant defense responses such as HR.

Compared to the immune system in animals, the inducible defense response in plants produces compounds less sophisticated and specific than immunoglobulins. However, the specificity of response in plants lies in the fact that they have developed a mechanism to detect intracellularly a specific type of proteins in the pathogen (elicitor), based on a gene-for-gene interaction recognition system for triggering

the biochemical attack against pathogens. There is now experimental evidence that *avr* genes are present in plant viruses, plant-pathogenic bacteria, and plant-pathogenic fungi. Also, about 25 plant genes involved in resistance to plant pathogens have been cloned, most of them encoding proteins with leucine-rich repeats, and they are highly conserved among angiosperms.

Another plant pathogenic bacterium which has been widely studied is *Agrobacterium tumefaciens*, a member of the family Rhizobiaceae which shares many features with symbiotic bacteria of the genus *Rhizobium*. The pathogen *A. tumefaciens* has been taken as a model of interkingdom genetic exchange in plants, because it causes tumors in several plants; such tumors are produced by the transfer of a T-DNA region of the Ti (tumor induction) plasmid to the plant cell, its integration into the chromosomes, and the expression of its encoded plant regulator genes. T-DNA is cleaved from Ti plasmid and the resultant single strands are coated with Vir proteins and secreted to the host plant cell by means of a type IV secretory pathway. Again, the *vir* cluster genes regulating the process are homologous to those encoding the secretion of pertussis toxin by the human pathogen *Bordetella pertussis*.

Certainly, plant pathogenic bacteria share many features with animal and human bacterial pathogens. However, as far as we know, none of the bacterial plant pathogens are true pathogens to humans or other animals, which also indicates a high specificity at the kingdom level. Only *Pseudomonas aeruginosa*, and less consistently *Burkholderia cepacia*, which are usually pathogens of animals, can also cause disease in plants that grow under adverse conditions.

The fact that plants are microbial living ecosystems has led to the discovery of another group of bacteria which are non-pathogenic epiphytes or endophytes of plants. Some of these bacteria interact with plants with a certain degree of specificity, and bring about beneficial traits to its host, but are unable to develop HR and to invade tissues. These beneficial plant bacteria include "plant-growth promoting rhizobacteria" (PGPR) and biological control agents (BCA). The detection of PGPR and BCA depends on efficient methods of screening, which often require the analysis of thousands of isolates to find only a few useful ones. The practical use of PGPR or BCA as microbial fertilizers or pesticides and their efficiency is strongly dose-dependent, as with chemical pesticides [4]. PGPR inhabit the rhizosphere, the volume of soil under the immediate influence of the plant root system. In the rhizosphere, secretion of organic compounds by the plant favors large amounts of an active microbial population. Inoculation of plants with PGPR, mainly of the genera *Pseudomonas*, *Serratia*, *Azospirillum* and *Bacillus*, enhances growth of the root system and of the entire plant, and often controls certain soilborne plant pathogens. These mechanisms are strongly dependent on bacteria and host plant, but in some cases a relationship has been found to exist with the synthesis of plant-growth regulators such as IAA, with siderophores which quelate iron, with biological control of soilborne plant pathogens or plant deleterious microorganisms, or even with induction of systemic defense responses. BCA are found either in the aerial plant part or in the root system. They are able to colonize, to compete for nutrients or sites of pathogen interaction with plants, and even to exert various types of antagonism against plant pathogens. In cases where the mechanism operating is antagonism, the biocontrol has been related to the synthesis of antimicrobial compounds such as bacteriocins and novel antibiotic compounds. Most BCA which provide an efficient protection of plants against infection by plant-pathogenic bacteria and fungi are non-pathogenic strains of *Pseudomonas*, *Erwinia* or *Agrobacterium*.

The advances in research on the molecular basis of plant–microbe interactions are now just being applied to improve methods to protect crops against infectious diseases. New molecules with no direct antimicrobial activity are being discovered or developed which elicit plant defense responses against pathogens, including salicylic acid, a derivative of which is the acetylsalicylic acid, commonly used in human therapy. Some of these molecules, such as benzothiadiazol (BTH), are registered for use in crop protection in some countries. Plant transformation techniques, based on the use of *A. tumefaciens* as a tool, have provided transgenic plants with engineered genes encoding HR elicitors, such as harpins, or overexpressing R genes or PR proteins, such as chitinases, with an increased resistance to many plant pathogenic bacteria and fungi. Inoculants of the plant-root system using PGPR consisting of formulations of *Bacillus subtilis* to increase plant growth and performance are currently commercially available in several countries. Several bioantimicrobials, successfully used as competitors or antagonists of bacterial and fungal plant pathogens, are also commercially delivered as formulations of *Agrobacterium radiobacter* strain K84 to prevent crown gall tumors; *Pseudomonas fluorescens* against fire blight and frost damage; *Pseudomonas syringae* for control of post-harvest fruit rot; and *Streptomyces griseoviridis* for control of many soilborne fungal diseases.

In summary, there is no doubt that, in the future, plant disease control, presently provided by chemicals, mainly fungicides and bactericides, will be complemented or replaced by new disease-control technologies emerging from the basic knowledge of plant–microbe interactions. However, before a general use in crop protection, non-targeted effects and social concern should be minimized or counteracted.

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