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Pathogenicity Genes in Plant Pathogenic Bacteria

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Abstract

A pathogen or pathogenic microorganism is usually defined as a biological agent that can cause damage to its host during, or as a consequence of, the host microorganism interaction. Damage may be inflicted directly by the microorganism (e.g. by toxins or other so-called virulence factors) or indirectly through the activity of the host immune responses. The ability of the pathogen to infect host is called pathogenicity. Microorganisms express their pathogenicity by means of their virulence, a term that refers to the relative, quantitative degree of pathogenicity. The majority of these pathogens are in the genera *Erwinia, Pectobacterium, Pantoea, Agrobacterium, Pseudomonas, Ralstonia, Burkholderia, Acidovorax, Xanthomonas, Clavibacter, Streptomyces, Xylella, Spiroplasma,* and *Phytoplasma* cause diverse, and sometimes devastating, diseases in many different plants, but they all share three characteristics: they colonize the intercellular spaces of plants, they are capable of killing plant cells, and they possess hrp genes. Plant pathogenic bacteria have evolved specialized strategies to exploit their respective hosts. Most of the mare gram-negative, of which biotropic pathogenic bacteria fundamentally possess a type III secretion system encoded by hrp genes and a variable group of genes encoding Avr effector proteins that seem to be delivered into host plant cells through this pathway to suppress plant defense responses and develop diseases symptoms. The bacterial pathogens, involve many virulence factors that are secreted in the extracellular environment of the host cells. The most studied factors are: Adherence to the host cell, with surface adhesion, Production of the degradative enzymes that destroy the plant cell walls, Toxins that are in the apoplastic cell and other complex molecules are also deployed including the exopolysaccharide (EPS) and those modulating the plant hormone production. The main objective of these review will be describe the most important types of pathogenicity genes of the main kinds of plant pathogens.

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Introduction

Bacterial diseases of plant are most severe in tropical and subtropical region of the countries, where bacteria receive ideal climatic conditions for their growth and multiplication resulting in more crop yield losses in these countries. Leaf and fruit spots, blights, cankers, vascular wilts, rots and tumors are the main characteristic symptoms of bacterial diseases. Pathogenicity has often been defined as the biochemical mechanisms whereby pathogenic microorganisms cause disease in a host plant organism (Fuchs, 1998). Virulence is defined as the degree or measure of pathogenicity shown by one or more plants.

Pathogenicity and/or virulence of gram-negative plant pathogenic bacteria are strictly dependent on the presence of secretion apparatuses in host cells, through

which they secrete proteins or nucleoproteins involved in their virulence within the apoplast or inject these substances into host cells (Buonaurio, 2008).

Pathogenicity factors that are encoded by pathogenicity genes (pat) and disease-specific genes (dsp) are crucially involved in the establishment of diseases. Some of these genes are essential for the recognition of the host by a pathogen, attachment of a pathogen to a plant's surface, formation of infection structures on or within the host tissues, penetration of the host, and colonization of host tissue. The pathogenicity genes that are involved in the synthesis and modification of the lipopolysaccharide cell wall of gram-negative bacteria may help condition the host range of a bacterium. Bacterial pathogenicity depends upon bacterial secretion systems (types i–iv), quorum sensing (QS), plant cell-wall-degrading enzymes, toxins, hormones, polysaccharides, proteinases, siderophores, and melanin. All of these systems and substances, which are essential for pathogenic infection and virulence, are produced by pathogens during bacterial pathogen–plant interactions (Agrios, 2005).

In general, plant pathogenic bacterial species belonging to *Xanthomonadaceae, Pseudomonadaceae*, and *Enterobacteriaceae* families target all types of plants that can supply them with appropriate food and shelter. The most devastating plant pathogens belong to genera such as *Erwinia, Pectobacterium, Pantoea, Agrobacterium, Pseudomonas, Ralstonia, Burkholderia, Acidovorax, Xanthomonas, Clavibacter, Streptomyces, Xylella, Spiroplasma,* and *Phytoplasma* (Prasannath, 2013). Among the about 7100 classified bacterial species, roughly 150 species cause diseases in plants obtaining nutrients from these plants for their own growth by more or less specialized mechanisms (Buonaurio, 2008).

Bacterial pathogens may involve in two main attacking strategies to extract the host nutrients such as biotrophy, in which the host plant cells are alive as long asand bacteria exploit nutrients from living cells and necrotrophy, in which plant cells are killed bybacteria and from dead cells, nutrients are extracted (Buonaurio, 2008). Bacterial interactions with plants can either be compatible or incompatible. Compatible interactions happen when the bacterium encounters susceptible host plants causing disease symptoms.

Incompatible interactions occurs when the bacterium infects a non-host plant (non-host resistance) or a resistant host plant (cultivar-specific resistance) and they will elicit the hypersensitive response (HR) that is a rapid, programmed death of plant cells in contact with the pathogen (Collmer, 1998).

Plant pathogens also express genes to adapt to the stressful conditions that are constitutively presentor that are generated by the host in response to microbial attack.

These include the production of proteins and enzymes to counter oxidative stress (e.g., glutathione S-transferase, superoxided is mutase, and catalase), as well as enzymes that may detoxifyantimicrobial compounds (Boch *et al.*, 2002). The main objective of these review will be describe the most important types of pathogenicity genes of the main kinds of plant pathogens.

Pathogenicity Genes in Plant Pathogenic Bacteria

Plant pathogenic bacteria enter the intercellular spaces of plants through wounds and/or natural openings, such as stomata and hydathodes. Therefore, bacteria do not need to penetrate the plant surface but they must have ways to adhere surface of leaves/plants.

Adhesion of bacteria to plant surfaces

Majority of bacteria do not require adhesion mechanisms except when they are moving through the xylem and phloem. The *Agrobacterium* causes crown gall disease requires attachment to plant surface as the first step in the transport of T-DNA and to develop the disease symptoms. The attachment needs three components; a glucan molecule, which requires three genes for its synthesis and export, genes for the production of cellulose and the att region of the bacteriagenome that contains several genes for adhesion (Rodríguez-Navarro *et al.*, 2007). Additionally, *Agrobacterium* also has more other genes with homology to genes of mammalian pathogens for adhesions and for pilus biosynthesis (Agrios, 2005).

Several other plant pathogenic bacteria also contain genes that encode proteins to be occupied in attachment and aggregation. *R. solanacearum, Xanthomonas, Pseudomonas* and *Xylella* have as many as 35 genes homologous to type IV pili genes, which are involved in cell to cell aggregation and protection from environmental stress in *Xanthomonas* and *Pseudomonas*, whereas type IV pili are essential in *Xylella* for the establishment of an aggregated bacterial population in the unstable environment of the xylem by adhering to the vessels in connection to components such as polysaccharides (Romantschuk, 1992).

Secretion systems of bacteria

Secretion systems are essential pathogenicity tools for bacteria because they make possible the translocation of bacterial proteins and other molecules into host plant cells. Five forms of secretion pathways are recognized on the basis of the proteins secreted by bacteria (Desvaux *et al.*, 2004). Type I and II pathways secrete proteins into the host intercellular spaces; whereas type III and IV secrete systems can deliver proteins or nucleic acids directly into the host plant cell (Ponciano *et al.*, 2003).

Type I secretion system (T1SS) has the simplest structure and it allows direct secretion of effectors from the bacterial cytosol to the outer environment. TISS present in almost all plant pathogenic bacteria and carries out the secretion of toxins such as hemolysins, cyclolysin, and rhizobiocin. They consist of ATP-binding cassette (ABC) proteins and are involved in the export and import of a variety of compounds through energy provided by the hydrolysis of ATP. Proteases and lipases from the soft rot pathogenic bacteria *Erwiniachrysanthemi* are examples of plant pathogen effectors secreted via the TISS (Palacios *et al.*, 2001). Type II secretion system (T2SS) is common in gram negative bacteria and involved in the delivery of various proteins, toxins, enzymes and other virulence factors into host. Proteins are exported in a two-step process: Firstly, unfolded proteins move to the periplasm via the Sec pathway across the inner membrane, then as processed, folded proteins go through the periplasm and across the outer membrane via an apparatus consisting of 12–14 proteins encoded by a cluster of genes (Van Sluys *et al.*, 2002). *Xanthomonas* and *Ralstonia*, which have two TIISS per cell, use them for delivery of virulence factors such as pectinolytic and cellulolytic enzymes outside the bacterium. *Agrobacterium* and *Xylella* have one Type II-SS per cell (Staceyand Keen, 2003).

Type III-SS is the most important in terms of pathogenicity of the bacteria in the genera *Xanthomonas, Pseudomonas, Ralstonia, Erwinia* and *Pantoea* is mainly due to their capability to produce a T3SS, also called injectisome (Desvaux *et al.*, 2004), by which the bacteria inject proteins (TIII-SS effectors) involved in their virulenceinto plant cells. The primary function of TIII-SS is the transportation of effector molecules across the bacterial membrane and into the host plant cell. The genes that encode protein components of the TIII-SS are called hrc genes, which have a two-third similarity at the amino acid level and such genes are called hypersensitive response conserved (Hrc) genes. The specific hrp genes encoding extracellular proteins (e.g. harpins) secreted by the T3SS have only35% amino acid similarity. The hrp genes are usually arranged in clusters of about 20 genes, one of which codes for a constituent of an outer membrane, whereas many others encode for the core secretion machinery, for regulatory genes, for harpins, for the Hrp-pilin, for avirulence (avr) genes (Galán and Collmer, 1999). Although the primary determinants of pathogenicity and virulence in many bacteria are secretary enzymes such aspectin lyases, cellulases and proteases that macerate plant tissues of many species, it is now known that the hrp genes determine the potential secondary pathogenesis (Agrios, 2005).

Many gram-negative plant pathogenic bacteria employ a type III secretion system (TIII-SS) to subvert and colonize their respective host organisms. The TIII-SS injects effector proteins directly into the cytosol of eukaryotic cells and thus allows the manipulation of host cellular activities to the benefit of the pathogen. In plant pathogenic bacteria, TIII-SSs are encoded by hrp (for hypersensitive response and pathogenicity) genes, which are required for bacteria to cause disease in susceptible plants and to elicit the hypersensitive response in resistant plants (Büttner and He 2009). The hypersensitive response is a rapid local cell death at the infection site that restricts bacterial multiplication and is triggered by individual effector proteins in plants carrying a corresponding resistance gene (Dangl and Jones, 2001). hrp genes were found in almost all major gram-negative bacterial plant pathogens (e.g. *Pseudomonas syringae*, *Xanthomonas* spp., *Ralstonia solanacearum*, and *Erwinia* spp.), illustrating a central role of the TIII-SS in mediating diverse plant bacteria interactions (Buttner and Bonas, 2006). Type IV secretion systems (TIVSS) present in a large number of bacterial species, are used to transfer DNA and proteins across the bacterial cell envelope. The *Agrobacterium* VirB/D4 system is a TIVSS used to delivers sDNA molecules (T-strand or T-DNA) and a set of virulence (Vir) proteins into a wide range of host cells. A network of interactions between the 12virB- and virD-encoded components of the *Agrobacterium*VirB/D4 system has been revealed using various biochemical and genetic assays (Li *et al.*, 2005).

Pathogenicity of Bacterial Enzymes That Degrade Cell Walls

Plant cell walls consist of three major polysaccharides: cellulose, hemicelluloses and lignin and pectins in

woody and some other plants. The number of genes encoding cell wall degrading enzymes varies greatly in the different plant pathogenic bacteria. Soft rotting *Erwinia* spp belonging to the *Pectobacterium* genus (e.g. *Pectobacterium carotovorum, Pectobacterium chrysanthemi, Pectobacterium atrosepticum*) produce a wider range of enzymes which able to degrade plant cell wall components than any other plant pathogenic bacteria. The enzymes include pectinases, cellulases, proteases, and xylanases (Preston *et al.*, 2005; Jha *et al.*, 2005).

The pectinases enzymes are play major role pathogenesis and they are responsible for tissue maceration by degrading the pectic substances in the middle lamella and, indirectly, for cell death. Four major types of pectin degrading enzymes are three (pectatelyase (Pel), pectin lyase (Pnl), and pectin methyl esterase (Pme)) with a high (~8.0) pH optimum, and one polygalacturonase, with a pH optimum of ~6. Among these pectinase enzymes, pectatelyases (Pels) are largely involved in the virulence of soft rot Pectobacterium species. All are found in many forms or isoenzymes, each encoded by independent genes. For example, *P. chrysanthemi* has five main Pelisoenzymes, encoded by the pelA, pelB, pelC, pelD and pelE genes, which are arranged in two clusters, pelADE and pelBC. In contrast, *P. carotovorum* produces three major Pels, an intercellular Pel and numerous minor plant induced Pels. Disruption of the gene encoding any one of the enzymes is not enough to stop cell maceration due to the higher number of pectinases involved (Hugouvieux-Cotte-Pattat *et al.*, 1996; Toth *et al.*, 2003).

Bacterial Toxins as Pathogenicity Factor

Toxins play a vital role in pathogenesis and parasitism of plants by several plant pathogenic bacteria. *Pseudomonas syringae*, *P. syringae* pv. *Tomato,* and *P. syringae* pv. *Maculicola*are primarily associated with production of the phytotoxin like coronatine. On the basis of the symptoms they produce in plants, phytotoxins of *Pseudomonas* spp. have been divided into necrosis inducing and chlorosis-inducing phytotoxins (Buonaurio, 2008). *P. syringae*pv. *syringae*, the cause of many diseases and kinds of symptoms in herbaceous and woody plants, generates necrosis-inducing phytotoxins, lipodepsipeptides, which are generally categorized into two groups, such as mycins and peptins (Melotto *et al.*, 2006). Both phytotoxins induce necrosis in plant cells and create pores in plant plasma membranes, thereby promoting trans-membrane ion flux and cell death (Bender *et al.*, 1999). Chlorosis inducing phytotoxins include coronatine formed by *P.syringae* vs. *atropurpurea, glycinea*, *maculicola*, *morsprunorum* and *tomato*, tabtoxin produced by *P.syringae*p vs. *tabaci*, *coronafaciens* and *garcae* and phaseolotoxin secreted by *Pseudomonas savastanoi* pv. *phaseolicola* and *P. syringae* pv. *Actinidiae* (Prasannath, 2013).

Coronatine functions primarily involved in suppressing the induction of defense related genes in host. Coronatine production genes are located in the coronatine gene cluster, which is usually harboured on a plasmid. Coronatine biosynthesis plays an important role in virulence of toxin-producing *P.syringae* strains. It plays important roles in early and late stages of disease development including suppression of stomatal immunity and thus enhancing entry into host tissue; promoting pathogen growth in the apoplast subsequent to entry (Zeng *et al.,* 2010); and enhancing disease symptom development (Brooks *et al.,* 2005). Studies with coronatine-defective mutants elucidated that coronatine synthesis contributes significantly to lesion expansion, development of chlorosis and bacterial multiplication in infected leaves. Coronatine is also believed to induce hypertrophy of storage tissue, thickening of plant cellwalls, and accumulation of protease inhibitors, compression of thylakoids, and inhibition of root elongation and stimulation of ethylene production in a few but not all plant species (Prasannath, 2013).

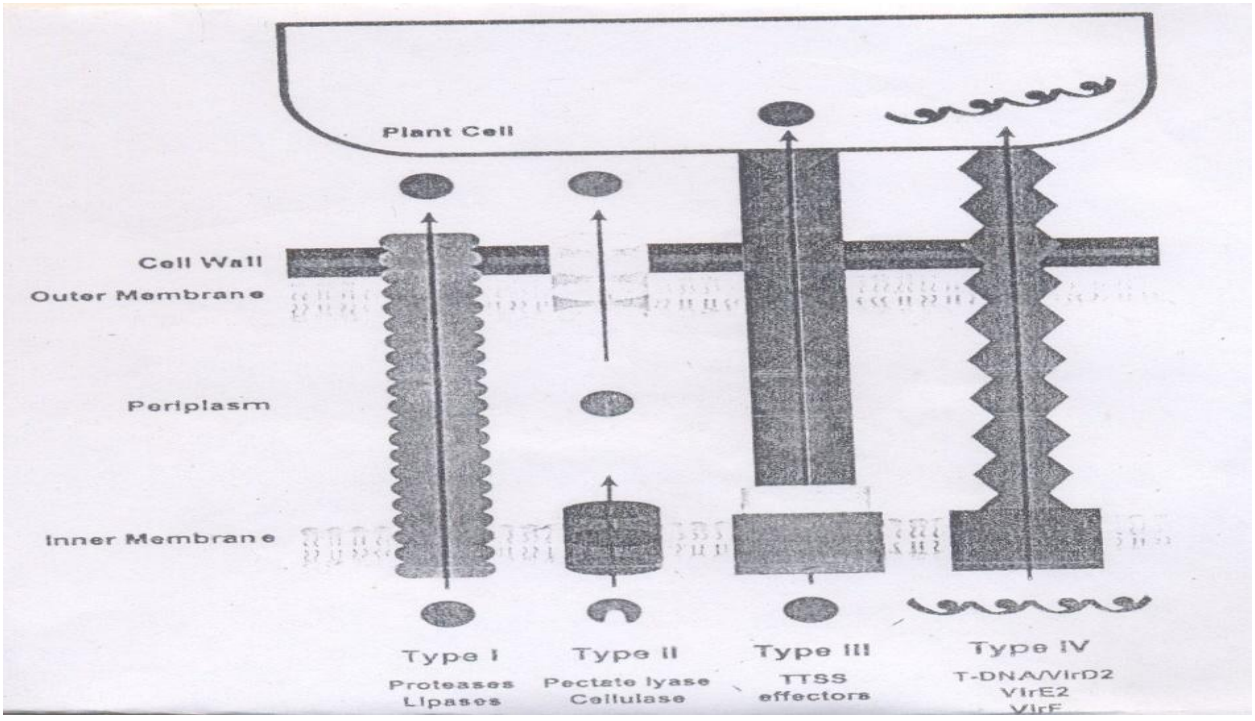
Albicidins, produced by *Xanthomonas albilineans*, block the replication of prokaryotic DNA and the development of plastids, thereby causing chlorosis in emerging leaves. Albicidins interfere with host defense mechanisms and thereby the bacteria gain systemic invasion of the host plant. Three PKS and NRPS genes encoded by XALB1 gene cluster, which is larger than any reported cluster of genes deployed in the production of a bacterial phytotoxin, are involved in albicid in biosynthesis in *X. albilineans* (Royer *et al.*, 2004).

Table.1 Pathogenicity Functions of Effector genes from some Plant Pathogenic Bacteria

	Gene	Pathogenicity function
Erwinia amylovora	dspEF, hrp and hrc genes	The genes are involved in causing the fire blight symptom in pear and apple.
Pseudomonas syringaepv. maculicola *P. syringae pv. phaseolicola*	avr RMP1 avrPphF, hrp and hrc genes	The genes are involved in causing water soaked lesions on leaves Expression and bacterial multiplication. Expression and bacterial multiplication in bean and soybean.
P. syringae pv. tomato	avrA, avrE avrPto, avrRpt2,hrp and hrc genes	Symptom expression and bacterial multiplication. Aggressiveness and bacterial multiplication in tomato.
Xanthomonas axonopodis pv.citri	pthA, hrp and hrc genes	Intercellular growth and induction of cankers in citrus.
X. campestris pv.malvacearum.	avrb6, pthN, hrp and hrc genes	Water soaked lesions on leaves Expression in cotton
X. oryzae pv. oryzae	avrxa5 avrxa7 hrpand hrcgenes	Lesion length and bacterial multiplication in rice. Aggressiveness, lesion length and bacterial multiplication in rice.

Table adopted from Klaas *et al.*, 2002

Fig.1 Roles of TTSS Effectors in Pathogenicity and Resistance



Extracellular Polysaccharides as Pathogenicity Factors

Many plant pathogens produce large amounts of Extracellular Polysaccharides (EPSs). EPSs are carbohydrate polymers that are secreted by bacteria and form either a closely attached capsule layer surrounding the bacterial cell wall or a loosely associated extracellular slime. EPS play a significant role in pathogenesis of many bacteria by both direct interference with host cells and by providing resistance to oxidative stress. The virulence of several pathogenic bacteria, including *R. solanacearum, E. amylovora, X. campestris*, and *P. syringae*, is associated with their ability to produce various EPS polymers during growth in plant tissue. EPSs are believed to provide a selective advantage to pathogenic bacteria through multiple functions including (1) facilitating absorption of water, minerals and nutrients; (2) providing protection from abiotic stresses encountered during epiphytic or saprophytic growth, as well as from toxic molecules encountered during growth in plant tissue; (3) promoting colonization and spread within host tissue; and (4) contributing to the production of disease symptoms such as water soaking and wilting. EPS1 is a polymer made of a trimeric repeat unit consisting of Nacetylgalactosamine, deoxyl- galacturonic acid and trideoxy-d-glucose. At least 12 genes are involved in EPS1 synthesis, where it is produced by the bacterium in huge quantity and constitutes more than 90% of the total polysaccharides (Denny 1995; Melotto and Kunkel, 2013)

Pathogenicity of phytohormones

Biosynthesis of the phytohormones such as auxins (e.g.indole-3-acetic acid-IAA) and cytokinins are major virulence factors for the gall-forming plant pathogenic bacteria. The genes involved in the production of IAA in these bacteria are iaaM and iaaH genes, which are exclusively located on a plasmid in *P. agglomerans pv. gypsophilae*, while in the chromosome or on a plasmid in *P. savastanoi*. Production of cytokinins in gall-forming pathogenic bacteria is similar to those occurringin higher plants, with isopentenyl transferase as chief biosynthesis enzyme. Mutations of the IAA orcytokinin synthesis genes stimulated reductions in thevirulence of *P. agglomeranspv. gypsophilae* and *P. savastanoi*, while mutations in hrp/hrc gene cluster annulled their pathogenicity (Barash and Manulis, 2005). Ethylene, the gaseous phytohormone formed by several microbes including plant pathogenic bacteria can also be considered a virulence factor for some of them. *P. savastanoipv. phaseolicola* and *P.syringaepv. glycinea* are very capable in ethylene production (Robert-Seilaniantz *et al.*, 2011).

Other Bacterial Factors Related to Pathogenicity

Several other components of the bacterial cell or released by the bacteria appear to play roles as pathogenicity determinants. Lipopolysaccharide (LPS) components of the outer cell wall of gram-negative bacteria play a role in the pathogenicity in Erwinias (Pérombelon, 2002). Evidence of this is given by the activation of pathogenesis-related proteins, such as glucanases in diseased plants and the fact that disruption of the LPS gene in the bacteria lessens their virulence and that protein–LPS complexes from bacteria hinder the HR. Catechol and hydroxamate siderophores appear to be virulence determinants for Erwinias. In the fire blight bacterium *E. amylovora*, its siderophore protects the bacteria by interacting with H_2O_2 and inhibiting the generation of toxic oxygen species. The peptide methionine sulfoxide reductase, which protects and repairs bacterial proteins against active oxygen damage, is essential for the expression of full virulence of the bacteria (Eastgate, 2000).

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death, which leads to HR; as a result, the infection by the bacteria in incompatible interactions fails. Avr proteins seem to also play a role in compatible host/bacteria interactions. *avr*genes usually determine host specificity at the pathovar and the species level.

Infection and disease factors will be synthesized by three groups of genes in pathogens:1) symbiosis genes, which codify factors that promote the process of infection from the first contact of the micro-organism with the plant to the full colonization of the host, passing through the intermediate stages of invasion and of finding a source of nutrients;2) true virulence genes, which codify factors (toxins, hormones, EPSs, degradative enzymes, and other compound) that interact with the host, and that damage the host directly in the process of infection; 3) virulence-associated genes, which codify those factors that are involved in the deployment (regulation, secretion, processing) of the products of the true virulence genes.

Pathogenic bacteria utilize a number of mechanisms to cause diseases in plant hosts. In order to develop effective strategies to protect crops from diseases, it is necessary to understand the molecular basis of the plant-bacterial interactions that result in disease or resistance. Past research emphasis, especially at a molecular level was engaged on detecting pathogen genes and proteins responsible for their pathogenesis. This review has described the genes and factors, possessed or released by bacteria for eliciting pathogenicity and virulence, causing diseases in host plants. Thus, the knowledge on pathogenic genes and virulence factors of bacteria can definitely be beneficial for the development of new control strategies. This paper, with its limits and its peculiarities, is not presented with the ambition to rewrite Plant Pathology dogma, or to discuss the latest details of the mechanisms of the interactions between plants and their microbial enemies.

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