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Review Article

Pathogenicity and Virulence Factors of Phytobacteria

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Abstract: Plant pathogenic bacteria have evolved specialized strategies to exploit their respective hosts. Most of them are 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. An exclusive infection tactic is provoked by *Agrobacterium tumefaciens*, which genetically transfers its T-DNA from its Ti plasmid to host plant cell via T-pilus belonging to the type IV secretion apparatus. Other key virulence factors of phytopathogenic bacteria are plant cell wall degrading enzymes, phytotoxins, extracellular polysaccharides and phytohormones, which are central for the pathogenesis of necrotrophic bacteria. In general, plant pathogenic bacterial strains mutated in any virulence factor comparatively reduce their virulence, while their pathogenicity remains unchanged. This review summarizes the facts of how bacteria manipulate plant physiology to infect their hosts.
Keywords: Effector proteins, *hrp* genes, Type III secretion system, Virulence factors

INTRODUCTION

Bacterial plant diseases are most severe in tropical and subtropical countries, where bacteria receive ideal climatic conditions for their growth resulting in more crop yield losses in these countries. Leaf and fruit spots, blights, cankers, vascular wilts, rots and tumors are characteristic symptoms of bacterial diseases. Most of the bacterial pathogens of plants are Gram-negative bacteria in the genera *Pseudomonas*, *Erwinia*, *Ralstonia* and *Xanthomonas* and certain Gram-positive bacteria (*Streptomyces* spp. and *Clavibacter* spp.) also cause diseases in plants. According to Buonaurio [1], about 150 species among the 7100 classified bacterial species are expected to cause diseases in plants.

Pathogenic bacteria incite diseases in plants by penetrating into host tissues through natural openings, such as hydathodes, stomata, lenticels, stigma, nectarthodes or through wounds and bacteria are directly deposited by insect vectors [2]. Commonly phytopathogenic bacteria colonize the apoplast (intracellular space of plants) and from this location outside the walls of plant cells they provoke a range of diseases in most economical plants [3]. Besides the endophytic nature, some bacterial species also have the epiphytic habitat on plant surfaces (rhizoplane, phylloplane, carpoplane, etc.). Once inside plant tissues, various species can inhabit the dead xylem vessels or live in phloem sieve elements; however, most of pathogenic bacteria are limited to intercelleular space, i.e. apopalst [4, 5]. This view may be changed as the apoplast is considered to be a nutrient-lacking niche for most microbes, containing certain antimicrobial compounds [6- 8]. In addition to this, plant defenses that are triggered upon microbial infection are often targeted the intercellular space [9-11].

Bacterial pathogens may involve in two main attack strategies to extract the host nutrients such as biotrophy, in which the host plant cells are alive as long as possible and bacteria exploit nutrients from living cells and necrotrophy, in which plant cells are killed by bacteria and from dead cells, nutrients are extracted [1]. Bacterial interactions with plants can either be compatible or incompatible. Compatible interactions happen when the bacterium encounters susceptible host plants causing disease symptoms [12]. Incompatible interactions occur 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.

In this review, I concentrate the pathogenicity and virulence factors used by phytopathogenic bacteria (mainly Gram-negative) causing diseases in plants.

PATHOGENICITY AND VIRULENCE OF BACTERIA

In order to avoid confusion in terminology, it is necessary to define the terms virulence and pathogenicity, which are often erroneously considered synonyms. Shurtleff and Averre [13] defined that pathogenicity is the ability of a pathogen to cause disease, whereas virulence is the degree of pathogenicity of a given pathogen. As the plant bacterial pathogens are extracellular, they deploy a delivery of secreted virulence factors to interfere with

host cell processes from outside plant cells. These include production of protein virulence factors (effectors), which are directly injected into host plant cell cytoplasm via a specialized type III secretion path [14-16], secretion of low molecular weight phytotoxins which are produced into apoplast [17], production of exopolysaccharides [18] and cell wall degrading enzymes [19, 20]. Bacteria evade, overcome or suppress antimicrobial plant defenses using these virulence factors, which elicit release of water and nutrients from host cells to colonize in the apoplast successfully.

Pathogenecity islands

It is worthy to note that proteins, which contribute to the host-pathogen interactions, are often encoded by pathogenicity islands (PAI) [21]. In addition to effector proteins genes, PAI in plant pathogenic bacteria carry genes for phytotoxin production and the type III protein secretion gene cluster. PAI are defined as regions of DNA that contain virulence genes and are found in the genome of pathogens, but absent from or only rarely present in non-pathogenic variants of the same or related strains [22]. Other characters include a variable G+C content, distinct boundaries from the rest of the genome and the presence of genes related to mobile elements such as transposases, integrases and insertion sequences. Hacker and Kaper [23] produced an extensive list of PAI from the genomes of different human, animal and plant pathogens.

PAI containing type III protein secretion gene cluster

Type III secretion system (T3SS) is utilized by majority of Gram-negative plant bacteria to promote pathogenesis except for *Xylella fastidiosa* and *Agrobacterium* spp. [19]. T3SS can produce several proteins, many of which affect the interaction with the plant. Several proteins are transmitted from the bacterial cell into the plant cell via this secretion system [24, 25]. If a bacterial gene or a secreted protein stimulates a defense reaction in the plant that leads to a resistance response termed the hypersensitive reaction, then it is termed an avirulence gene/protein [26]. Alternatively, they are designated virulence genes/proteins if they trigger the development of disease symptoms in the plant. T3SS is encoded by a distinct cluster of genes, termed *hrp/hrc* (hypersensitive response and pathogenicity/conserved) genes in the plant pathogens [27]. Gene clusters encoding T3SS are found in many Gram-negative bacterial pathogens of humans, animals, plants and plant growth-promoting rhizobacteria [28-31]. Type III gene clusters are also present in *Ralstonia*, *Erwinia*, *Xanthomonas* and *Pantoea* plant pathogens. Sometimes the T3SS genes are located on plasmids, for examples, the wilt pathogen *Ralstonia solanacearum* (the 2.09 Mb megaplasmid) [32] and the gall pathogen *Erwinia herbicola* (the 150 kb pPATH plasmid) [33]. It revealed that the T3SS gene cluster in *R. solanacearum* is not a PAI origin [34].

PAI containing effector genes

A number of effector genes on PAI, which are often associated with mobile elements, are not linked to the T3SS gene cluster [35]. For instance, in *Pseudomanas syringae* pv. *phaseolicola*, the gene *virPphA* is essential for virulence towards the host bean plant and also functions in *P. syringae* pv. *phaseolicola* as an avirulence gene towards soybean, inducing a rapid cultivar-specific HR [36]. A DNA region of 30 kb from the *P. syringae* pv. *phaseolicola* plasmid could be used to restore virulence. Sequencing of this 30 kb region revealed that effector genes *avrD*, *avrPphC* and *avrPphF*, as well as *virPphA* were present and a number of genes and sequences that share homology to insertion sequences and transposons, indicating mobility were also found [22].

PAI containing phytotoxin genes

Phytotoxins are biosynthetic products of plant pathogens that contribute to disease development. Several *P. syringae* toxins have been identified and been demonstrated that although the phytotoxins are not required for basic pathogenicity towards the host plant, they generally function as virulence factors for this pathogen, leading to increased severity of disease symptoms. *P. syringae* pv. *syringae* forms two classes of necrosis inducing lipopeptide phytotoxins, syringomycins and syringopeptins. Other three phytotoxins produced by *P. syringae*, coronatine, tabtoxin and phaseolotoxin, all usually induce chlorosis [17]. The primary symptom of coronatine is a diffuse chlorosis that is induced on a wide range of plant species and contributes to the virulence of several pathovars of *P. syringae* [37]. Tabtoxin is formed by different isolates of *P. syringae* [38] and is the precursor for the biologically active tabtoxinine- β-lactam. Phaseolotoxin acts as a virulence factor causing chlorosis by inhibiting the enzymatic activity of ornithine carbamoyl transferase in host plants [17].

Pathogenicity genes in plant pathogenic bacteria

Adhesion of bacteria to plant surfaces

Majority of bacteria do not require adhesion mechanisms except when they are moving through the xylem and phloem [39]. However, the crown gall bacterium *Agrobacterium* requires attachment to plant surface as the first step in the transport of T-DNA and to develop 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 bacteria genome that contains several genes for adhesion [40]. Additionally, *Agrobacterium* also has more other genes with homology to genes of mammalian pathogens for adhesions and for pilus biosynthesis [4]).

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 [42].

Secretion systems of bacteria

Plant pathogenic bacteria use a number of secretion systems to deliver effector proteins, either directly into the host cells or into the intercellular spaces. Five forms of secretion pathways are recognized on the basis of the proteins that form them [43]. Type I and II pathways secrete proteins to the host intercellular spaces, whereas type III and IV systems can deliver proteins or nucleic acids directly into the host plant cell [44]. Type I secretion system (T1SS) has the simplest structure and it allows direct secretion of effectors from the bacterial cytosol to the outer environment. T1SS is found in almost all phytopathogenic bacteria and involved in the secretion of toxins such as cyclolysin, hemolysins and rhizobiocin. They contain ATP-binding cassette proteins and carry out the export and import of several compounds using energy produced by the hydrolysis of ATP [45]. Proteases and lipases from the soft rot pathogenic bacteria *Erwinia chrysanthemi* are examples of plant pathogen effectors secreted via the T1SS [46, 47].

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. T2SS is more complex in secretion structure and 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 [48]. Pathogen effectors involved in host cell wall degradation, such as pectate lyase, polygalacturonase and cellulase from *Erwinia* and *Xanthomonas* species, are produced by the T2SS (49, 20). *Xanthomonas* and *Ralstonia*, which have two T2SS 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 and actually, *Agrobacterium* has the genes for only the first step of protein transfer across the inner membrane and for the rest using type IV secretion system (T4SS) [50].

The pathogenicity of several biotrophic Gram-negative bacteria in the genera *Xanthomonas*, *Pseudomonas*, *Ralstonia*, *Erwinia* and *Pantoea* is mainly due to their capability to produce a T3SS, also called injectisome [43], by which the bacteria inject proteins (T3SS effectors) involved in their virulence into plant cells. The primary function of T3SS is the transportation of effector molecules across the bacterial membrane and into the plant cell. The genes that encode protein components of the T3SS are called *hrc g*enes, which have a two-third similarity at the amino acid level. The specific *hrp* genes encoding extracellular proteins (e.g. harpins) secreted by the T3SS have only 35% amino acid similarity [51, 52]. 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 and so on [52]. Although the primary determinants of pathogenicity and virulence in many bacteria are secreted enzymes such as pectin lyases, cellulases and proteases that macerate plant tissues of many species, it is now known that the *hrp* genes determine the potential secondary pathogenesis [4].

The characteristic feature of the T3SS structure, a needle-like protruding structure with a channel along which proteins travel, resembles to bacterial flagella [53], both at structural and functional level. The injectisome consists of two parts, an envelope-embedded multi-ring base and a long protruding surface appendage, called the hrp pilus [54]. Hrp pili, described for *P. syringae* [15, 55], *R. solanacearum* [32, 56], *Erwinia amylovora* [57, 58] and *Xanthomonas campestris* pv. *vesicatoria* [59, 60], elongate distally with the addition of their major component, Hrp pilin subunits, whereas T3SS effectors are secreted from the hrp pilus tip [61]. This proves that Hrp pili function as conduits through which substrates are transported [62]. Having considered the dimension of the pilus, we have to assume that the effector proteins, which are up to 200 kDa in size, travel within the channel in an at least partially unfolded form [15]. Stebbins and Galan [63] have shown that most T3SS effector molecules are dependent on chaperones, which keep the effectors in a partially unfolded state in the bacterial cytosol. Even though the pilus proteins, HrpA (*P. syringae* and *E. amylovora*), HrpY (*R. solanacearum*) and HprE (*X. campestris* pv. *vesicatoria*) do not share any significant homologous sequence, they exhibit a number of physico-chemical features in common [64].

T4SS transfers macromolecules from the bacterium to the host plant cell. T4SS pathway is best known from studies on *Agrobacterium tumefaciens*, which is a Gram-negative soil bacterium. This pathway is the only secretion system of both proteins and nucleic acids. There is the evidence that the VirD2/T-DNA nucleoprotein complex is injected through the type IV pilus from *A. tumefaciens* directly into the plant cell [65]. T4SS is composed of proteins encoded by the *virB* oper*on* and the *virD4* gene [66, 67]. The *A. tumefaciens virB* operon encodes 11 proteins that make an organized structure and are involved in the transport of the T-

DNA strand from the bacterium to the plant cell cytoplasm. The *virB* gene products, termed the mating pair formation (Mpf) proteins, elaborate a cell envelope-spanning structure required for substrate transfer [66], as well as an extracellular appendage termed the T-pilus that mediates attachment to recipient plant cells [68]. VirD4 is a coupling protein, which does not carry out T-DNA processing or biogenesis of the T-pilus but acts together with the Mpf structure to deliver substrates across the cell envelope [69, 70]. Type V secretion autotransporter is found in *Xylella* and *Xanthomonas* and consists of genes that encode surface-associated adhesins [71].

Pathogenicity of plant cell wall degrading enzymes

Plant cell walls consist of three major polysaccharides such as cellulose, hemicellulose and pectins and in woody and some other plants, lignin. The number of genes coding cell wall degrading enzymes varies to a greater extent in a variety of phytopathogenic bacteria. Soft rot causing erwinias now belonging to the *Pectobacterium* genus (e.g. *Pectobacterium carotovorum*, *Pectobacterium chrysanthemi*, *Pectobacterium atrosepticum*) produce a wider spectrum of enzymes able to deconstruct plant cell wall compounds than any other plant pathogenic bacteria [72]. The enzymes include pectinases, proteases, cellulases and xylanases. Proteases are secreted by the T1SS, whereas the rest of the above said enzymes by the T2SS [73].

Pectinases are expected to be most important in pathogenesis, because they are responsible for tissue maceration by degenerating the pectic substances in the middle lamella and eventually, for cell death. Four major types of pectin degrading enzymes are secreted *viz.* pectate lyase, pectin lyase, pectin methyl esterase and polygalacturonase. Among these pectinase enzymes, pectate lyases (Pels) are largely involved in the virulence of soft rot *Pectobacterium* species [74]. All are found in many forms or isoenzymes, each encoded by independent genes. For example, *P. chrysanthemi* has five main Pel isoenzymes, encoded by the *pelA*, *pelB*, *pelC*, *pelD* and *pelE* genes, which are arranged in two clusters, *pelADE* and *pelBC* [75]. In contrast, *P. carotovorum* produces three major Pels, an intercellular Pel and numerous minor plant induced Pels [74]. 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.

The expression of pectinase genes is triggered by the fragmented products of pectin and pectate, particularly 2-keto-3-deoxygluconate, 5-keto-4-deoxyuronate and 2,5-diketo-3-deoxygluconate [76]. Bauer *et al.* [77] demonstrated that *hrp* cluster is also present in the genome of *P. chrysanthemi* and the mutations in this cluster provoke a slight decline in virulence in susceptible hosts. However, mutations in individual major *pel* genes do not bring about any significant difference in *P. chrysanthemi* virulence [78, 79].

Cell wall degrading enzymes are believed to play a role in pathogenesis by facilitating penetration and tissue colonization, but they are also virulence determinants responsible for development of symptom once growth of the bacteria has been started. A few Xanthomonads, e.g., *X. campestris* pv. *campestris*, the causal agent of black rot of crucifers, have genes for two pectin esterases and polygalacturonases, four pectate lyases, five xylanases and nine cellulases [48]. *Xanthomonas citri* has no pectin esterases, one less pectate lyase and three fewer cellulases [80]. Since pectin esterases are essential in tissue maceration, their presence in the crucifer rot bacterium and absence in the citrus canker bacterium may explain the symptoms of the two diseases. Other deprived pectinolytic bacteria include *A. tumefaciens*, which has only four genes encoding pectinases of any form and *Xylella*, which has only one gene coding for a polygalacturonase [81].

Pathogenicity of bacterial toxins

Toxins play a vital role in pathogenesis and parasitism of plants by several plant pathogenic bacteria. *Pseudomonas* spp. secret a wide range of non-host specific phytotoxins [17]. On the basis of the symptoms they produce in plants, phytotoxins of *Pseudomonas* spp. have been divided into necrosis-inducing and chlorosis-inducing phytotoxins [1].

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 [82]. Both phytotoxins induce necrosis in plant cells and create pores in plant plasma membranes, thereby promoting transmembrane ion flux and cell death [17]. Chlorosis-inducing phytotoxins include coronatine formed by *P. syringae* pvs. *atropurpurea*, *glycinea*, *maculicola*, *morsprunorum* and *tomato*, tabtoxin produced by *P. syringae* pvs. *tabaci*, *coronafaciens* and *garcae* and phaseolotoxin secreted by *Pseudomonas savastanoi* pv. *phaseolicola* and *P. syringae* pv. *actinidiae* [83].

Coronatine production genes are located in the coronatine gene cluster, which is usually harboured on a plasmid [37]. Coronatine biosynthesis plays an important role in virulence of toxin-producing *P. syringae* strains. Studies with coronatine-defective mutants elucidated that coronatine synthesis contributes significantly to lesion expansion, development of chlorosis and bacterial multiplication in infected leaves [84, 85]. Coronatine is also believed to induce hypertrophy of storage tissue, thickening of plant cell walls, accumulation of protease inhibitors, compression of thylakoids, inhibition of root elongation and stimulation of ethylene production in a few but not all plant species [86]. A recent study revealed that stomatal

closure in Arabidopsis is part of a plant innate immune response, which checks bacteria from entering the plant leaf and that coronatine suppresses this defense mechanism by re-opening stomata [82].

Albicidins produced by *Xanthomonas albilineans* cause chlorosis in emerging leaves and interfere with host defense mechanisms and thereby the bacteria gain systemic invasion of the host plant. Mutants deficient in albicidin production failed to cause chlorosis in inoculated sugarcane [87]. 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 albicidin biosynthesis in *X. albilineans* [88].

Pathogenicity of extracellular polysaccharides

Extracellular polysaccharides (EPS) may be connected to the bacterial cell as a capsule, be produced as fluidal slime, or be present in both forms [89]. EPS play a significant role in pathogenesis of many bacteria by both direct interference with host cells and by providing resistance to oxidative stress. EPS1 is the chief virulence factor of the bacterial wilt disease caused by *R. solanacearum* in solanaceous crops, since *eps* mutants were severely reduced in systemic colonization of tomato plants when introduced through unwounded roots and did not produce typical wilt symptoms even when directly inoculated into stem wounds [90]. EPS1 is a polymer made of a trimeric repeat unit consisting of *N*acetyl galactosamine, deoxy-l-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 [89]. EPS incite wilt by clogging the xylem vessels and by causing them to burst from the high osmotic pressure. Besides their involvement in wilting, EPS might play further roles such as safeguard bacteria from toxic plant materials, reduce contact with plant cells to lessen host defense responses, promote multiplication by prolonging water-soaking of tissues, or else help invasion or systemic colonization [18].

Oh and Beer [91] demonstrated that EPS, amylovoran and levan are pathogenicity and virulence factors, respectively, of *E. amylovora*, the pathogen causing fire blight in some rosaceous plants. Amylovoran is an acidic heteropolysaccharide, whereas levan is a neutral homopolysaccharide [92]. Amylovoran affects host plants principally by blocking the vascular tissues, thereby inducing wilt of shoots and is known a pathogenicity factor, as amylovoran-deficient mutants are not pathogenic [93]. The production of amylovoran needs the *ams* operon, consisting of 12 genes. Levan is synthesized by levansucrase, coded by the *lsc* gene, which mutation resulted in a slow symptom development on shoots of host plants [94].

Xanthan, the major exopolysaccharide secreted by *Xanthomonas* spp., plays a key role in *X. campestris* pv. *campestris* pathogenesis [95]. They observed that a xanthan deficient mutant and a mutant producing truncated xanthan failed to cause disease in both *Arabidopsis* and *Nicotiana benthamiana* plants. They also demonstrated that xanthan suppressed callose deposition in plant cell wall, a primary form of resistance to bacterial invasion.

Pathogenicity of phytohormones

Biosynthesis of the phytohormones, auxins (e.g. indole-3-acetic acid-IAA) and cytokinins are major virulence factors for the gall-forming plant pathogenic bacteria, *Pantoea agglomerans* pv. *gypsophilae*, the causative agent of crown and root gall disease of *Gypsophila paniculata* and *P. savastanoi* pvs. *savastanoi* and *nerii*, which incite olive and oleander knot diseases, respectively [96]. 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* [97].

Production of cytokinins in gall-forming phytopathogenic bacteria is similar to those occurring in higher plants, with isopentenyl transferase as chief biosynthesis enzyme [98]. Mutations of the IAA or cytokinin synthesis genes stimulated reductions in the virulence of *P. agglomerans* pv. *gypsophilae* and *P. savastanoi* [99], while mutations in *hrp/hrc* gene cluster annulled their pathogenicity [100, 101].

Ethylene, the gaseous phytohormone formed by several microbes including plant pathogenic bacteria [102], can also be considered a virulence factor for some of them. *P. savastanoi* pv. *phaseolicola* and *P. syringae* pv. *glycinea* are very capable in ethylene production. Weingart *et al*. [103] showed an interesting point that when bean and soybean plants were inoculated with the ethylene-negative mutants of these bacterial species, only *P. syringae* pv. *glycinea* reduced its virulence significantly.

Other factors related to bacterial pathogenicity

Several other compounds of pathogenic bacteria or released by the bacteria seem to play role as pathogenicity determinants. Lipopolysaccharide (LPS) components of the outer cell wall of Gram-negative bacteria result in the pathogenicity of erwinias [104]. 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 [105]. Catechol and hydroxamate siderophores also appear to be virulence factors for erwinias [106]. In the fireblight bacterium *E. amylovora*, its siderophores save the bacteria by interacting with H_2O_2 and inhibiting the formation of toxic oxygen species [58]. The peptide

methionine sulfoxide reductase, which defends and repairs bacterial proteins against active oxygen damage, is important for the expression of full virulence of the *E. chrysanthemi* [107].

Bacterial virulence by avr genes

avr genes in bacteria are expected to encode or to direct the synthesis of molecules that are recognized by the host plants and bring out the rapid induction of defense responses on resistant host plants [108]. However, their prevalence among pathogens suggests that they may offer some benefits to the pathogens in addition to warning host plants that they are about to be attacked. In many plant-bacteria combinations, it has been demonstrated that the proteins (Avr proteins) encoded by *avr* genes, encourage growth of pathogens and development of diseases in susceptible hosts. Avr proteins can interfere with the resistance mediated by the *avr* genes. Since the Avr proteins are encoded by the *avr* genes, it is obvious that *avr* genes can alter the signaling of host defense systems in resistant host plants [109]. In the absence of a resistance R gene, the particular *avr* gene acts as a virulence factor that not only upholds the growth of the particular bacterium in several host plants, including some that show different degrees of resistance, but transgenic plants that express the *avr* gene actually exhibit increased susceptibility to the pathogen and/or aggressiveness of the pathogen [12, 110]. However, different *avr* genes, even of the same bacterium, contribute varying degrees of susceptibility/aggressiveness to bacteria that harbour these genes [111]. It reveals that the particular Avr protein functions inside the host plant cell and enhances bacterial virulence.

CONCLUSION

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.

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