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Pathogenicity Events in Plant Pathogenic Bacteria: A brief note

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ABSTRACT

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. Plant pathogenic bacteria have evolved specialized strategies includes quorum sensing, type secretions, to exploit their respective hosts. Quorum sensing (QS) allows bacteria to assess their local population density and/or physical confinement via the secretion and detection of small, diffusible signal molecules. Five forms of secretion pathways are recognized on the basis of the proteins that form them.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.

Key words: Quorum Sensing, Type Secretions, Local Population density, Diffusible signal molecules, Secretion Pathways

INTRODUCTION

Pathogenic bacteria incites 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 (Buonaurio 2008; Melotto and Kunkel 2013). 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. 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 intercellular space, i.e. apoplast. Plant pathogenic bacteria have evolved specialized strategies includes quorum sensing, type secretions, to exploit their respective hosts. Most of them are Gram-negative, of which biotrophic pathogenic bacteria fundamentally possess a type III secretion system encoded by *hrp* genes encoding *Avr* effector proteins that delivered into host plant cells to suppress plant defense responses (Daniela et al. 2009). *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 (Meng 2013), phytotoxins (Zheng et al. 2012), effectors (Block and Alfano 2011; Lindeberg 2012) extracellular polysaccharides (Yuki et al. 2013) and phytohormones, which are central for the pathogenesis of necrotrophic bacteria.

BACTERIAL-BACTERIAL COMMUNICATION -QUORUM SENSING

Quorum sensing (QS) allows bacteria to assess their local population density and/or physical confinement via the secretion and detection of small, diffusible signal molecules. The term quorum sensing (QS) describes a well-understood mechanism of bacterial cell-cell communication and conveys the concept that certain traits are only expressed when bacteria are crowded together (Fuqua 1994). This allows them to act in a coordinated manner and reinforces the notion that individual bacteria benefit from co-operative group behavior to survive, compete, and persist in nature or to colonize a particular host. QS involves the exchange of low molecular weight, diffusible signal molecules between members of a localized population. If signal production by the population is greater than its loss by diffusion or inactivation, the signal accumulates to a threshold level and activates cognate receptor proteins. These in turn may trigger widespread changes in gene expression in members of the population. A key requirement for quorum sensing is, therefore, growth of cells in close proximity, as in a biofilm or when confined in an enclosed, diffusion-limited environment. In Gram-negative systems the bacteria produce autoinducers, which are diffusible signal molecules that can easily pass in and out through bacterial membranes. At high cell density, these reach a threshold level within the external environment that is detected by the bacteria and this result in the regulation of gene expression.

In gram-positive bacteria, there is an involvement of modified oligopeptides secreted via ABC transport mechanisms and detected by two-component histidine kinase signal transduction systems.Bacteria are dynamic creatures that are able to regulate their metabolism and lifestyle in response to a variety of environmental cues. These cues include changes in their chemical, physical, and biological surroundings. In recent decades, microbiologists have come to appreciate that bacteria are even able to recognize changes in their own population density. Cell density-dependent regulation has been termed "quorum sensing."

Mechanism of quorum-sensing

A model of the quorum-sensing (Fig. 1) control of gene regulation is the luminescence (lux) operon in *Vibrio fischeri*. In addition to the luciferase genes required for light production, this operon encodes Lux R, an acyl-homoserine lactone (acyl-HSL)-dependent transcriptional activator, and Lux I, an acyl-HSL synthase that catalyzes the production of 3-oxohexanoyl-homoserine lactone (3OC6HSL). Each bacterium expresses the Lux proteins at low basal levels throughout its entire lifecycle. At low cell densities, the small amounts of the amphipathic 3OC6HSL signal that are produced diffuse away from the cells. However, as a local population increases in density, 3OC6HSL concentrations increase. This results in a shift of the Lux R equilibrium towards its 3OC6HSL-bound, active state. Acyl-HSL binding leads to dimerization of Lux R and binding to the lux box, a 20-base pair inverted repeat located in the Plux promoter. There the acyl-HSL-bound LuxR dimer activates expression of the lux genes after the recruitment of RNA polymerase.

In gram negative bacteria, acyl-homoserine lactone type molecules serve as the main signalling molecules while lipid, peptide, and amino acid based signalling molecules infrequently serve as signalling molecules. Furthermore, in gram-negative bacteria, there is one well conserved mechanism for controlling quorum response. Gram-positive bacteria, on the other hand, use peptides or modified peptides as the primary means of signaling; and also differing from gram-negative bacteria, there are several different mechanisms found within the class which are used to gain quorum responses.

In gram-positive bacteria, a two component signal transduction is the main quorum mechanism. In the two component regulatory system, a cell-density dependent peptide signal is encoded by a locus and is secreted into the surrounding environment for other bacteria to sense. The peptide signal works by binding to a sensor protein, histidine kinase, located in the cell membrane of the bacterium. The activation of the histidine kinase leads to phosphorylation of response-regulating protein, and interaction with another regulatory protein facilitates transcriptional activation. After the transcription of RNA III, RNA III affects the transcription or translation of the target gene (Dunny 1997).

The second main quorum mechanism in gram-positive bacteria is termed internalization. With internalization, the pheromone induction involves the transport of signal molecules into the responder cell to interact with intracellular effectors. This differs from the two - component signal transduction which involves the signal molecule interacting with the HK sensor protein to produce a transmembrane signal. The internalized pheromone interacts with the ribosomes of the ribonucleoprotein complex which results in an increase in translation and modified ribosomes which translate the message for the target gene. The target gene then results in the quorum response such as the Aggregation Substance which is a surface adhesion and conjugation function in *Enterococcus faecalis* (Dunny 1997).

In gram negative bacteria, autoinduction is the sensing system that works by the production of diffusible compounds called autoinducers or signaling molecules (Fig. 2). The autoinducers accumulate in the surrounding environment and in the presence of a large population of cells (10^{10} to 10^{11} cells), the concentration accumulates to a level needed for transcriptional activation. In most cases, the concentration needed for activation of

transcription is approximately 10 nM. The cell membrane is permeable to the autoinducers so at high enough

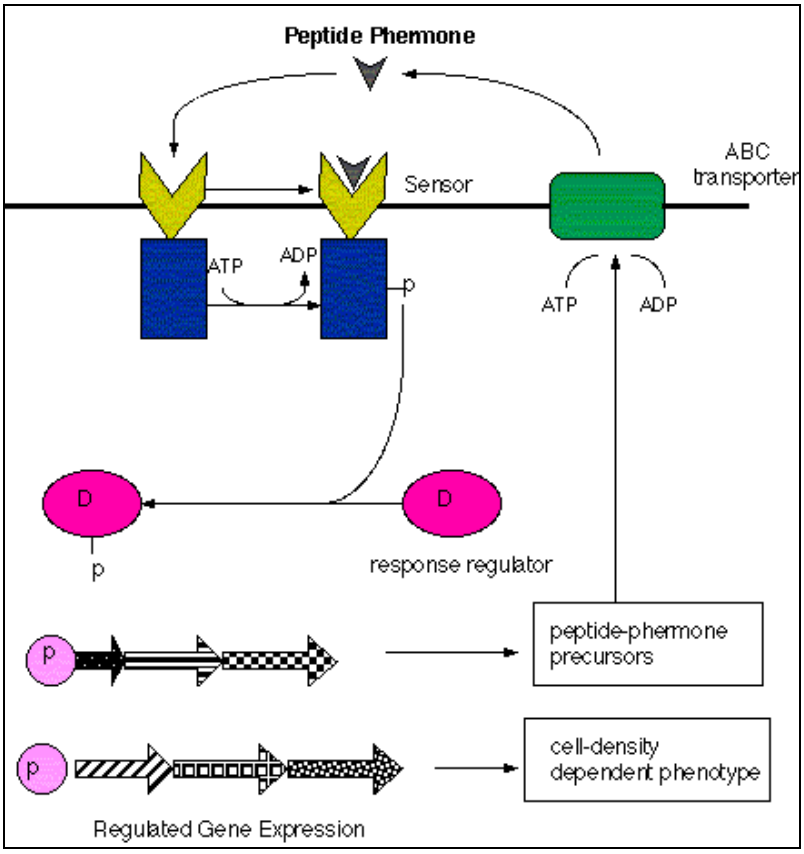

Fig.1. Two Component signal transduction in Gram Positive Bacteria.

concentrations, the autoinducer diffuses into the cell where it interacts with cell density dependent transcriptional activators, also termed response regulators. This results in the induction of the quorum response and the positive regulation of an autoinduce synthetase by a signal generator which will provide more autoinducer for response. Autoinduction is the sensing system which is used by *Vibrio fisheri* and *Vibrio Harvey*. Sensing systems with very similar regulatory mechanisms are found in conjugal transfer of *Agrobacterium tumefaciens* Ti plasmid, autoinduction in *Erwinia carotovora*, regulation of rhizosphere genes in *Rhizobium leguminosarum*, and cell division in *Escherichia coli*. The quorum mechanisms are basically the same for these organisms; what differs is the resulting phenotype from the quorum response and the components of the mechanism(Fig 3).

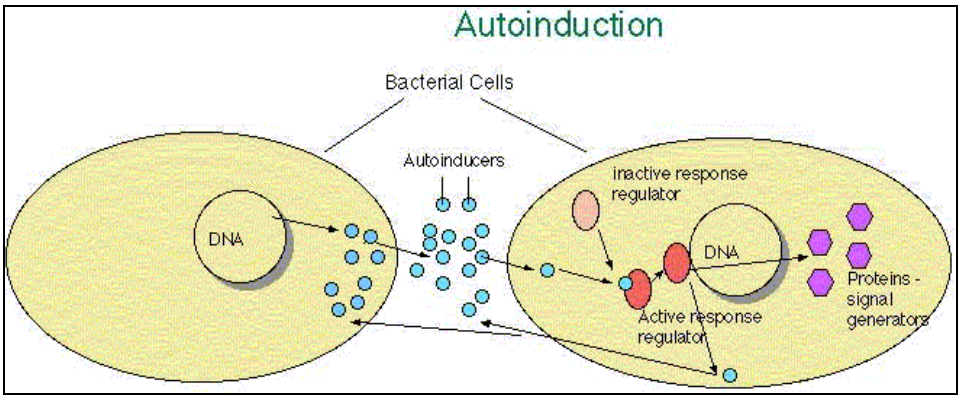

Fig.2. Autoinduction system used by Gram Negative Bacteria.

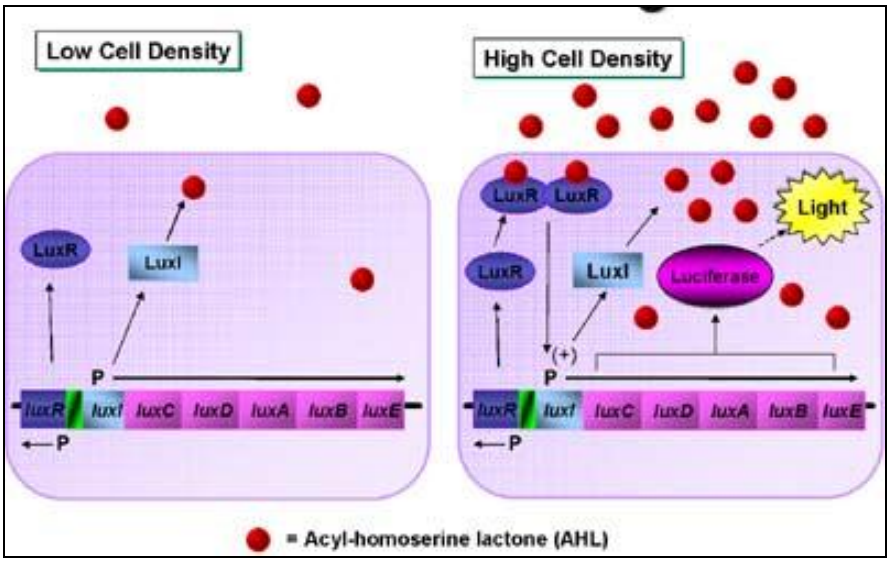

Fig.3. Bacterial Quorum Sensing.

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. (Desvaux *et al.* 2004). 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. (Ponciano *etal.* 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. T1SS is found in almost all phytopathogenic bacteria (Fig 4) 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 (Hennecke *et al.* 1991). Proteases and lipases from the soft rot pathogenic bacteria *Erwinia chrysanthemi* are examples of plant pathogen effectors secreted via the T1SS. (Palacios *et al.* 2001).

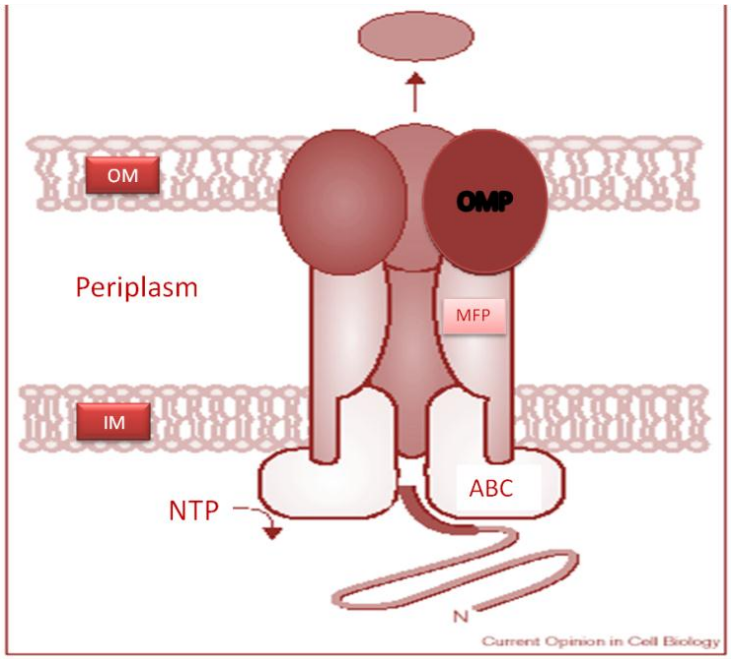

Fig.4. Type I Secretion Pathway.

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 (Fig 5). 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. 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. *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). (Stacey and Keen. 2003).

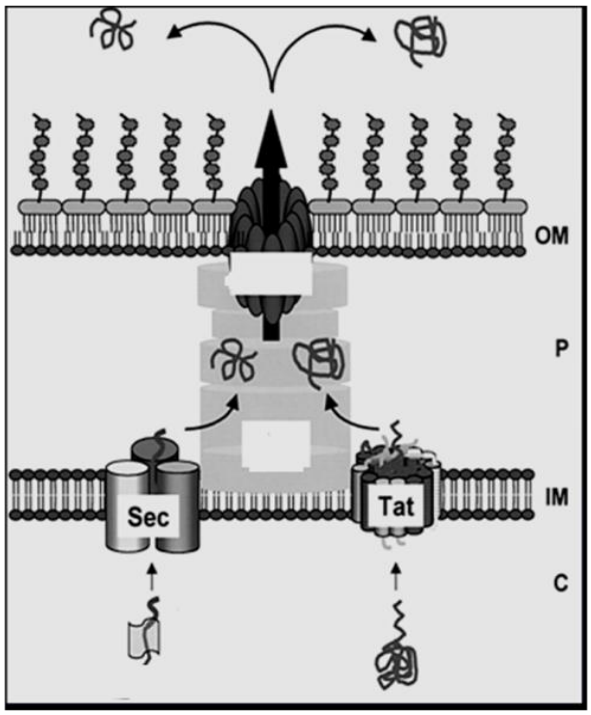

Fig.5. Type II Secretion Pathway

The type II secretion pathway is a two-step process

1. In the first step, the protein precursors are exported through cytoplasmic membrane to the periplasm using either the Sec-dependent pathway or Tat pathway, depending on the nature of the signal peptide.
2. In the second step, the proteins are secreted from the periplasm through the outer membrane to the extracellular space using T2SS apparatus.

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, by which the bacteria inject proteins (T3SS effectors) involved in their virulence into plant cells (Desvaux *et al*. 2004). The primary function of T3SS is the transportation of effector molecules across the bacterial membrane and into the plant cell (Fig 6). The genes that encode protein components of the T3SS are called hrc genes, 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. 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. 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. The characteristic feature of the T3SS structure, a needle-like protruding structure with a channel along which proteins travel, resembles to bacterial flagella, 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. Hrp pili, described for *P. syringae, R. solanacearum, Erwinia amylovora* and *Xanthomonas campestris pv. vesicatoria*, elongate distally with the addition of their major component, Hrp pilin subunits, whereas T3SS effectors are secreted from the hrp pilus tip. This proves that Hrp pili function as conduits through which substrates are transported. 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. Stebbins and Galan 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.

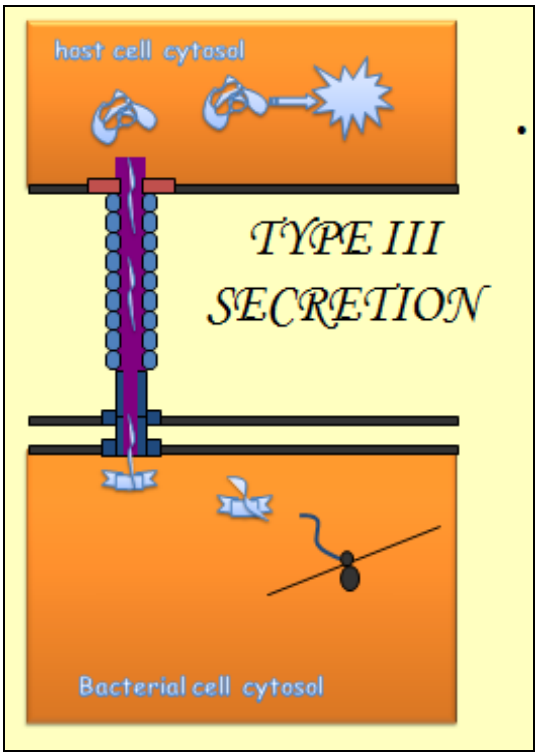

Fig.6. Type III Secretion Pathway.

Five functions of Type 111 secretion:

a. Bring bacterial & host cells close together.
b. Export proteins across bacterial envelope.
c. Translocate proteins across host cell membrane.
d. Translocated proteins subvert host cell functions.

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. 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 also appear to be virulence factors for erwinias. 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 (Zhao Y and Qi M. 2011). 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* . 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. 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. 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 . However, different avr genes, even of the same bacterium, contribute varying degrees of susceptibility/aggressiveness to bacteria that harbor these genes. It reveals that the particular Avr protein functions inside the host plant cell and enhances bacterial virulence.

CONCLUSIONS

- QS inhibitors have provided evidence of alternative method for fighting bacterial infections
- Initially the lure of interference with QS-controlled virulence poised researchers to identify different kinds of compounds or enzymes able to block QS.
- The enzyme based QSIs or quorum quenchers have successfully been applied in plant models, where the virulence of plant pathogens has been abolished
- The pathogenic bacteria throughout the infection process have played a important role in pathogenecis,virulence,sporulation ,biofilm formation
- The AHLs mediated quorum sensing plays an imporatant role in regulating the virulene factors,such as extracellular enzymes in *P.c.c*, conjugation in *Ag. Tumefaciens* and toxin production in *Burkholderia glumae*
- So the number of quorum quenching enzymes have been identified that degrade the signal molecules which is a new hope in attenuate the disease
- This approach is highly attractive because it does not impose harsh selective pressure for the development of resistance as with antibiotics .

Bacterial protein secretion systems are most important virulence determinants.

- There are 4 protein secretion systems in G-bacteria
- Type I secrete toxins and Type II secrete degradative enzymes.
- Type III and IV seem to be specific for phytopathogenesis.
- Type III and IV Secretion Systems are multi-protein complexes.
- Most of the bacteria are Gram-negative, of which biotrophic pathogenic bacteria fundamentally possess a type III secretion system
- *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.

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