

Plant-Pathogen Interactions: A Brief Insight into a Complicated Story

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Summary

Pseudomonas syringae is a bacterial plant pathogen that can lead to heavy losses in crop production. This bacteria is a very good model to study the infection processes, as it can cause disease in *Arabidopsis thaliana*, a well-studied plant model. This text presents an overview of the bacterial pathogenesis from a molecular biology perspective, and explains the role of plant responses in stopping the spread of the infection. Plant hormones are important elements for plant defence. Their role and how the pathogen interferes with their action will be discussed further.

Key words

Pseudomonas syringae, pathogenesis, plant-pathogen interactions, *Arabidopsis thaliana*

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Introduction

Modern agriculture has promoted monocultures because of their high-yield and economic benefits. Due to these cultivating practices, some pathogens are able to spread extensively, especially because there is a limitation in the varieties displaying resistance genes. It is difficult to quantify the threat and the damage this could bring to global food security and hence it is important to understand the plant-pathogen interactions as much as possible (Pennisi, 2010; Jones, 2013).

Gram-negative, rod shaped *Pseudomonas syringae* is one of the bacterial plant pathogens capable of producing heavy losses in crop yield (Ciarroni et al., 2015). Among other features, this bacteria is hemibiotrophic, has polar flagella, and causes different symptoms in plants (Vanneste et al., 2014). The outbreaks of this pathogen can inflict severe damage to tomato (*Solanum lycopersicum*) and crucifer plants. The exposed plant displays brown black leaf spots surrounded with chlorotic margin and dark specks on fruit that may become sunken and show zones of delayed ripening. Young plants can show stunting and yield loss upon infection and have reduced market value.

P. syringae is further divided into pathogenic variants (pathovars, pv) that differ in their host range (Peñaloza-Vazquez et al., 2000). More than 50 different pathovars have been described (Young, 2010), some of which are subdivided into races based on their host preference (González et al., 2000; Hirano and Upper, 2000). In order to study infection processes, and specifically the interaction of *P. syringae* with the host, it is important to have models that are easily reproduced in laboratory conditions. *P. syringae* pv. tomato (Pto) DC3000 has been the main choice, and the fact that it can cause disease in *Arabidopsis thaliana*, a widely used model plant, makes it an excellent system to understand the interaction for further applications in relevant crops.

The genome of Pto DC3000 (6.5 megabytes) is composed from a main chromosome of circular nature, and a pair of additional plasmids (Collmer et al., 2002). The pathogenesis and virulence of *P. syringae* is complex from a genetic point of view, and it is determined and shaped by global regulators (Hrabak and Willis, 1992; Rich et al., 1994; Kitten et al., 1998), the *hrp* gene cluster encoding the Type III Secretion System (T3SS), virulence factors, such as the phytotoxin coronatine and exopolysaccharides (Bender et al., 1999; Yu et al., 1999).

In general the infection with the *Pseudomonas syringae* species starts on the surfaces of the leaves where the bacteria can live epiphytically. They pass from the external environment to intercellular space through different types of openings in the leaves, known as stomata or simply through the wounds on the leaf surface (Hirano and Upper, 2000).

What happens once the pathogen is inside? Where does it go? It has been shown that after entering, the bacteria are localized in the apoplast where they can grow. The necessary growth elements have to be produced by the host molecular machinery, which the pathogen utilizes for its own benefit. This is when the bacteria, using the T3SS, introduces the effectors, a group of specialized proteins, into the cytosol of the host surrounding cells. That occurs through the plant cell wall, followed by the modified molecular behaviour of targeted cells and consequent spread of the infection.

However, plants also have an immune system to defend themselves from this type of biological interactions. For a successful establishment of infection, the pathogen must be able to overcome the plant immune barrier. This is enabled by the aforementioned effector proteins that allow the growth of the pathogen in the apoplast (Göhre and Robatzek, 2008). Some studies have described how the mutations in the T3SS gave rise to bacteria that are unable to introduce their effectors into the host cell, and show severely restricted growth in the host due to the action of the immune system of the plant (Mohr et al., 2008).

Mechanism of T3SS

As mentioned previously, T3SS is a key element for an effective establishment of infection. This is a very sophisticated structure, formed by approximately 30 different proteins, which drive secretion through both membranes of the bacteria into the cytoplasm of eukaryotes (Nguyen et al., 2000). The importance of this apparatus is so crucial that mutations that cause impaired function of the T3SS dramatically affect the capability of pathogenesis (Cunnac et al., 2009).

It is known that the genes that encode for T3SS components are grouped into clusters. While in some other species they can be found on plasmids exclusive for pathogenic bacteria, that is not the case in non-pathogenic ones (*Shigella flexneri*, and *Ralstonia solanacearum*) (Galán and Collmer, 1999). In the case of *Pseudomonas syringae* pv tomato, and in some other ones (e.g. *Salmonella typhimurium*, *Escherichia coli* (EPEC), *P. syringae*) (Orth et al., 2000), the gene cluster for the T3SS seems to be acquired through horizontal transfer, and is located only in the chromosome.

These genes encode for three groups of functional proteins: (a) structural, (b) effector proteins, introduced into the host cell to promote infection by the suppression of plant defences, (c) and chaperones, which protect effectors from aggregation and degradation, by driving them to the secretion apparatus (Anderson et al., 2010). There are additional encoded proteins in some strains of *P. syringae*, which are secreted in a T3SS dependent manner to help effectors to translocate across the cell membrane of the plant (Choi et al., 2013). A study from Collmer and colleagues (Collmer et al., 2002) has shown, through functional analysis of Pto DC3000 genome, that there are several groups of genes which encode effectors of type III secretion system (T3Es), with 31 confirmed, and some other 19 predicted. These effectors were described as “Hop” proteins (HR and pathogenicity outer protein) or “AVR” proteins (avirulence), and they are determining a successful infection, as it has been demonstrated for 28 of them (Lindeberg et al., 2006). The combination of all T3Es is essential to overcome the host defences, to grow or to cause symptoms in plants, but taken individually they can be dispensable.

The response of plant defence against *P. syringae*

When a plant is attacked by a pathogen, there are successive defence responses from the plant to overcome the infection. The mechanism of the defence depends on the type of molecule from the pathogen that is recognized by the plant, and also the intensity and speed of the respective responses. Pattern recognition

receptors (PRR) are involved in the first stage of defence upon pathogen detection.

Pattern recognition receptors and pathogen-associated molecular patterns

Plants are able to limit the colonization and proliferation of many microbial pathogens, mainly due to the activation of cell surface receptors, known as Pattern Recognition Receptors (PRRs). These proteins recognize specific conserved molecules of the microorganism, known as pathogen-associated molecular patterns (PAMP) (according to some authors also known as microbe-associated molecular patterns or MAMPs), with a number of PRRs (47 so far) that have been identified in the plant model *Arabidopsis*. FLS2 is one of the best described PRRs, showing a high conservation among different plant species (Zipfel et al., 2004). It is known to recognize flagellin, the main component of the bacterial flagella (Boller and Felix, 2009), reporting the presence of potentially invasive bacteria even if it has not entered the leaf (Melotto et al., 2006). The relationship between PRRs and flagella from bacteria is especially relevant for plant defence, because many pathogenic bacteria have flagella that are built from flagellin polymers, where a 22 amino acids residue at the N-terminus of flagella (flg22) is the recognized pattern (Felix et al., 1999), making this PRRs the prime targets for pathogen effectors.

After PAMPs recognition, the plant immune response is triggered. This response involves molecular mechanisms through MAPK (mitogen-activated protein kinase), resulting in the restriction of the bacterial growth through induction of pathogen-response genes, production of reactive oxygen species (ROS), and callose deposition where the infection occurs (in order to reinforce the plant cell wall) (Schwessinger and Zipfel, 2008). This is a slow process, known as PAMP-triggered immunity (PTI), whose intensity can increase with time. This is a general mechanism of protection against most microbes, regardless if they are pathogenic or not. Since this initial activation is slow, PTI adapted pathogens have evolved to acquire additional features that specifically suppress PTI.

PTI suppression by pathogen effectors

During evolution, effectors have contributed to increase the virulence of the pathogen, and to overcome defences of the host. One of the major roles of T3SS is to suppress PTI responses in the host plant.

The host defences can be defeated in several ways. Recent studies have shown three main strategies that allow pathogens to pass the PTI: (a) suppression of the activation of PTI through the action of effectors, (b) circumnavigation of PTI activities through production of toxins, (c) and degradation of PTI bioactive products through complex detoxification mechanisms (Anderson and Singh, 2011).

In *Arabidopsis* PTI is triggered by recognition of flg22, while the T3Es AvrPto, AvrPtoB and HopA11, suppress PTI through a blockade in the MAPK activation (De Torres et al., 2006; He et al., 2006; Zhang et al., 2007). Additionally, host PRRs can be evaded if pathogen effectors interfere with downstream signalling mechanisms (Boller and He, 2009). Therefore, pathogens use effectors to overcome PTI, and are able to proliferate. In this

case the plant is subjected to a process known as effector-triggered susceptibility (ETS) that leads to the development of the disease, which is a result of the interaction of the pathogen with the plant. This type of interaction is also known as “compatible”.

The plant-pathogen relationship is a story of co-evolution characterized as the “zigzag model” (Fig. 1), in which plants have acquired resistance genes (*R*) for pathogen effector recognition (or their effect over the plant). The resistance genes additionally activate defence responses with increased intensity and speed, being established as resistance against the pathogen (Effector-Triggered Immunity) (ETI) (Jones and Dangl, 2006).

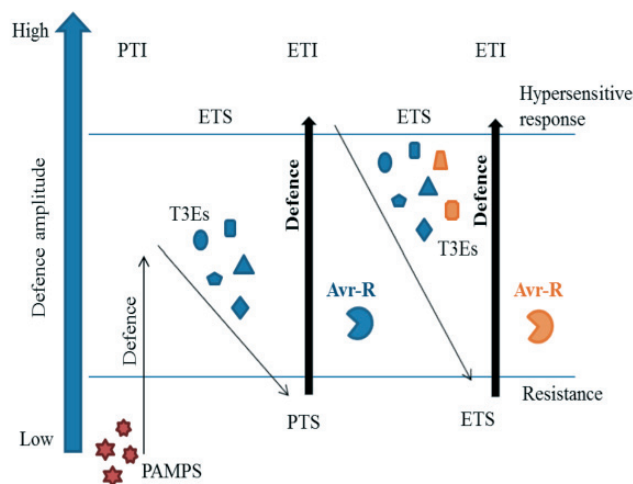


Figure 1. The „zigzag model” discloses the steps of the interaction between the plant and its pathogen.

Firstly, pathogen-associated molecular patterns (PAMPs/MAMPs, marked as red stars) are recognized by their corresponding PRRs in plants and that leads to the PAMP-triggered immunity (PTI). Secondly, the pathogens possess effectors (marked in blue) that disable this PAMP recognition leading to effector-triggered susceptibility (PTS). Thirdly, these effectors become avirulence factors (Avrs) once the plant developed cognate resistance protein (*R*-protein, marked in blue), leading to effector-triggered immunity which is amplified version of PTI that culminates into hypersensitive response (HR) or cell death. Lastly, the pathogen may develop new effectors (marked in orange) or/and loses its Avrs to bypass ETI, leading to effector-triggered susceptibility (ETS). The “arms race” can continue. Adopted and extended from Jones and Dangl (2006).

ETI and its suppression

The plant can also detect the invading microorganisms by avirulence proteins (Avr) which specifically recognize pathogen effectors. Often it is done indirectly, through the detection of changes caused by the activity of effector proteins in the plant cell (Fig. 1). This model of indirect detection is known as the “guard hypothesis” (Van der Biezen and Jones, 1998). According to it, *R* proteins interact with or guard another protein, the guard. Guard proteins detect the interference with the guardee.

protein, activating a strong resistance response known as ETI. To restrict the spread of the pathogen during ETI, the plant cells surrounding the pathogen location enter a programmed cell death, known as the hypersensitive response (HR), which includes the senescence of the affected cells (Jones and Dangl, 2006) through tissue necrosis in a localized manner. Also, there is a production of phenolic compounds and antimicrobial agents where the pathogen is located. This type of interaction is known as “incompatible”, where the pathogen is considered avirulent, and the host (plant) resistant.

While PAMPs are not pathogen-specific, the recognition of effector molecules (in this case pathogen specific) leads to a more intense and efficient response, which is thought to be more difficult to suppress (Katagiri and Tsuda, 2010). Through ETI, the spread of the bacteria is associated with the activation of a systemic immunity (systemic acquired resistance, SAR) (Cameron et al., 1994), which protects unrelated parts of the plant from successive attacks from the same pathogen. Notwithstanding, there are pathogens that can also prevent the ETI activation, either by suppression before it starts, or by downstream interference. Those pathogens that succeed in suppression are able to spread through the plant and develop a disease, known as effector-triggered susceptibility (ETS). Some authors have studied the less known mechanisms by which T3Es suppresses ETI, having found that while some mechanisms are very specific, while others are more general (Macho and Beuzón, 2010; Macho et al., 2010).

In this evolutionary competition plants have developed R proteins to recognize ETI-suppressor effectors. The balance among them determines if this interaction has ETS (plant disease and success for the pathogen), or ETI (strength and success for the host plant) as final product.

Salicylic acid and plant coronatine during host-pathogen interaction

Endogenous plant hormones can influence and mediate the plant defence against pathogens (Hayat et al., 2007). In *Arabidopsis thaliana* Col-0 there are two main responses of this type: those mediated by salicylic acid (SA), and those mediated by jasmonic acid or methyl jasmonate (JA or MeJA). Both are capable of suppressing the proliferation of a many bacterial and microbial pathogens (Kunkel and Brooks, 2002; Bostock, 2005; Glazebrook, 2005). It has been described that resistance against hemibiotrophic or biotrophic pathogens, e.g. *P. syringae*, is mediated by SA signalling; while MeJA signalling is generally a response to wounding or necrotrophic pathogens (Ryan and Pearce, 1998). The recognition of PAMPs leads to an accumulation of SA (Tsuda et al., 2008), with a posterior activation of gene expression of basal defence (Asai et al., 2002). Coronatine (COR) is a phytotoxin produced by *Pto* that mimics MeJA, and can activate JA response mechanisms (Brooks et al., 2005). This is relevant because the signalling of JA and SA can be antagonistic (Kunkel and Brooks, 2002). It means that the activation of MeJA induced signalling, can at the same time suppress SA signalling, which impairs the basal defence against *P. syringae* attack (Delaney et al., 1995; Wildermuth et al., 2001; Nawrath et al., 2002). COR production is under HrpL control, and has been shown to be involved in suppression of stomatal closure during PTI (Melotto et al., 2008). Additionally, it interferes with

SA dependent defence (Brooks et al., 2005). Experimental evidence has demonstrated that a mutant *A. thaliana* (coronatine insensitive mutant 1, *coil*), displays a constitutive expression of SA-dependent defences, presenting a resistant phenotype during infection with *P. syringae* (Feys et al., 1994). As coronatine cannot interfere with the SA signalling, the plant is not susceptible, and provides evidence that MeJA (and coronatine) negatively influence the SA-mediated defences (Kloek et al., 2001). COR can aid the pathogen in the inhibition or the delay of the plant defence mechanism, providing a window of opportunity for the pathogen to effectively infect the host (Reymond and Farmer, 1998).

Conclusions

Biological control of *P. syringae* pv. tomato has been largely unexplored in contrast to the control of other bacterial and viral leaf pathogens. The better understanding of this interaction is a key factor in improving the quality and quantity of crop production in a world with an increasing demand for food. It is interesting that the evolutionary competition between the host and the pathogen, which has led to different responses to overcome each other's actions (defence or attack), is an on-going story. From the side of the bacteria, effectors are key elements in the establishment of an infection, and many studies have highlighted the importance of these specialised proteins in the course of pathogenesis. Plant hormones play a central role in the defence, and the bacteria have developed mechanisms to interfere with this response in order to grow and proliferate in the plant.

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