A PLANT-PATHOGEN INTERACTIONS: A BRIEF INSIGHT INTO A COMPLICATED STORY

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ABSTRACT

Pseudomonas syringae pv. *tomato,* a bacterial plant pathogen, poses a significant threat to crop production and often leads to considerable yield losses. Due to its infectivity in *Arabidopsis thaliana*, a widely studied plant model, *P. syringae* serves as an exemplary model organism for studying the intricacies of infection processes. This short communication offers a molecular biology-based overview of bacterial pathogenesis and highlights the interplay between the virulence factors of the pathogen and the defence mechanisms of the host. Phytohormones, central regulators of the immune response, and the methylation status of the host are of central importance for plant defence. Wide genome methylation changes are dynamic under the influence of pathogens, and it is hypothesised that loss of DNA methylation in the host plant (*Arabidopsis thaliana* Col-0) plays a role in resistance to bacteria. This overview tackles the multiple functions of these important defence mechanisms and examines how the pathogen subverts their functions to facilitate the progression of infection.

Keywords: Pseudomonas syringae pv. *tomato*, pathogenesis, plant-pathogen interactions, methylation, *Arabidopsis thaliana*

INTRODUCTION

The endorsement of monocultures in agriculture, primarily due to their higher yields and economic benefits, has favoured the widespread dissemination of pathogens (Ciarroni *et al*., 2015). These cultivation practices have played a pivotal role in limiting the range of available plant varieties with 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). *Pseudomonas syringae* pv. *tomato*, a Gram-negative, rod-shaped bacterium, stands out among bacterial plant pathogens that can cause significant crop yield losses. With its haemibiotrophic properties, polar flagellation and ability to cause various symptoms in plants, this pathogen poses a significant threat to crops such as tomatoes (*Solanum lycopersicum*) and cruciferous plants. Infection with *P. syringae* pv. *tomato* (Pto) DC3000 can cause severe damage, resulting in brown-black leaf spots surrounded by chlorotic edges and dark spots on the fruit, which may be sunken and show zones of delayed ripening (Vanneste et al., 2014). Young plants may experience stunting, yield loss and reduced market value. As Pto DC3000 is able to cause diseases in *Arabidopsis thaliana*, a widely used model plant, it serves as an excellent system for understanding interactions that can subsequently be transferred to relevant crops (Zeng *et al*., 2011; Xin and He, 2013). A response that a host plant can offer is related also to DNA methylation. It has been reported that the loss of DNA methylation

enhances resistance to pathogen bacteria in a non-specific manner (Xie and Duan, 2023; Saijo and Reimer-Michalski, 2013). By studying the methylation status of the host plant (*Arabidopsis thaliana* Col-0) once infected with Pto DC3000, we can gain an insight in further understanding the intricate interplay between the pathogen and its host (Zhang *et al*., 2018).

THE RESPONSE OF PLANT DEFENCE AGAINST PSEUDOMONAS SYRINGAE. EVOLUTIONARY ARM RACE

The interaction between *Pseudomonas syringae* pv. *tomato* (Pto) and *Arabidopsis thaliana* has become a valuable model system for the study of plant-pathogen interactions. This interaction is controlled by two important branches of plant immunity: pattern-Triggered Immunity (PTI) and Effector-Triggered Immunity (ETI) (Anderson and Singh, 2011). Pattern-Triggered Immunity (PTI) serves as the first line of defence against invading pathogens. Upon recognition of conserved microbial molecules known as pathogen-associated molecular patterns (PAMPs) by pattern recognition receptors (PRRs), plants activate a broad-spectrum immune response aimed at limiting pathogen invasion and colonization (Schwessinger and Zipfel, 2008; Jones and Dangl, 2006). In the case of Pto, the recognition of PAMPs, such as bacterial flagellin or lipopolysaccharides, triggers PTI in Arabidopsis (Zipfel et al., 2004; Melotto et al., 2006). Effector-triggered immunity (ETI), on the other hand, is a more specific and robust form of immunity that is triggered by the recognition of pathogen effector proteins by plant resistance proteins (R). Arabidopsis possesses a variety of R proteins that can specifically recognize and bind to effectors delivered by Pto during infection (Boller and Falix, 2009). Upon recognition, the R proteins initiate a signalling cascade that leads to a rapid and often hypersensitive response characterized by localized cell death known as the hypersensitive response (HR). This localized cell death limits the spread of the pathogen and activates systemic defence responses, conferring resistance to Pto and other pathogens expressing the recognized effectors (Jones and Dangl, 2006). The interaction between Pto and the PTI and ETI pathways of Arabidopsis is complex and dynamic, with the pathogen employing different virulence strategies to evade or suppress plant immunity (Hulak and González Plaza, 2015).

METHYLATION CHANGES UPON PSEUDOMONAS SYRINGAE ATTACK

A fascinating aspect of this plant-pathogen interaction is the role of DNA methylation in modulating the plant's response to pathogen attack. Research has shown that Pto infection induces changes in the DNA methylation patterns of *Arabidopsis* (Yu *et al.*, 2013). These changes in DNA methylation can affect the expression of genes involved in plant defence responses and thus influence the outcome of the interaction. For example, certain defence-related genes may be hypermethylated, leading to their repression, while others may be hypomethylated, leading to their upregulation. The effects of DNA methylation on gene expression during Pto infection are complex and context-dependent. Studies have shown both positive and negative correlations between DNA methylation changes and gene expression levels in response to pathogen attack (Pavet *et al.,* 2006*;* Laird, 2010; Dowen *et al.,*2012*)*. This suggests that DNA methylation acts as a dynamic regulatory

mechanism that fine-tunes plant defence responses to optimize survival under different environmental conditions. Furthermore, the role of DNA methylation in plant immunity goes beyond the direct regulation of gene expression. It has been proposed that DNA methylation can also influence the structure and accessibility of chromatin, modulating the accessibility of defence-related genes to transcription factors and other regulatory proteins (Gong et al., 2002; Huettel *et al.,* 2007; Metzke *et al.,* 2009). Interestingly, Pto has evolved mechanisms to manipulate the host DNA methylation machinery to promote its own survival and proliferation in the plant. For example, it has been reported how certain bacterial effectors target components of the plant's DNA methylation machinery (Canonne and Rivas, 2012) resulting in changes in DNA methylation patterns that promote bacterial colonization and disease progression (Marois *et al.,* 2002). Overall, the interplay between Pto and *Arabidopsis thaliana* provides a fascinating context for studying the role of DNA methylation in plant immunity. Further research in this area promises to deepen our understanding of the molecular mechanisms underlying plant-pathogen interactions and may ultimately lead to the development of novel strategies to improve plant resistance to bacterial pathogens.

CONCLUSION

Biological control strategies targeting Pto have received relatively little attention compared to efforts aimed at controlling other bacterial and viral foliar pathogens. However, a deeper understanding of this intricate interaction is essential to increase both the quality and quantity of crop yields, especially in the context of increasing global food demand. The ongoing evolutionary arms race between host plants and pathogens underscores the dynamic nature of their interactions, with each side constantly adapting to counter the defence and attack strategies of the other. Effector proteins prove to be central components in the pathogen's arsenal and facilitate the establishment of infection, as shown by numerous studies highlighting their importance in pathogenesis. The methylation status of *Arabidopsis thaliana*, especially under the influence of Pto, provides interesting insights into the complex dynamics and epigenetic influence of plant-pathogen interactions. Changes in DNA methylation patterns serve as a molecular fingerprint of the plant's response to pathogen invasion and provide insight into the underlying regulatory mechanisms controlling defence responses. By studying the methylation landscape of *Arabidopsis*, researchers can decipher the extent to which the pathogen disrupts the host's epigenetic machinery to facilitate infection. The dialog between the plant and the pathogen provides to be valuable to unravel the interaction of plant immunity and pathogen virulence. The study of methylation status in *Arabidopsis thaliana* during infection with *Pseudomonas syringae* pv. *tomato* therefore offers a promising avenue to improve our understanding of this interactions at the epigenetic level.

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