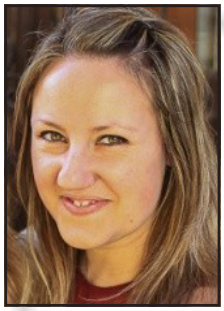
MOLECULAR MECHANISMS OF BACTERIAL VIRULENCE



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Pseudomonas syringae (*P. syringae*) is a widespread bacterial pathogen that causes disease on a broad range of economically important plant species. *Pseudomonas* colonize the leaf surfaces of plants and reach dense bacterial populations without causing disease. Pathogen ingress to host tissue is the critical first step in infection, as nutrients on the leaf surfaces are believed to be very limited [1]. Motile *Pseudomonas* bacteria do not have the means to penetrate the leaf epidermis directly and enter by natural surface openings such as stomata or wounds [2]. *P. syringae* bacteria multiply in the apoplastic intercellular spaces of plant cells and remain extracellular. Concomitantly, plant cells sense the microbial presence and activate an array of defence responses using an innate immune system based on extracellular recognition of highly conserved elicitor molecules called pathogen-associated molecular patterns (PAMPs) through plasma membrane receptors [3], whose objective is to restrict bacterial growth.

Globally, plant immunity relies on a complex network of small-molecule hormone signalling pathways [4]. Classically, salicylic acid (SA) signalling mediates resistance against biotrophic and hemi-biotrophic microbes such as *P. syringae*, whereas a combination of jasmonic acid (JA) and ethylene (ET) pathways activates resistance against necrotrophs such as the fungal pathogen *Botrytis cinerea* [4]. SA and JA/ET defence pathways generally antagonize each other and thus, elevated resistance against biotrophs is often correlated with increased susceptibility to necrotrophs, and *vice versa* [5]. The collective contribution of these two hormones during plant-pathogen interactions is crucial to the success of the interaction.

In order to infect, adapted bacterial pathogens secrete phytotoxins and virulence effector molecules into the plant cell via a specialised type-III secretion apparatus (TTSS) that contribute collectively to pathogenesis [6]. This mechanism is essential for successful infection by both plant- and animal-associated bacteria as bacterial mutants deficient in the TTSS are no longer pathogenic [7]. Effectors collectively contribute to pathogenesis inside the cell by targeting host molecules and defeating plant defences. Each bacterial strain possesses a set of 20–30 effectors, which have overlapping activities, are functionally interchangeable, and diverge in composition between strains [8].

However, despite the fact that elucidating effector action is essential to understanding bacterial pathogenesis and plant resistance, the molecular function and host targets of the vast majority of effectors remain largely unknown. In another remarkable example of pathogen adaptation, some *P. syringae* strains have developed sophisticated strategies for manipulating hormonal homeostasis by producing coronatine (COR), a mimic of the bioactive JA hormone [9]. COR contributes to disease symptomatology by inducing chlorotic lesions [10- 12], facilitates entry of the bacteria into the plant host by stimulating the opening of stomata [1, 2] and promotes bacterial growth by inhibiting SA-dependent defences required for *P. syringae* resistance, due to its activation of the antagonistic JA pathway [13, 14]. Despite the importance of this phytotoxin to the global infectious process of *Pseudomonas*, the molecular mechanisms by which COR hijacks bacterial-induced plant immunity during microbial invasion remains poorly understood. Understanding, at the molecular level, how bacterial phytotoxins and effectors act inside the plant to suppress plant immunity is crucial towards designing novel strategies to protect crop plant in the field to specific pests.

Selena Giménez Ibáñez is a researcher in plant-microbe interaction with a special interest in understanding plant defence mechanisms and how phytopathogenic *Pseudomonas* are able to become successful pathogens through it repertoire of effectors and phytotoxins. Specifically, she is studying:

1. Mode of action of *Pseudomonas* effectorome: Identification of host targets and processes hijacked by the repertoire of *Pseudomonas* effectors to modify and/or suppress plant immunity.
2. Mode of action of *Pseudomonas* phytotoxin COR.
3. Application of our current results to engineer specific crop plants with long-lasting resistance to economically important pests.