Plant diseases are a major cause of crop loss globally, representing a substantial obstacle in sustainable production of food and energy crops that are essential for basic human nutrition and health. Understanding how pathogens cause diseases therefore has broad implications in agriculture and human health. Sheng Yang He’s group is studying plant*-Pseudomonas syringae* interactions to gain insights into some of the basic principles underlying bacterial pathogenesis and disease susceptibility in plants. To cause disease, *P. syringae* bacteria produce a variety of virulence factors, including numerous "effector" proteins that are secreted through the type III protein secretion system (T3SS), and the phytotoxin coronatine, which functions as a molecular mimic of the plant hormone jasmonate.

**Bacterial Effector Proteins**  
Our early work revealed the secretion function and part of the supramolecular structure of the T3SS of *P. syringae*. More recently, our work contributed to the discovery of two basic functions of *P. syringae* effector proteins: (i) suppression of plant immune responses and (ii) creation of an aqueous apoplast in which bacteria multiply in infected plant leaves (Figure 1). Over the years, we have studied a number of different *P. syringae* effectors (e.g., AvrPto, HopAO1, HopZ1, HopM1, AvrE, HopO1-1). Our current effort is directed at understanding how these various effectors contribute to disease development, with the hope that, one day, we could achieve the challenging goal of reconstituting disease susceptibility using host plant mutants that could recapitulate the collective virulence activities of *P. syringae* effectors.

**Figure 1:** *Pst* DC3000 bacteria use a needle-like type III secretion system to inject “effector” proteins (effector names indicated in red or green) into the plant cell to attack plant proteins (blue boxes); many of which are components of the plant immune signaling pathway and/or are involved in regulating the water content in the leaf apoplast.

**The Immune Function of Plant Stomata**  
Plant stomata, formed by pairs of guard cells, are microscopic pores on the surface of all land plants. We found that plant stomata have an important immune function. Specifically, stomata close in response to plant and human pathogenic bacteria (Figure 2). Stomatal guard cells could perceive bacteria through pattern recognition receptors, such as flagellin receptor FLS2, activating a signaling cascade that requires the plant stress hormones salicylic acid. The signal transduction pathway underlying stomatal closure to pathogens is not well characterized. We are taking several approaches to increase our understanding in this area.

**Figure 2:** Stomata in the leaf epidermis and mesophyll cells inside a leaf can both respond to pathogens, leading to stomatal closure to restrict bacterial invasion or establishing a hostile mesophyll apoplast environment to prevent aggressive bacterial multiplication. Some virulent pathogens, like *Pst* DC3000 bacteria, evolved the jasmonate-mimicking toxin coronatine (COR) to activate JA signaling cascade, leading to suppression of stomatal and apoplast defenses to facilitate massive entry and multiplication.

**Jasmonate Signaling in Disease**

For many years, we have been interested in identifying the host target of coronatine, a toxin produced by *P. syringae*. Coronatine shares striking structural similarities to the plant hormone jasmonate, which plays an important role in plant growth, development, and immunity. A few years ago, we used coronatine as a molecular probe to identify key regulators (e.g., JAZ repressors) of jasmonate signaling and components of the JAZ-COI1 jasmonate receptor complex (Figure 2). Our current work is aimed at achieving a deep understanding of the jasmonate signaling pathway, with the goal of modifying this pathway for enhanced pathogen resistance.

**New Research Initiatives: The Next Phase of Study**

Our current understanding of bacterial pathogenesis and disease susceptibility in plants remains largely one-dimensional, reflecting the heavy reliance on simplistic bilateral interactions of one pathogen and one host under static laboratory conditions. As a result, our knowledge of disease susceptibility does not accurately reflect the multi-dimensional features of plant disease development that occur in nature. To break new ground for the next phase of research on bacterial pathogenesis and disease susceptibility in plants, we have initiated two new projects:

(i) We are studying the molecular bases of the effects of temperature and humidity, which are known to significantly influence disease outbreaks in crop fields. (ii) We are developing a soil-based gnotobiotic plant growth system (called “FlowPot”) that enables the study of plant-microbe interactions in soil substrates, in the presence or absence of the endogenous microbiome. With further optimization, we hope that the FlowPot gnotobiotic system may be broadly useful in the study of interplay between the microbiome and plant biology.