Multilayered strategies for attack, defense and counter-defense in the plant cell wall: the infection of tomato by *Phytophthora infestans* as a model system

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Plant pathogens can be classified into three groups, based on their mechanism of infection: biotrophs, necrotrophs and hemibiotrophs. Biotrophic pathogens penetrate the plant wall, parasitize host cells while evading or suppressing defense responses and require viable host tissue for nutrition and reproduction. In contrast, necrotrophs overwhelm plants by secreting mixtures of degradative enzymes that allow the pathogens to subsist on necrotized host tissue. Hemibiotrophic pathogens employ elements of both these strategies in a biphasic 'stealth' infection mechanism. This involves an initial biotrophic phase, when the pathogen proliferates asymptomatically in the host and efficient mechanisms must be employed to evade and suppress host defenses. Subsequently, in the second stage, hemibiotrophs orchestrate a physiological switch from asymptomatic infection to large-scale cell death and tissue dissolution, presumably resulting from the coordinated secretion of factors such as lytic enzymes and cell death elicitors.

Hemibiotrophic bacteria, such as *Pseudomonas syringae*, use the type three secretion system to introduce suites of proteins directly into plant cells. These include avirulence (Avr) effectors that trigger programmed cell death (also referred to as the hypersensitive response), when recognized by the corresponding resistance (R) proteins of the host plant, as well as a variety of other proteins, some of which are effectors that function as suppressors of host defenses. This form of recognition, of an Avr protein by a plant R protein, has been termed effector-triggered immunity (ETI). Microbial pathogens may also trigger basal plant immune response by invariant structures on their surfaces, termed pathogen-associated molecular patterns (PAMPs), the perception of which at the host cell surface induces PAMP-triggered immunity (PTI). Both ETI and PTI can be suppressed by secreted effector proteins from bacteria and several of these have now been described. However, far less is known of analogous effectors that might suppress host defenses in order to mediate hemibiotrophy in eukaryotic phytopathogens, notably oomycetes or fungi.

We have been addressing the hypothesis that hemibiotrophic eukaryotes may maintain a biotrophic interaction with their hosts, and trigger the subsequent transition to necrotrophy, by the coordinated and temporally regulated expression of distinct subsets of genes that encode secreted protein effectors. As an experimental model system we have focused on the interaction between tomato and the oomycete *Phytophthora infestans*. Results will be presented describing the use of RNA-Seq analysis of the infection process, together with functional screens for secreted proteins, and molecular modeling. This strategy is providing new insights into the complex arms race in the plant cell wall that is played out over many time scales, providing a remarkable example of molecular co-evolution.