Title Mechanisms of induced resistance against *Botrytis.*

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Abstract

Necrotrophic fungal pathogens constitute a significant problem in agricultural and horticultural crops production worldwide. Many foliar, soil-borne and storage pathogens are necrotrophs surviving by extracting nutrients from killed cells. To acquire nutrients, necrotrophs induce cell death (necrosis) and degrade plant cell wall by releasing toxins and cell wall degrading enzymes into host tissue. Failure to limit necrosis or cell wall degradation by the plant leads to enhanced infections resulting in diseases that culminate in the death and decay of the entire infected plant or its parts. Over the last decades, significant progress has been made towards understanding the genetic control of plant responses to biotrophic pathogens. In contrast, the biological processes underlying host responses to infection by necrotrophic fungi are not understood that well. We are using host responses to *Botrytis cinerea* as a model to understand the genetic components of plant resistance to necrotrophs. *B. cinerea* is a typical necrotrophic pathogen and causes the gray mold disease in diverse crops. Plant responses to *Botrytis* and other necrotrophic pathogens are regulated by a complex network of interacting genetic, molecular and hormonal factors in the plant. In addition, diverse pathogen derived molecules and significant environmental factors contribute to disease development. Thus, identification of genetic resistance to necrotrophic pathogens particularly *Botrytis* has been challenging. Recent efforts by many laboratories have started to shed light on the mechanisms of host responses to necrotrophic pathogens. Comparisons of basal resistance in *Arabidopsis* and tomato reveal resistance mechanisms that show a functional conservation in the two plant systems but also differences in disease development. I will discuss recent progress in the area of host responses to necrotrophic pathogens, cross-talk with plant responses to insect pests and abiotic stress factors.