Sclerotinia PATHOGENESIS: INTRICACIES OF A BROAD HOST RANGE NECROTROPH

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Sclerotinia sclerotiorum has the ability to blight and rot the stems, leaves, and fruits of a broad range of dicotyledonous plants. These attributes have made it a model of necrotrophic pathogenicity. An understanding of the basis for this aggressiveness and broad host range is of fundamental interest to the long-term goal of effectively managing Sclerotinia diseases. Towards this goal a number of genes that contribute to virulence have been functionally characterized. Chief among these is the oxaloacetate acetyl hydrolase (*oah1*)-encoding gene that catalyzes the final step required for the biosynthesis of oxalic acid in *S. sclerotiorum*. Deletion of *oah1* results in the complete elimination of oxalic acid accumulation under all tested conditions. Surprisingly, these mutants retain the ability to infect susceptible hosts. In some host species, this infection is confined to a small primary lesion. In others, primary lesions are able to expand and blight host tissues. These phenotypes, coupled with cytological observations, and evidence from other independent studies have led us to propose a two-phase model of pathogenicity for *S. sclerotiorum*. Phase one constitutes a battle for basic compatibility and may be facilitated by lineage-specific as well as shared necrotrophic effectors. Phase two colonization appears to be largely mediated by acidification of host tissue primarily through oxalic acid accumulation. Transcriptomic analysis of infected hosts and *in vitro* inducedinfectious development have allowed us to refine the list of candidate factors mediating theinitial phase of compatibility. Functional analysis of genes encoding these factors has revealed that hyper-accumulation of oxalic acid in the early infection court blocks compatibility. This finding further support the hypothesis that oxalic acid independent factors play critical roles in establishing initial compatibility. We have recently developed CRISPR-Cas9 transformation procedure that allow for high rates of insertional mutagenesis in *S. sclerotiorum*. We are utilizing these techniques to characterize candidate effectors to test our two-phase model of pathogenesis and identify critical points in the infection process that may be targetedto enhance host resistance.