SOYBEAN DEFENSE MECHANISMS AGAINST *Sclerotinia sclerotiorum*

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Sclerotinia sclerotiorum, a necrotrophic fungal pathogen with a broad host range, causes a devastating disease on soybean called Sclerotinia stem rot (SSR) that can lead to losses as high as 50-60%. Resistance mechanisms against SSR are poorly understood, thus hindering the commercial deployment of SSR resistant varieties. We used a dual approach utilizing RNAsequencing and GC-MS based metabolomics to decipher the molecular mechanisms governing resistance to *S. sclerotiorum* in soybean. We have focused on the plant membrane-localized NADPH oxidases, also known as respiratory burst oxidase homologues (RBOHs). These proteins play crucial roles in various cellular activities, including plant disease responses, and are a major source of reactive oxygen species (ROS). *S. sclerotiorum* produces a key virulence factor, oxalic acid, that induces programmed cell death (PCD) in the host plant, a process that is reliant on ROS generation. Using protein sequence similarity searches, we identified 17 soybean RBOHs (GmRBOHs) and studied their contribution to SSR disease development, drought tolerance and nodulation. We clustered the soybean RBOH genes into six groups of orthologues based on phylogenetic analysis with their Arabidopsis counterparts. Transcript analysis of all 17 GmRBOHs revealed that, of the six identified groups, group VI (GmRBOH-VI) was specifically and drastically induced following *S. sclerotiorum* challenge. Virus-induced gene silencing (VIGS) of GmRBOH-VI using Bean pod mottle virus (BPMV) resulted in enhanced resistance to *S. sclerotiorum* and markedly reduced ROS levels during disease development. Coincidently, GmRBOH-VI-silenced plants were also found to be drought tolerant, but showed a reduced capacity to form nodules. Our results indicate that the pathogenic development of S. sclerotiorum in soybean requires the active participation of specific host RBOHs, to induce ROS and cell death, thus leading to the establishment of disease.