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DUAL RNA-SEQ IDENTIFIES EARLY MOLECULAR PLAYERS IN SOYBEAN-*SCLEROTINIA SCLEROTIORUM* INTERACTIONS.

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Sclerotinia stem rot (white mold), caused by fungal pathogen *Sclerotinia sclerotiorum*, poses a threat to soybean production worldwide. No naturally complete resistance has been reported to exist in soybean. Studying the molecular interactions between soybean and *S. sclerotiorum* is a promising approach to identify the soybeans genes that are controlling the quantitative resistance and, on the other hand, to discover pathogenicity and virulence factors in *S. sclerotiorum* which could be considered as targets to disable and weaken the aggressiveness of the pathogen. This study utilized dual RNA-sequencing strategy to characterize the transcriptomes of both soybean and *S. sclerotiorum*. The RNA samples were obtained from infected leaves of a susceptible soybean line (AC) and an AC transgenic line (OxO) which contains an enzyme that degrades the oxalic acid (OA) produced by *S. sclerotiorum* thereby conferring resistance. Noticeable host transcriptomic changes were revealed in both soybean genotypes at 4 and 8 hours post inoculation (hpi), with more than 600 differentially expressed genes (DEGs) responding after inoculation with an fdr-corrected p-value cutoff of 0.02. At these early time points, most of the soybean DEGs were similarly induced in both genotypes, but the levels of induction were higher in OxO for some genes, including dirigent proteins, thaumatin-like proteins, and glutathione-S-transferases. Functional annotations of soybean DEGs induced in both genotypes identified approximately 50 transcription factors and signaling components that might be critical regulators of initiation of basal defense to *S. sclerotiorum* infection. GO enrichment analysis were reflective of a quick induction of a host oxidative burst, JA and ethylene signaling, and biosynthesis of several soybean anti-microbial secondary metabolites in both genotypes. In addition, the GO suggested that both OxO and AC negatively regulated programmed cell death, possibly via the manipulation of JA/ET and SA pathways. The expression changes of selected genes were supported by qRT-PCR. This RNA-Seq study also revealed dramatic transcriptomic changes in the pathogen, *S. sclerotiorum,* at 4 and 8 hpi in leaves versus in axenic culture, with close to three thousand genes detected as DEGs. Pathogen-encoded plant-cell-wall-degrading enzymes (PCWDEs) were shown to be one of the major players in the early infection stages and the expression patterns suggested more important roles of pectinases and cutinases for infection at these early time points. 160 secretory proteins were predicted as fungal effector candidates, for example: proteins harboring chitin-binding domains, LysM domains, and necrosis-inducing domains. Also, numerous genes exhibited higher expressions in *S. sclerotiorum* infecting OxO than AC, including genes involved in oxalic acid biosynthesis, botcinic acid metabolism, and various PCWDEs. It was proposed that the removal of oxalic acid in the OxO transgenic could have an impact on the gene expression of other virulence factors.

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