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Using high-throughput sequencing to characterize the genetic and genomic architecture of brown stem rot resistance in soybean

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Breeding for pathogen resistance is an important objective to improve and protect soybean yields. In 2010, 14.4% of total soybean yield was suppressed by diseases. Brown stem rot (BSR), caused by the fungus *Phialophora gregata*, reduces yield by as much as 38%. To date, three dominant BSR resistance genes have been identified: *Rbs1*, *Rbs2*, and *Rbs3*, however the gene networks regulating defense responses to BSR remain unknown. Further, identifying resistant germplasm by genotyping or phenotyping remains difficult due to complexities of soybean/*P. gregata* interactions. We conducted RNA-Seq of *P. Gregata* infected and mock infected leaf, stem, and root tissues of both a resistant (PI 437970, *Rbs3*) and a susceptible (Corsoy 79) soybean genotype. Our bioinformatics analyses focused on treatment, genotype, and treatment by genotype effects on gene expression. Our results indicate there is little overlap in differential gene expression between tissues when infected by *P. gregata*. Further, defense, DNA replication, and iron homeostasis are the hallmarks of *P. gregata* resistance. *De novo* assembly of the *Rbs3* region identified many receptor-like proteins (RLPs) with differential expression between genotypes, indicating their importance in resistance. All R genes in the *Rbs* loci were identified, characterized and compared to expression data, revealing that RLPs are likely candidate *Rbs3* resistance genes. *Rbs3*-mediated resistance is likely initiated in root tissue. The RNA-Seq data was also used to generate novel SNPs within the *Rbs3* locus that could be used for phenotyping earlier than current protocols allow and also fine mapping *Rbs3.*