B-174

Identification of molecular mechanisms underpinning partial resistance to *Phytophthora sojae* in *Glycine max* using a genetical genomics approach

*Cassidy Gedling**, Department of Plant Pathology, The Ohio State University, Ohio, USA

Saranga Wijeratne, Molecular and Cellular Imaging Center, The Ohio State University, Ohio, USA

Stephanie Karhoff, Translational Plant Sciences Graduate Program, The Ohio State University, Ohio, USA

Anna Stasko, Ohio Agricultural Research and Development Center, The Ohio State University, Ohio, USA

Phytophthora sojae, causing Phytophthora root and stem rot (PRR), is one of the main pathogens of soybean across the Midwestern U.S. Race-specific *R*-genes are commonly deployed to manage *P. sojae*. However, due to a large number of pathotypes in the U.S., partial resistance is important in several regions and is thought to put less selection pressure on *P. sojae* populations. Multiple phenotypic quantitative trait loci (pQTL) for partial resistance against *P. sojae* were previously mapped in a $F_{9:11}$ Conrad x Sloan recombinant inbred line (RIL) population on chromosomes 1, 4, 16, 18, and 19. However, these regions encompass large portions of the chromosome. Our objective was to identify the molecular mechanisms contributing towards partial resistance using an expression QTL (eQTL) approach. Thus, the overall goal of this study was to reduce the list of candidate genes underpinning pQTL that contribute to resistance in Conrad using a new approach that incorporates functional genomics. A subset of 93 RILs from the Conrad x Sloan population were inoculated with isolate 1.S.1.1 using the tray test method. Tissue at the inoculation site was collected 24 hours after inoculation (hai) from both mock and inoculated samples. RNA was extracted from a pool of 10 seedlings and sequenced using Illumina Hi-seq. In the inoculated treatment, 7,048 eQTLs were identified with 55 of those classified as *cis*, 6,953 as *trans*, and 41 unclassified. Fourteen eQTL hotspots were identified with the majority of hotspots classified as *trans*. Fifty-five eQTL are derived from genes located in the previously identified pQTL 1, 4, 16, 18, 19-1, 19-2 and 19-3. Analysis of regulatory networks and modules of expression affecting the resistance phenotype, transcriptional networks, SNPs, and eQTLs are in progress. Putative genes regulating co-expression networks will be validated in our functional gene analysis pipeline.