



## North Central Soybean Research Program

### **Increasing profits through genetic resistance to SDS**

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Sudden death syndrome (SDS) is an important disease of soybean and was estimated to cause annual losses averaging 34 million bushels each year from 2006-2010. SDS has recently moved north and has now become a major concern in MG I and II growing regions. Resistant varieties are the most effective management option for growers to control this disease. Some varieties show good resistance to SDS, however, the inheritance of this resistance is complex because it is controlled by many genes. The research in this proposal is focused on improving our understanding of the genetic basis of resistance to SDS, which should help breeders become more efficient in developing new, high yielding, SDS resistant varieties. Growers will see the benefits of this project through increased SDS resistance in private and public varieties.

### **Project Objectives**

1. Map the locations on chromosomes of genes that confer resistance to SDS and develop molecular markers linked to resistance genes;
2. Confirm the effects of mapped genes so they can be bred into elite soybean breeding lines as part of the breeding proposal; and
3. Identify genes related to SDS resistance by gene expression profiling soybean roots and leaves.

### **Summary of Results**

The goal of this objective was to identify genes breeders can be used to successfully improve SDS resistance. All genes were detected in at least one environment, resulting in their confirmation.

With the exception of lines in the CHR18 population, lines with a resistance gene had a reduced average disease severity, disease incidence, and/or disease severity compared to lines with the susceptible allele. cqSDS-001 was detected in the LD02-4485, LD01-5907, and LD00-3309 backgrounds and within multiple environments. The chr 1 resistance allele from PI507371 significantly reduced SDS foliar symptoms within three environments and across all environments. While

differences between lines containing the resistance alleles and lines containing the susceptible alleles were observed, they were often minor. It is possible these genes would have a greater effect on resistance in different genetic backgrounds or within different environments. Additionally, cqSDS-001 and CHR10 were detected in populations and environments where symptom development was minor. Nonetheless, yield loss can still occur when symptoms are mild.

The genetic backgrounds used in this study were selected because they are characteristic of high yielding, SCN resistant MG II-IV cultivars grown across Illinois and other soybean growing states in the midwestern USA. LD02-4485, LD01-5907, and LD00-3309 are also moderately resistant to SDS, and this could have been another barrier to obtaining good foliar symptoms and an explanation for the small effects observed for the genes within the populations. These three backgrounds contain rhg1 for SCN resistance, and this genetic region also has been associated with improved resistance to SDS. For the most part, we were able to consistently achieve symptoms and collect leaf scorch data in the susceptible background, Spencer. In future SDS confirmation studies, it may be more effective to select high-yielding genetic backgrounds that have been previously evaluated to be moderately susceptible or susceptible to the disease.

In some environments, SDS symptoms were most likely present, but we could not collect reliable data due to the presence of other pathogens. Several other fungal diseases of soybean including brown stem rot (causal agent-*Phialophora gregata*), red crown rot (*Cylindrocladium parasiticum*), charcoal rot (*Macrophomina phaseolina*), and stem canker (*Diaporthe phaseolorum* var. *meridionalis*, *Diaporthe phaseolorum* var. *caulivora*) have foliar leaf scorch symptoms similar to SDS. Destructive sampling is often the only way to tell the diseases apart, which is not necessarily feasible in field experiments with many locations, replications, and entries. Additionally, it can be common for plants to be infected with several other pathogens that produce symptoms that make the visual diagnosis of SDS difficult or impossible. This is especially the case when SDS symptoms appear late in the growing season around the R7 growth stage or at a lower position a plant's canopy. The Shawneetown, IL location often had other foliar symptoms that confounded our SDS ratings. The complex nature of screening for resistance to SDS demonstrates the importance of extensive multi-environment field experiments in effectively breeding for enhanced SDS resistance.

The genes evaluated in this study could be used to pyramid resistance genes into elite cultivars for more durable resistance. While the genetic mechanisms of the genes we evaluated are unknown, stacking the two distinct SDS resistance mechanisms, resistance to root rot and leaf scorch, could also be a strategy to increase resistance. Although stacking multiple genes into a single cultivar is a time

consuming endeavor, MAS improves efficiency and reduces the time needed to do so. In the future, predictive modeling in combination with high throughput genetic data could conserve the resources and time needed to incorporate multiple resistance genes into elite cultivars. It would also help capture the resistance of multiple small effect genes.

There are numerous challenges a breeder must overcome to improve resistance to SDS in their programs. These include difficulty in accurately evaluating symptoms and the complex genetic nature of resistance. Nonetheless, genetic resistance remains critical in limiting the yield loss caused by SDS. This study indicates there are several sources including PI567374 and PI507371 a breeder can use to enhance SDS resistance in their program. It also highlights both the necessity and complicated nature of multiyear field testing to evaluate symptoms, and the importance of evaluating genes across multiple genetic backgrounds prior to incorporating them into a breeding program.

It is expected that advances in high throughput genotyping and phenotyping will offer better insight into the complexity of sudden death syndrome and will ultimately result in improved cultivars for producers. In the meantime, implementing MAS using confirmed genes such as cqSDS-001 and CHR1 and the continued evaluation of mapped genes are effective methods to mitigate losses due to this devastating disease.