Project Abstract

Project Title:	Developing FHB resistant durum wheat germplasm	
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Durum lines that are genetically resistant to FHB are critical to continued production of this important crop. Despite efforts of durum researchers and advances in recent years, there is a need to develop even more resistant cultivars. Various studies suggest that the cultivated durum genome carries either a persistent suppressor or is missing enhancers of FHB resistance. To test these hypotheses, we treated several popular durum cultivars with a chemical that removes CG methylation. These lines were advanced and tested for FHB resistance over several years in multiple field nurseries and greenhouses identifying five lines that are significantly more resistant than parental lines and checks. These lines also have significantly lower *Fusarium* damaged kernels (FDK) and DON. We crossed these lines with a durum cultivar to test the stability and inheritance of resistance. Our results, and those of the NDSU durum breeding program, indicate that the changes are heritable and backcross derived lines with resistance levels similar to the resistant parent can be identified.

Additionally, we obtained RNA sequence data from various tissues with and without Fusarium infections from three of these lines along with parental controls, to determine what changes are responsible for the enhanced resistance. A total of 25 genes with significantly altered gene expression patterns have been identified that could play a critical role in the resistance mechanism. Gene network analysis indicates that multiple genes or a single up-stream regulator may act to enhance the resistance.

The ultimate objective of this project is to enhance the resistance in durum cultivars by removal of persistent suppression mechanism. This work is complementary to other efforts to introgress resistance as it would provide a genetic background that could increase and magnify on the resistance. This project includes the following objectives which address DUR-CP Research Priorities 1-5:

1. Develop backcross derived nested association mapping (NAM) panel for the top five resistant mutants;
2. Phenotype the NAM panel for FHB and tag resistance genes with molecular markers for use in breeding and;
3. Validate altered gene expression patterns of candidate genes and characterize their impact on FHB resistance individually and in combination.

The outcomes of objective 2 and 3 will provide a more detailed understanding of FHB resistance mechanism and impact of various genes on the trait. We will utilize this understanding to identify lines from our NAM panel (objective 1) that pyramid the genes with the largest impact for germplasm release.