

**PI: Shahryar Kianian**

**PI's E-mail: [Shahryar.Kianian@ARS.USDA.GOV](mailto:Shahryar.Kianian@ARS.USDA.GOV)**

**Project ID: FY16-DU-004**

**ARS Agreement #: N/A**

**Research Category: DUR-CP**

**Duration of Award: 1 Year**

**Project Title: Enhancing FHB Resistance by Epigenetic Modification of Durum Cultivars.**

### **PROJECT 1 ABSTRACT**

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Despite concerted effort of DUR-CP researchers and advances in recent years, there is a need to develop even more resistant durum cultivars. Past attempts at transfer of resistance genes/QTLs from hexaploid sources into durum wheat have met with limited success. Various studies, including several by our groups, suggest that either the cultivated durum genome carries a suppressor of FHB resistance or is missing enhancers of resistance on D-genome chromosomes. To test these hypotheses, we treated six advanced durum breeding lines with 5-Methyl-azacytadine that removes CG methylation. The resulting lines were advanced to the M<sub>4</sub> generation and tested for FHB resistance under both the greenhouse and field conditions with 24 lines identified that show great promise having less than 20% infection as compared with 80-100% value for parental lines and checks, a significant difference. These lines were tested in multiple replications at several FHB nursery locations in 2015. The most resistant lines are now being crossed with the parental cultivars to test the stability and inheritance of resistance. Additionally, we plan to analyze the changes in DNA methylation and transcription levels. In a similar project we plan to initiate the development of deletions for portions of chromosome 2A.

The immediate objectives are:

- 1. characterize the epigenetic changes of FHB resistant durum cultivars produced by altering the DNA methylation pattern, and**
- 2. characterize durum cultivars missing portions of chromosome 2A region that may contain a FHB suppressor locus.**

The ultimate objectives of this project are to incorporate the QTL regions identified in Tunisian derived germplasm into advanced durum breeding lines, and to enhance the resistance in durum cultivars by removal of a persistent suppression mechanism.