PI: Steve Scofield **PI's E-mail: scofield@purdue.edu**

Project ID: FY12-SC-015 **ARS Agreement #: NA**

Research Category: **GDER** **Duration of Award: 1 Year**

Project Title: Engineering Improved Fusarium Head Blight Resistance.

PROJECT 1 ABSTRACT

(1 Page Limit)

Five objectives are proposed in this application. The first three follow from our previous USWBSI-supported work, while the last two represent new directions. In our current project we have demonstrated very significant roles for two pathways in wheat's resistance to Fusarium head blight (FHB). Through virus-induced gene silencing (VIGS) experiments we have shown that inactivating ethylene (ET)-signaling or signaling by receptors for pathogen-associated molecular patterns (PAMPS) causes clear conversion of resistance to susceptibility. In the case of ET-signaling, we have independently confirmed the VIGS results by treatments with chemicals that inhibit ET perception. More significantly, we demonstrated that treatment with chemicals that raise ET-signaling result in susceptible genotypes becoming significantly more resistant in assays evaluating both type I and type II resistance. Transgenic wheat plants are just now becoming available that will allow us to assess our strategies to improve FHB resistance by overexpressing the key ET-responsive transcription factors, TaERF1 and TaPEIP1, as well as the PAMP perception receptor (PPR), TaBAK1. In association with this, we propose to complete transformations with other PPRs TaBAK 2, 3, 4 and 5. The new concept we propose to address stems from recent work by others showing that polyamines are produced in wheat floral tissue during infection by *F. graminearum*. These compounds were shown to induce DON synthesis in the fungus, which is crucial for the infection spreading into neighboring spikelets. We will use VIGS to silence genes required for polyamine synthesis in FHB susceptible genotypes to see if this results in reduced fungal spreading. If this expectation is confirmed, we will transform wheat so that it stably silences polyamine synthesis in wheat spikes. Success in these objectives would yield two very different anti-FHB mechanisms that could be deployed simultaneously: one that strengthens plant resistance and the other that impedes fungal pathogenesis. Our proposed objectives are:

1. Characterize the FHB resistance of transgenic wheat plants overexpressing TaERF1, TaPEIP1, TaBAK1 and TaBRI1 in greenhouse and field assays.
2. Use VIGS to assess the roles of TaBAK2,3,4&5 in FHB resistance. If significant roles in FHB resistance are detected for any of these genes, assemble overexpression constructs and initiate wheat transformations.
3. Characterize the role of ET signaling on FHB resistance in barley.
4. Perform VIGS analysis to assess the role of host polyamine biosynthesis in the induction of deoxynivalenol (DON) production by *Fusarium graminearum* during FHB pathogenesis.

If a significant role for host polyamine synthesis is detected, assemble RNAi constructs to inhibit polyamine synthesis in the spikes of wheat, transform wheat and test the transgenics for increased resistance.