FY09 USWBSI Project Abstract

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

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Research Category: GDER Duration of Award: 1 Year

Project Title: Exploring the Role of Ethylene Signaling in FHB Resistance and Susceptibility.

PROJECT 1 ABSTRACT

(1 Page Limit)

In our current USWBSI funded project we are using virus-induced gene silencing (VIGS) assay to assess whether or not candidate genes have essential roles in type II resistance to Fusarium head blight (FHB) in wheat. VIGS analysis has indicated that the ethylene signaling pathway has a crucial role in supporting FHB resistance in wheat. Specifically, we have found that when the SAM synthase, EIN2 or ERF genes, which all act to promoter ethylene signaling, are silenced in resistant wheat plants, of the genotype Ning 7840, resistance is abolished, while silencing the CTR or ETO genes, which act to suppress ethylene signaling, had no effect on resistance.

In the experimentation proposed here, we will test whether components of the ethylene signaling pathway can be used to engineer resistance to FHB in wheat.

Objectives:

- 1. Full-length cDNAs will be obtained for the wheat SAM-synthase, EIN2 and ERF genes from Ning7840 wheat.
- 2. Transgenic plants will be generated in the susceptible Bobwhite genotype that constitutively express SAM-synthase, EIN2 and ERF as RNAi constructs. These RNAi lines will be crossed into FHB1 resistant lines. These lines will permit us to confirm that silencing SAM-synthase, EIN2 and ERF, by a method other than VIGS, abolishes FHB resistance.
- 3. Transgenic plants will be created in the susceptible wheat genotype Bobwhite that express the SAM-synthase, EIN2 or ERF cDNAs constitutively from the maize ubiquitin promoter or specifically in the lemma and palea tissues using the barley Lem1 promoter.
- 4. To seed will be collected from transgenic lines identified as expressing each construct.
- 5. T1 transgenic plants expressing each construct will be assessed for their resistance or susceptibility to FHB.
- 6. T0 transgenic lines will be crossed into FHB1 lines to see if the transgenes will augment the resistance provided by FHB1.

If successful, this work would directly serve the overall goal of the GDER research area and the USWBSI by developing novel sources of FHB resistance. Furthermore, as a transgenic strategy for FHB resistance this should be readily accepted by the regulatory agencies as the transgenes consist of naturally occurring cereal coding sequences and promoters.