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Project Title: The Role of Mating-type Genes in Pathogenicity of *Fusarium graminearum* to Wheat

PROJECT 1 ABSTRACT

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Self-fertility in *Fusarium graminearum* is regulated by a pair of mating specificity proteins called MAT1-1-1 and MAT1-2-1. The proteins interact to produce a heterodimeric transcription factor that controls cascades of genes involved in sexual development. We produced strains in which one or both of the mating specificity genes were deleted (KOs). Consistent with a previous report, mutation of both genes together caused sterility, but there was no effect on pathogenicity or toxigenicity. If just one or the other of the genes was deleted, the strains were self-sterile and heterothallic. Surprisingly however, they were also significantly reduced in aggressiveness to wheat heads and in mycotoxin production. **This new finding reveals a previously unsuspected role for the mating specificity proteins in pathogenicity.** The goal of this proposal is to test the hypothesis that MAT1-1-1 and MAT1-2-1 proteins, when present in isolation, regulate genes that negatively impact aggressiveness and toxigenicity of the heterothallic strains.

We have three objectives:

1. A comparative Illumina RNA-seq analysis of the wild type (WT) and KO transcriptomes in wheat heads, to reveal genes that are altered by activity of the heterodimeric mating specificity proteins versus by the non-dimerized forms.
2. Cytological analysis of KO transformants expressing fluorescent proteins in inoculated wheat heads, to characterize the reduced aggressiveness of the MAT1-1-1 and MAT1-2-1 specificity gene KOs in detail.
3. Produce complementation strains for each of the specificity gene KOs and confirm function in aggressiveness to wheat heads.

This work may lead to development of new strategies for reducing the impact of Fusarium Head Blight disease and mycotoxin contamination in wheat, based on new knowledge of pathogen biology and genetics (PCB Goal #2). Targeting the MAT1-1-1 or MAT1-2-1 gene products may recapitulate the effect of the KOs, resulting in suppression of pathogenicity, as well as a reduction in primary inoculum. Downstream genes regulated by the non-dimerized specificity proteins may also provide novel therapeutic targets. This work addresses several stated specific research needs: 1) Discover genes for pathogenesis and trichothecene reduction. 2) Develop molecular approaches to modulate pathogen genes for disease control and mycotoxin reduction. 3) Develop new strategies to reduce sporulation on potential inoculum sources of the pathogen. And 4) Determine patterns of pathogen gene expression and protein accumulation vital to disease and trichothecene accumulation.