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Project Title: Exploring Molecular Mechanisms Conferring DON Tolerance in F. graminearum.

PROJECT 1 ABSTRACT

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Fusarium graminearum is the causal agent of Fusarium head blight (FHB) in the US. It also is a producer of mycotoxin DON, a potent inhibitor of eukaryotic protein synthesis. *TRI12* and *TRI101*, two *TRI* cluster genes, are involved in DON tolerance but the *tri12* and *tri101* mutants still produce DON. Other mechanisms must exist in *F. graminearum* and confer DON-tolerance in the *tri101* and *tri12* mutants. The same mechanisms may be responsible for the protection against DON in fungi such as *F. verticilloides* that are closely related to *F. graminearum* but lack the *TRI* genes.

In this study, we aim to identify and characterize other non-*TRI* cluster genes that are involved in protecting *F. graminearum* against trichothecene mycotoxins. In objective 1, we will identify and characterize genes that are specifically induced by DON (named *DIG* genes). Some of these *DIG* genes may be directly involved in DON detoxification or avoidance. For objective 2, we will generate and identify *DOS* mutants that have increased sensitivity to DON. Genes tagged in the *DOS* mutants will be characterized. Some of the *DOS* genes may be important for self-protection or DON-tolerance in *F. graminearum*. In objective 3, we will express the *DIG* and *DOS* genes in the budding yeast and determine their functions in conferring DON tolerance to yeast cells. Results from proposed study will lead to the identification of non-*TRI* genes that are important for DON tolerance in *F. graminearum*. These genes can be used to generate transgenic wheat lines with improved resistance to FHB.

This research is directly relevant with the USWBSI's goal on developing possible effective scab control measures. It addresses priority 2 of PBP: 'Develop new strategies for reducing impact of FHB disease and mycotoxin contamination'. To our knowledge, the underlying mechanisms for self-protection against many mycotoxins are not well studied in fungal pathogens. Even for HC-toxins that were studied extensively, the exact mechanism for protecting *Cochliobolus* against this potent HDAC inhibitor is not clear. We will seek funding from other agencies to further characterize the *DOS* and *DIG* genes identified in this study.