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PROJECT 1 ABSTRACT

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Fusarium head blight caused by *Fusarium graminearum* is an important disease of wheat and barley. Losses are due to reduction in yield and mycotoxin contamination. In a previous study, the *F. graminearum* transducin-beta like gene *FTL1*, a component of a well-conserved protein complex, was found to be essential for plant infection. The *FgHOS2* gene encodes a histone deacetylase (HDAC) that is a key component of the *FTL1* complex. In this study, we aim to determine the role of the *FgHOS2* HDAC gene in plant infection and identify genomic regions and genes regulated by *FgHOS2*. The first objective is to generate and characterize the Δ*Fghos2* deletion mutant and test the inhibitory effects of several cyclic peptide HDAC inhibitors. HDAC inhibitors have been extensively explored for pharmaceutical applications. If *FgHOS2* plays a critical role in plant infection, HDAC inhibitors and corresponding nonribosomal peptide synthase (NRPS) genes have the potential to be used for developing novel scab control strategies. For the second objective, we will identify genomic regions and genes regulated by *FgHOS2* by ChIP-chip and microarray analysis. Some of these *FgHOS2*-regulated genes may be required for plant infection and distributed in chromosomal regions that are not expressed during saprophytic growth. If ChIP-chip data from this study confirm the regulatory role of chromatin modifications in fungal pathogenesis, interfering with chromatin structures can be explored as a disease control measure.

Overall, experiments proposed in this project are relevant to the FY07 research priorities of the Pathogen Biology and Genetics (PBG) research areas on 'Develop new strategies for reducing the impact of FHB and associated mycotoxin contamination in barley and wheat' and 'Characterize plant-fungal interactions in plant lines being developed by researchers in the USWBSI'. Results from this study will provide important information on the role of the *FgHOS2* HDAC gene and chromatin modifications in plant infection, and may ultimately lead to the development of new approaches for controlling scab disease by interfering with fungal chromatin structures.