Fusarium diseases are exacerbated by the production of harmful mycotoxins. For example, Fusarium head blight (scab) caused by numerous spp. (e.g. *F. graminearum*, *F. culmorum*, and *F. sporotrichioides*) is associated with production of trichothecene and zearalenone mycotoxins and likely other, uncharacterized metabolites. Genes encoding fungal mycotoxins (including trichothecenes and zearalenone) are found in co-regulated genetic clusters. Recent progress in fungal toxin clusters has suggested that these clusters are globally regulated by higher order chromatin conformation. To address this possibility in Fusarium, we propose to

1. Discern if LaeA, a putative activator of heterochromatin, will regulated *F. graminearum* toxin production
2. Inactivate genes involved in heterochromatin formation in *F. graminearum*
3. Analyze heterochromatin mutants in *F. graminearum* for effects on toxin production

Our first goal is to determine if trichothecene and zearalenone gene clusters are regulated by heterochromatin. If yes, as initial results suggest, we will follow up with designing methods to modify chromatin to a toxin silent state. These efforts will help in developing efficient control measures to minimize this persistent disease problem and spread in the USA and other parts of the world. This work meets the priorities of the USWBSI research area, Pathogen Genetics and Genomics (PGG). The specific priorities in the PGG research area that this work meets are: Identify and characterize genes that control important pathogen traits; and Identify and characterize important pathogen gene and protein expression profiles, regulatory networks, and developmental or metabolic pathways.