0203-TU-028 Modification of the Ribosomal Target to Enhance Resistance to Trichothecene Mycotoxins.

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PROJECT ABSTRACT
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Trichothecenes are a highly diverse class of toxic, sesquiterpenoid secondary metabolites that are produced mainly by plant pathogenic fungi. The contamination of important agricultural products, such as wheat, barley or maize with the trichothecene mycotoxin deoxynivalenol (DON) due to infection with *Fusarium graminearum* and *F. culmorum* is a worldwide problem. Trichothecene mycotoxins act as inhibitors of eukaryotic protein biosynthesis and their formation is considered to be avirulence factor in a number of plant diseases. Investigation of trichothecene resistance in the yeast *Saccharomyces cerevisiae* indicated that semi-dominant toxin resistant yeast mutants contain either alterations in the target of trichothecenes, the ribosomal protein L3 or show increased drug efflux due to overexpression of a membrane transporter protein encoded by the *PDR5* gene. Overexpression of the *Saccharomyces cerevisiae PDR5* gene or the *Fusarium sporotrichioides TRI*101 gene, which encodes a trichothecene 3-O-acetyltransferase in transgenic tobacco conferred increased tolerance to 4,15-diacetoxyscirpenol (DAS). Ribosomal protein L3 is a highly conserved ribosomal protein that participates in the formation of the peptidyltransferase center. The trichothecene mycotoxin, trichodermin, which belongs to the general class of 12, 13-epoxytrichothecenes, is known to inhibit peptide bond formation by binding to the peptidyltransferase center. Mutations in the *RPL3* gene, encoding ribosomal protein L3 were initially identified in yeast by conferring resistance to trichodermin. Further evidence to suggest that mutations in L3 could confer resistance to fungal toxins is described through work with other trichothecenes. These studies point to the fact that trichothecene resistance may be attained through a mutation in L3 that would result in decreased fungal infection. Therefore, expression of mutant forms of L3 may contribute to resistance to fungal pathogens, such as *Fusarium* head blight (scab) in wheat. Our primary goal in this project is to determine whether trichothecene resistance contributes to resistance towards *Fusarium* head blight (scab) in wheat and using yeast as a model system, to identify trichothecene resistant forms of ribosomal protein L3. Using the powerful reagents developed in our prior studies, we are now in a unique position to determine if trichothecene resistance contributes to resistance to *Fusarium* head blight in wheat. Information gained from these studies could be used to design novel strategies to combat wheat scab and improve *Fusarium* resistance of cereals.