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Project Title: Cytological and physiological role of deoxynivalenol in Fusarium head blight.

PROJECT 1 ABSTRACT

(1 Page Limit)

Several lines of evidence, both genetic and physiological, indicate that the trichothecene mycotoxins, especially deoxynivalenol (DON), have a role in pathogenesis of Fusarium head blight. DON is postulated to cause injury to host tissues and to contribute to the virulence of head blight pathogens. DON is known to be a potent inhibitor of protein synthesis. However, its physiological effects on plant tissues have not been well-elucidated. Using detached green leaf segments of barley, we have learned that DON causes tissues to lose all chloroplast pigments (the tissues turn white) if DON-treated specimens are incubated in light. Over 4-5 days, the toxin also induces characteristic alterations in the ultrastructure of chloroplasts as well as loss of electrolytes. We propose here to learn if similar changes are induced by DON in the lemma and floret tissues. Tox+ and tox- strains of *F. graminearum* will be used to confirm if alterations are attributable to DON. As a second objective, we will determine if programmed cell death (PCD) is involved in DON-induced alterations in leaf tissues. DON is known to induce PCD in animal tissues. We will: a) determine if DON induces cytological changes in plant nuclei characteristic of PCD (fragmentation or change in nuclear shape and fragmentation of DNA as indicated by positive TUNEL staining reactions); b) test whether known inducers of PCD (cycloheximide, fumonisin, mastoparan) produce alterations similar to those induced by DON; c) test whether known inhibitors of PCD (such as Ac-DEVD-CHO) inhibit DON-induced loss of chloroplast pigments; and d) determine if transgenic plants containing anti-PCD genes (from animals) resist effects of DON. By defining the physiological role of DON in Fusarium head blight, the proposed research will help in the design of disease control strategies directed toward limiting the toxic activities of DON and related trichothecene toxins in diseased tissues.