FOOD SAFETY, TOXICOLOGY 
AND UTILIZATION OF 
MYCOTOXIN-CONTAMINATED 
GRAIN
WHEAT KERNEL BLACK POINT AND FUMONISIN CONTAMINATION BY *FUSARIUM PROLIFERATUM*.
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ABSTRACT

Fumonisins are mycotoxins produced by several *Fusarium* species, especially *Fusarium proliferatum* and *Fusarium verticillioides*, which are common pathogens of maize worldwide. Consumption of fumonisins has been shown to cause a number of mycotoxicoses, including leucoencephalomalacia in horses, pulmonary edema in swine, and liver cancer and neural tube defects in experimental rodents. Consumption of fumonisin-contaminated maize also has been associated epidemiologically with human esophageal cancer in some areas of the world where maize is a dietary staple. Although *F. proliferatum* is a major cause of maize ear rot, this species also is a minor component of the wheat head blight complex worldwide and has been associated with incidents of black point disease of wheat kernels in the USA. The major aim of the present study was to characterize nine *F. proliferatum* strains from wheat from Nepal for ability to cause wheat kernel black point under greenhouse conditions and for fumonisin contamination of infected kernels. For comparative purposes, the study also included three *Fusarium* strains isolated from US maize: two *F. proliferatum* strains and one *F. verticillioides* strain. Fungal strains were applied by spray or injection of macroconidia to spikes of five wheat cultivars (two soft white spring wheats, one hard red spring wheat, and two durum wheats). All strains produced kernel discoloration and black point, and most strains had some effect on kernel weight and germination. Most strains also produced fumonisins in kernels, but at relatively low levels of less than 10 μg/g (combined fumonisin B1, B2 and B3) as determined by liquid chromatography-mass spectroscopy. However, one strain from Nepal produced high levels of more than 100 μg/g of fumonisins in kernels. These preliminary data indicate a potential for fumonisin contamination of wheat infected with *F. proliferatum*. Surveys are underway to determine the natural occurrence of *F. proliferatum* and fumonisins in US wheat with black-point disease. Those interested in contributing black-point wheat samples for fumonisin analysis are encouraged to contact the corresponding author.
TISSUE DISTRIBUTION AND PROINFLAMMATORY CYTOKINE INDUCTION BY THE TRICHOTHECENE DEOXYNIVALENOL IN THE MOUSE: COMPARISON OF NASAL VS. ORAL EXPOSURE.

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ABSTRACT

Ingestion of the trichothecene mycotoxin deoxynivalenol (DON), a common contaminant of cereal grain, causes adverse effects on growth and immune function in experimental animals and thus poses a health risk to consumers. However, relatively little is known about the risks posed to farmers and grain handlers who are exposed to DON via inhalation of contaminated grain dust. The purpose of this research was to test the hypothesis that DON will distribute in tissues and induce proinflammatory cytokines to a greater extent following nasal exposure than by oral challenge. B6C3F1 mice were treated with a single dose of DON (5 mg/kg) either by nasal instillation or oral gavage. After 0, 15, 30, 60, 120 minutes, mice were euthanized and serum, spleen, liver, lung and kidney samples were collected. An ELISA was devised for the sensitive measurement of DON burden in serum and tissue. In both oral and nasal treatment groups, DON serum and tissue concentrations peaked after 15 minutes and declined by 75 to 90% after 120 minutes. However, DON concentrations were 1.5 to 3.5 times higher in serum and other tissues of mice exposed by the nasal route at all time points assayed as compared to orally exposed mice. The functional significance of different DON tissue concentrations was assessed by measuring the expression of the pro-inflammatory cytokines IL-6, TNFα, and IL-1β in spleen. As expected, oral exposure to DON induced cytokine mRNA expression after 60 and 120 minutes. However, mRNAs for these cytokines were 2 to 3 times higher in spleens of mice exposed to DON by the nasal route. Taken together, these toxicokinetic and functional data indicate that DON is potentially more toxic when inhaled than when ingested. Furthermore, they suggest that adverse human health effects could potentially result from inhalation of DON-contaminated grain dust.

ACKNOWLEDGEMENTS

This material is based upon work supported in part by the U.S. Department of Agriculture under Award No. 59-0790-4-119. This is a cooperative project with the U.S. Wheat and Barley Scab Initiative. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the authors and do not necessarily reflect the view of the U.S. Department of Agriculture.
GASEOUS OZONE TREATMENT OF FUSARIUM-INFECTED MALTING BARLEY.
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ABSTRACT

Fusarium head blight (FHB) or scab, caused predominantly by Fusarium graminearum, is a serious disease problem of barley in the Northern Plains area. The fungus is known to produce the mycotoxin deoxynivalenol (DON) in infected grains which poses safety concerns for human and livestock. The utilization of Fusarium-infected grains and the persistence of mycotoxin in malting barley may lead to decreased malt quality. The only effective control is testing and diversion or dilution. Our initial laboratory test of gaseous ozone treatments (GOT) using Fusarium-infected grains of malting barley, Robust, (with 1.27 g/g DON) at 11 and 26 mg/g O3 for 15-30 min showed a significant decrease (24-36%) in Fusarium survival (Kottapalli et al. 2005). In our present work, we tested the effectiveness of ozone treatment on Fusarium graminearum (NRRL R-6574) in culture (potato dextrose broth) at 0 and 2 days after inoculation (dai). We also extended the exposure time to 2 hrs. using naturally infected malting barley grain samples (20 g) with 2 ppm DON and under steeping condition. Fusarium conidia did not survive the GOT at 0 dai and no apparent further growth in mycelia (i.e. change in fungal biomass) was observed with GOT at 2 dai even with 5 days of incubation of culture after the O3 treatment. Fusarium survival in ozonated grains with 0 ≤ 0.1 and 2 ppm DON significantly decreased from 23% to 8% and from 49% to 12%, respectively, without affecting germination. Under steeping condition, O3-treated grains (with 2 ppm DON) that were plated on HPDA and incubated for 3-5 days did not show any Fusarium growth. Our initial results strongly suggest that Fusarium cannot survive if GOT is prolonged to at least 2 hrs at the same dosage. Hence, the treatment can effectively reduce Fusarium survival in stored grains. Moreover, exposure of grains to O3 during steeping for at least 2 hrs could eliminate Fusarium.