

Defining gene's role may lead to prevention of dangerous corn toxin

WEST LAFAYETTE, Ind. - Discovery that a specific gene is integral to both fungal invasion of corn and development of a potentially deadly toxin in the kernels may lead to ways to control the pathogen and the poison.

Purdue University researchers evaluated the fungal gene ZFR1 and found that it is vital to the process of the fungus growing on corn kernels. Production of the toxin decreased when the scientists disabled the gene.

At certain levels, the toxin can cause illness in humans and most domestic livestock. Horses and pigs are at particular risk and can develop fatal diseases by ingesting feed containing one of a group of toxins called fumonisins (few-mahn-ah-sins). About \$40 million of the U.S. corn crop is lost annually due to presence of these toxins, according to experts.

"Our main research question has been what triggers toxin production when the fungus attacks the corn kernel; it appears that kernel starch plays an important role," said Charles Woloshuk, a Purdue plant pathologist. "When ZFR1 is deleted, the resulting mutant fungus has a problem transporting sugars that are produced from the degradation of kernel starch."

The resulting sugars must be transported to cells as fuel for other biochemical processes.

"The pathogen - the fungus *Fusarium verticillioides* - has a number of putative sugar transporter genes that are expressed during its growth on kernels and toxin production," Woloshuk said. "Disruption of ZFR1 also affects expression of the sugar transporter genes."

Woloshuk and his colleague, Bert Bluhm, now at the University of Arkansas, report in the current issue of *Molecular Plant Pathology* that when the gene ZFR1 is turned off, it reduces manifestation of genes involved in production of the most prevalent and dangerous fumonisin, FB1.

The researchers studied ZFR1 regulation of fungal growth and toxin production in the starch-rich areas of corn kernels and the conversion of starch to glucose, glucose recognition and the expression of sugar transporter genes. From this information, Woloshuk and his team identified a specific sugar transporter, FST1 (fusarium sugar transporter1), that is necessary for FB1 production.

Although FST1 is required for FB1 production, it is not involved with the fungus infecting corn kernels. This led the scientists to hypothesize that FST1 acts as a molecular sensor necessary for toxin production.

Kernels with lower starch content, most notably immature kernels, don't support toxin production, Woloshuk said. This is evidence that the kernel makeup dictates how this pathogen controls toxin production.

Corn and fungal growth were unaffected when the sugar transporter gene was disrupted, but toxin production on the kernels was cut by about 82 percent, Woloshuk said.

When fusarium invades corn in the field, it causes an ear rot disease. Even knowing that ear rot is present doesn't help identify corn containing toxin because obvious signs of the fungus don't correlate with presence of toxins. The only way to confirm toxin is present is to test for it. Testing is so expensive, however, that it usually isn't done unless the disease is highly evident.

Weather and insect damage impact development of a variety of fungi and toxins and also influence the level of poisons that are present. Toxins are more likely to develop in corn when hot, dry weather is followed by highly humid or wet weather.

The group of toxins associated with varieties of fusarium species are known as mycotoxins. Some clinical evidence links these toxins with certain human cancers.

Grains grown for cereal and feeds are susceptible to one or more of the fusarium fungi species. Wheat and barley attacked by one of the species closely related to *Fusarium verticillioides* can develop head blight and accumulate mycotoxins, causing billions of dollars in crop losses worldwide.

Further study is needed because the researchers still don't know what triggers the biochemical process that regulates ZFR1 and consequently leads to toxin production, Woloshuk said. The scientists also are investigating the sugar transporter genes to discover if they have other roles in the fungus and what molecular interactions between the fungus and the plant allow infection and toxin production.

"We're closer to finding some of the triggers in corn that assist the fungus in toxin production," Woloshuk said.

The other researchers involved in this study were Department of Botany and Plant Pathology doctoral student Hun Kim and Robert Butchko of the USDA National Center for Agricultural Utilization Research Service in Peoria, Ill. Bluhm is a former graduate student in Woloshuk's laboratory who recently joined the University of Arkansas faculty as an assistant professor.

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