# Gene guards grain-producing grasses so people and animals can eat

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Research by this trio of Purdue and USDA-ARS scientists is the first to show that a gene in all grasses, including important grain-producing plants, stops infection by a highly destructive fungus. A resistance gene was first discovered in corn, according to Guri Johal (front), Steve Schofield (at left) and Michael Zanis. Their findings were published by the National Academy of Sciences. Credit: Purdue Agricultural Communication photo/Tom Campbell

Purdue University and USDA-Agricultural Research Service scientists have discovered that a type of gene in grain-producing plants halts infection by a disease-causing fungus that can destroy crops vital for human food supplies.

The research team is the first to show that the same biochemical process protects an entire plant family - grasses - from the devastating, fungal pathogen. The naturally occurring disease resistance probably is

responsible for the survival of grains and other grasses over the past 60 million years.

The findings will stimulate the design of new resistance strategies against additional diseases in grasses and other plants. Grasses' ability to ward off pathogens is a major concern because grasses, including corn, barley, rice, oats and sorghum, provide most of the calories people consume, and some species also increasingly are investigated for conversion into energy.

A resistance gene, first discovered in corn, and the fungal toxin-fighting enzyme it produces apparently provide a biological mechanism that guards all grass species from this fungus, said Guri Johal, a Purdue associate professor of botany and plant pathology. He is senior and corresponding author of the study published this week (Jan. 28-Feb. 1) in Early Edition, the online version of the *Proceedings of the National Academy of Sciences*. It will appear in the Feb. 5 print edition.

"We think that if the gene Hm1 had not evolved, then grasses would have had a hard time surviving, thriving or, at least, the geographic distribution would have been restricted," Johal said. "This plant resistance gene is durable and is indispensible against this fungal group, which has the ability to destroy any part of the plant at any stage of development."

In 1943 a related fungus decimated rice crops in Bengal, causing a catastrophic famine in which 5 million people starved. The same fungal group was responsible for the other two recorded epidemics in grasses in the 20th century, including the 1970 southern corn leaf blight that destroyed 15 percent of the U.S. corn crop.

The study, part of an effort to prevent future crop crises, also provides new information about the evolution of plant-pathogen interaction,

report Johal and his colleagues, including USDA-Agricultural Research Service researcher Steven Scofield and plant geneticist Michael Zanis. The findings have implications for continued survival and further evolution of grasses, which also include rye, bluegrass, reed canary grass and bamboo.

Johal and the research team began this study because they had a hunch that a genetic mechanism similar to the one protecting corn from a fungus, called Cochliobolus carbonum race 1 (CCR1), might also be at work in other grasses. They knew that all grasses had genes similar, or homologous, to Hm1, but not whether the same genetic mechanism was providing resistance against the fungus and its toxin.

To determine if the same biochemical processes were at work to prevent grass susceptibility to the fungus family, Johal and his team shut off the Hm1 homologue in some barley plants. Next, they infected the test barley with fungus.

In barley that no longer had a functioning Hm1 homologous gene, the fungus, with the help of its toxin, caused disease in the plant. The resulting tissue damage on the barley leaves was typical of maize leaf blight symptoms in corn.

Some of the research barley, which had a functioning Hm1 gene, was inoculated with the fungus. The results showed that the resistance mechanism was the same as the one that prevents the fungus' disease infection in corn.

As in corn, the Hm1-like gene produced an enzyme that disarmed the fungus' disease-causing toxin. The detoxification isolated the infection at the site where the fungus invaded. The research with the barley also showed that, as in corn susceptible to the fungus, infection isolation occurs if the fungus doesn't produce the toxin.

Now that the researchers know that Hm1 homologues in all grasses apparently trigger the same resistance to the fungal family, the next step will be to investigate how the fungal toxin facilitates disease when not degraded by Hm1.

Source: Purdue University