Grain Pest's Own Genes Turned Against It

Waking up in the morning is tough enough, much less pouring some cereal only to see an unwelcome intruder tumble out: the red flour beetle, *Tribolium castaneum.* It's a rare but unsavory occurrence, to say the least.

Nationally, such infestations cost millions of dollars in losses annually to stored grains and food products made from them. Warehouse sanitation usually keeps beetle numbers down, but severe cases can necessitate insecticide use or fumigation. The problem is that the sturdy beetle has shown a propensity to develop insecticide resistance.

But now, the very secrets to the pest's success—its genes—could prove its undoing. Aided by a genomic map of all 16,000 *T. castaneum* genes, ARS entomologist Richard W. Beeman and colleagues have begun plotting a kind of genetic sabotage on the beetle's basic life functions—from digestion to locomotion.

In a 2009 issue of *Insect Biochemistry and Molecular Biology,* Beeman and colleagues at Kansas State University-Manhattan (KSU) report on nine genes that regulate how a key biochemical building material—chitin—is used to form the red flour beetle's outer shell, or exoskeleton.

The genes encode for a group of specialized enzymes called "chitin deacetylases" (CDAs). They trim off branches of a long chain of simple sugars that make up raw chitin. Which branches get trimmed depends on where chitin is needed on a developing beetle's body and for what purpose.

"The whole process is complex and delicately controlled," says Beeman, with the ARS Grain Marketing and Production Research Center in Manhattan, Kansas.

Around leg joints, for example, chitin's branched-chain structure must be enzymatically snipped in a way that allows for flexibility and movement. But around the head and thorax, where protection of vital organs is key, a heavier, stiffer chitin deposition is needed, requiring a different form of trimming as well as the activation of specific CDA-encoding genes.

Chitin also plays a role inside the beetle by lining its midgut as a buffer against ingested pathogens and abrasives and to create a vessel for digestion.

A biotech procedure called "RNA interference" has proven critical to demarcating the genes' roles. Using the method, "We can knock out the function of any one of these particular deacetylase genes and observe whether the insect can survive and exactly how its development is disrupted in the absence of each gene," says Beeman, whose colleagues are Karl J. Kramer, ARS retired; and KSU researchers Yasuyuki Arakane, Khurshida Begum, Radhika Dixit, Yoonseong Park, and Subbaratnam Muthukrishnan.

In lab studies, the larvae of one CDA-deficient strain developed normally until becoming adult beetles, at which point improper chitin formation kept the middle leg joints from bending, making it impossible for the insects to walk, mate, or feed. In another strain, shutting off a different CDA gene prevented the insect from shedding its old skin, or exoskeleton.

Although not every CDA gene deficiency killed the beetles outright, the inability to digest a virus particle or bend a hind leg, for example, could doom such insects under natural conditions.

"The ultimate goal," says Beeman, "is to identify which genes are essential to the insect. Beyond that, you can start thinking about ways of knocking them out for pest control."

One possibility is to formulate chitin-disabling biopesticides. Another is to engineer crop plants with anti-chitin proteins that will deter beetle feeding, or perhaps entomb them in their own skins.—By **Jan Suszkiw,** ARS.

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PEGGY GREB (D269-1)

Red flour beetle, ***Tribolium castaneum.***