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Characterizing resistance to the pesticide Bt toxin.

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Abstract

Cloning and characterization of the *Bacillus thuringiensis* toxin-resistance gene *bre-5* from *Caenorhabditis elegans* has revealed it is a glycosyltransferase.

Significance and context

Bacillus thuringiensis (Bt) is a widely used bio-pesticide whose toxin genes have also been incorporated into transgenic crop plants such as cotton. When ingested by an insect, the protein toxins produced by the bacterium interact with membrane receptors in the mid-gut, forming pores in the gut wall and killing the insect. Although Bt toxins are 'environmentally friendly' in that they do not harm vertebrates, this mode of biological control carries the potential danger of insect resistance to a toxin developing through mutation and selection. Griffitts *et al*. are attempting to identify genes that, when mutated, confer resistance to Bt toxins, and to study the mode of action of the Bt toxin at the molecular level. As Cry5B, one of the Bt toxins, acts on the nematode *Caenorhabditis elegans* as well as on insects, they studied resistance in this genetically well-characterized model organism. As nematodes are themselves important crop pests, the study of toxin action on them, and their resistance mechanisms, are of considerable importance. Five previously reported *bre* (Bt resistance) genes confer resistance to Cry5B when mutated. Here, Griffitts *et al*. report the molecular mechanism of resistance for one of these genes.

Key results

Griffitts *et al.* mapped the Bt-resistance gene *bre-5* to the right end of chromosome IV of *C. elegans*. They then found that toxin-resistant *bre-5* mutants could be 'rescued' to toxin susceptibility by a cloned fragment containing a gene, presumed to be wild-type *BRE-5*, with sequence similarity to mammalian glycosyltransferases. They isolated the corresponding cDNA, which encodes a protein, BRE-5, of 322 amino acids. Sequence analysis predicted that BRE-5 belongs to the β-1,3-galactosyltransferase family of enzymes, which add galactose to proteins and lipids. BRE-5 also showed significant similarity to the *Drosophila* protein Brainiac, which influences ligand-receptor interactions in the Notch signaling pathway. The greatest homology to mouse and human β-1,3-galactosyltransferase was found in the catalytic domain of BRE-5. Griffitts *et al.*used RNA interference (RNAi) to confirm that loss of this galactosyltransferase leads to toxin resistance. The authors hypothesize that BRE-5 acts in the

glycosylation of proteins or lipids that will be exposed at the gut surface, forming a carbohydrate structure that is necessary for toxin binding. Fluorescence-based expression experiments and co-localization experiments suggested that the Bt toxin binds to the nematode gut via receptors and is then endocytosed.

Links

Supplementary data to *Science* **293:**860-864 is freely available.

Conclusions

The identification and characterization of *bre-5* as a Bt-resistance gene proves the importance of carbohydrate receptors in Bt toxicity.

Reporter's comments

It has previously been observed that one toxin can bind to at least two receptors that are completely unrelated in sequence. From their results,the authors hypothesize that these disparate receptors are able to bind the same toxin through a common carbohydrate motif. The identification and characterization of different types of Bt toxin resistance if they arise in insects and other invertebrates will be essential for the long-term effectiveness of the toxin as a bio-pesticide. The study of Bt toxins and their mode of action is also of interest for other potential applications; the results of Griffitts *et al*. will help in the development of engineered toxins and the development of transgenic crop plants.

Table of links

Science

Supplementary data to *Science* **293:**860-864

References

1. Griffitts JS, Whitacre JL, Stevens DE, Aroian RV: Bt toxin resistance from loss of a putativecarbohydrate-modifying enzyme. Science. 2001, 293: 860-864. 0036-8075

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