# *BACILLUS THURINGIENSIS* TOXINS TRIGGER RECEPTOR SHEDDING FROM GYPSY MOTH MIDGUT CELLS

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## ABSTRACT

The mechanism of action of the Cry1 insecticidal proteins produced by *Bacillus thuringiensis* (Bt) begins with the processing of these proteins in the larval gut. After proteolytic activation, the Bt toxins bind to specific midgut receptors and insert into the membrane of the gut epithelial cells, causing insect death. Aminopeptidases, cadherins, other proteins and glycoconjugates have been found to function as Bt toxin receptors in different insect species. However, what happens after the toxin-receptor interaction remains controversial. Based on earlier studies it was proposed that the Bt toxins form pores that cause cell swelling and death by colloidal osmotic lysis. More recent studies suggest that the binding of the Bt toxin to its receptor induces an aberrant signal transduction response that kills the gut cells. In this study we looked at the fate of aminopeptidase N (APN) in gypsy moth larvae after challenge with Bt toxins and secondary messengers including AMP and cyclic AMP. Bt toxins induced rapid shedding of APN from the gut epithelial cells in a dose- and time-dependent manner. Moreover, the release of APN was also induced by AMP but was blocked by administering cyclic AMP along with the toxin. These findings show that Bt toxins trigger receptor shedding and suggest an involvement of a secondary messenger signaling pathway in the dysfunction and death of the intestinal epithelial cells.