The major histopathological effects caused by Btk HD-1 δ-endotoxin on the midgut of *H. armigera* were disruption of the acellular peritrophic membrane, brush border membrane vesicles and epithelial cells.

Similar histopathological effects were observed of Bti δ-endotoxin on the midgut of *A. aegypti* larvae.

Binding of Bti δ-endotoxin to the midgut section of *A. aegypti* larvae occurred mainly in the brush border and to some other parts of the epithelial cells.

The binding of the Btk HD-1 δ-endotoxin to the brush border membrane of *H. armigera* was qualitatively determined by dot blot immunoassay.

A 120 kDa polypeptide was identified as a receptor protein from *H. armigera* brush border membrane for the cry IAc type of insecticidal crystal proteins of Btk.

Aminopeptidase-N activity in the brush border membrane preparation was measured as the hydrolysis of leucine-p-nitroanilide. The specific activity of aminopeptidase-N was 35 units at pH 7.5, while specific activity of phosphatase was 5 units at pH 9.5.

Aminopeptidase-N activity was inhibited by the Zn$^{++}$ chelating agent, 2,2'-dipyridyl and 1,10-phenanthroline at 10 mM.

Addition of CryIAc δ-endotoxin to the brush border membrane preparation, increased aminopeptidase-N activity upto 60 ng, but further increase in toxin lowered the activity to its original level.