Genetics of Bt toxins and its Mode of action: In the early 1980s, it was established that most genes coding for the ICPs reside on large transmissible plasmids, of which most are readily exchanged between strains by conjugation [44].Experimental data suggest that the C-terminal and middle domains of the toxin are involved in epithelial cell receptor binding and structural functions, while the N-terminal domain is primarily involved in ion channel and pore formation[56].and can be summarized in the following stages: 1) ingestion of sporulated Bt and ICP by an insect larva; 2) solubilization of the crystalline ICP in the midgut; 3) activation of the ICP by proteases; 4) binding of the activated ICP to specific receptors in the midgut cell membrane; 5) insertion of the toxin in the cell membrane and formation of pores and channels in the gut cell membrane, followed by destruction of the epithelial cells [53].In the midgut of the target larva the parasporal crystalline ICP is dissociated to the protoxin form, and the protoxin is then activated to a biologically active holotoxin by the proteolytic enzymes and specifically the alkaline environment of the gut [55]..Since these initial studies, numerous ICP genes have been cloned, sequenced and used to construct Bt strains with novel insecticidal spectra [45].?-endotoxins are encoded by the Cry and Cyt genes These genes become active during sporulation because they are controlled by a dedicated RNA polymerase that is also synthesized specifically while spores areforming.Pore or ion channel formation occurs after the binding to the receptor and insertion of the N-terminal domain into the membrane, whereby the regulation of the transmembrane electric potential is disturbed.