

4 Biochemical Mechanisms of Resistance

4.1 Altered toxin binding

Altered toxin binding is a common biochemical mechanism by which insects develop resistance to Bt toxins. This mechanism involves changes in the receptors on the insect midgut cells, where Bt toxins typically bind. Mutations or modifications in these receptors can reduce the binding affinity of the toxins, thereby diminishing their efficacy. This resistance mechanism has been observed in various insect species and poses a significant challenge in managing Bt resistance. Due to receptor alterations, the toxins cannot effectively bind to their target sites, reducing their toxic effect and allowing insects to survive exposure to Bt toxins. The widespread occurrence of this mechanism makes it a crucial area of study (Jurat-Fuentes et al., 2021).

4.2 Enhanced detoxification enzymes

Insects also resist Bt toxins through enhanced detoxification enzyme activity, which is another critical biochemical resistance mechanism. These enzymes can metabolize and neutralize Bt toxins before they reach their target sites, thereby reducing the toxins' effectiveness. Enhanced detoxification capacity is often associated with the overexpression or increased activity of these enzymes.

4.2.1 Cytochrome P450 monooxygenases

Cytochrome P450 monooxygenases (P450s) play a crucial role in detoxifying xenobiotics, including Bt toxins. These enzymes are involved in the metabolic detoxification of a wide range of substances, such as phytochemicals, insecticides, and environmental pollutants. The overexpression of P450s is closely linked to the enhanced detoxification capabilities observed in resistant insects. P450s can work through multiple pathways to detoxify xenobiotics, and their regulation involves complex networks of transcription factors and signaling pathways. Studies have shown that P450s can be induced through specific signaling pathways in resistant insects, significantly enhancing their detoxification capabilities (Lu et al., 2020; Nauen et al., 2021).

4.2.2 Glutathione S-transferases

Glutathione S-transferases (GSTs) are another group of critical detoxification enzymes involved in the resistance to Bt toxins. GSTs catalyze the conjugation of glutathione to toxic substances, making these substances more water-soluble and easier to excrete. Although the role of GSTs in Bt toxin resistance is less documented compared to P450s, studies indicate that they play a significant role in detoxifying other insecticides and xenobiotics. Therefore, GSTs' potential role in Bt resistance is an important area for further investigation (Jurat-Fuentes et al., 2021).

4.2.3 Esterases

Esterases are enzymes that hydrolyze ester bonds in various substrates, including insecticides. These enzymes can detoxify Bt toxins by breaking down their ester linkages, thereby reducing the toxins' potency. In resistant insect populations, the expression or activity of esterases is often significantly increased, further accelerating the detoxification process of Bt toxins. Esterases, like P450s and GSTs, contribute to the enhanced detoxification capabilities observed in resistant insects, and their role in Bt resistance is increasingly recognized (Nauen et al., 2021).

4.3 Changes in gut microbiota

Changes in the gut microbiota of insects can also play a critical role in Bt toxin resistance. The gut microbiota not only affects the overall health and metabolic capacity of the host insect but can also directly or indirectly participate in the detoxification of xenobiotics. Research has shown that changes in the composition and function of the gut microbiota can enhance insect resistance to Bt toxins in several ways. Some microbes can directly degrade Bt toxins, reducing their toxicity, while others may enhance the host's immune response and detoxification enzyme activity, further increasing resistance. Although this area of research is still developing, preliminary studies suggest that gut microbiota plays an essential role in Bt resistance, and future research will provide more scientific insights into this mechanism (Pinos et al., 2021).