

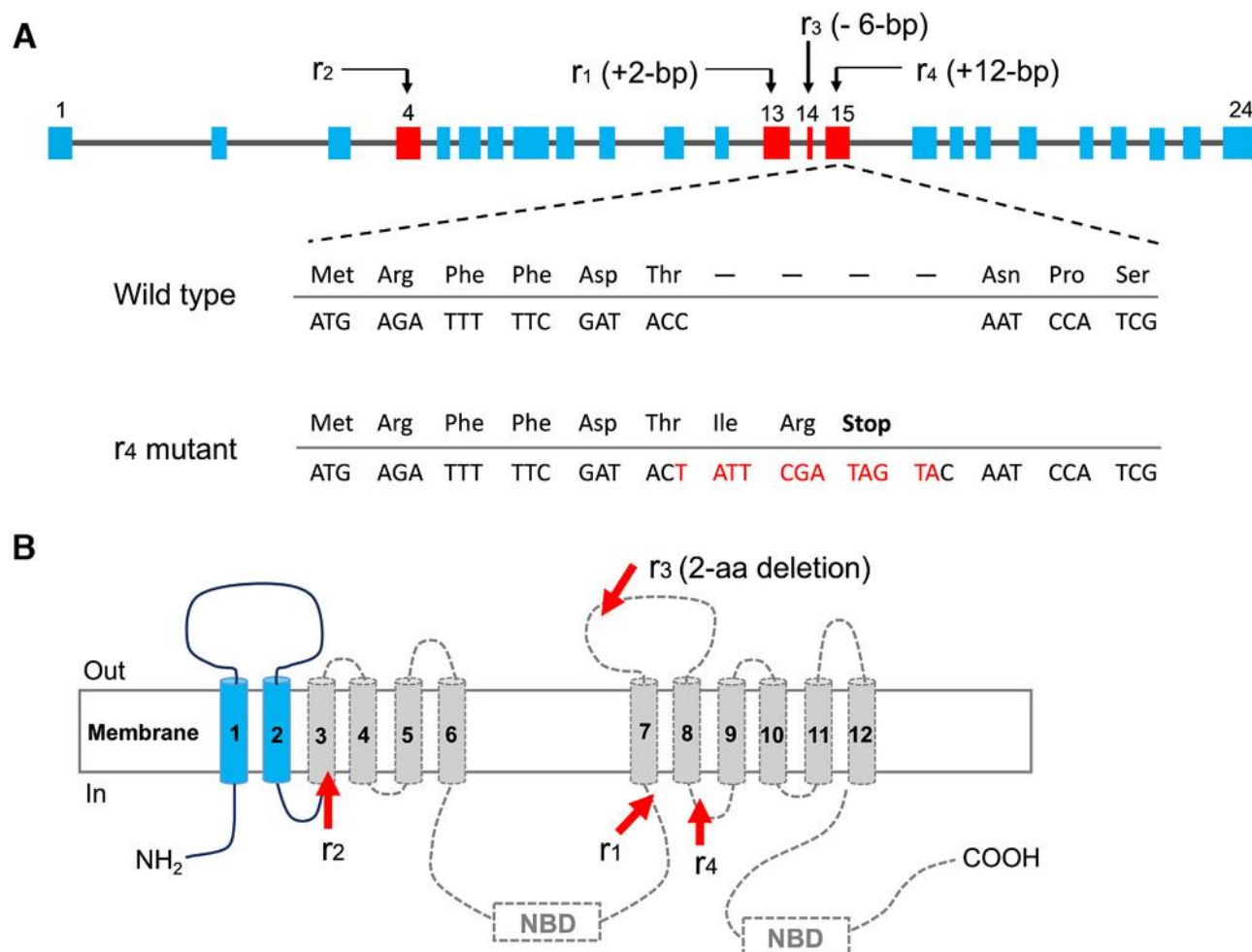


Figure 3 Four mutant alleles (r1–r4) of SfABCC2 associated with Cry1F resistance in *Spodoptera frugiperda*. (A) Genomic structure of SfABCC2. (B) Protein structure of SfABCC2. The r1, r2 and r3 alleles were reported respectively by Banerjee et al. (2017), Flagel et al. (2018) and Boaventura et al. (2019). The r4 allele was detected in the present study (Adopted from Guo et al., 2019)

3.1.3 Gene silencing

Gene silencing mechanisms, such as RNA interference (RNAi), can also play a role in resistance. Although specific examples of RNAi-mediated resistance to Bt toxins were not detailed in the provided papers, the down-regulation of key genes involved in Bt toxin binding and activation could theoretically contribute to resistance. For instance, the down-regulation of ABCC2 and ABCC3 genes has been linked to resistance in several lepidopteran species (Guo et al., 2019).

3.2 Gene amplification

Gene amplification, where multiple copies of a resistance gene are produced, can enhance resistance levels. While the provided papers did not specifically mention gene amplification as a resistance mechanism to Bt toxins, it is a well-documented phenomenon in other contexts of insecticide resistance. The increased expression of resistance genes can lead to higher levels of the corresponding proteins, which can sequester or degrade Bt toxins more effectively.

3.3 Epigenetic modifications

Epigenetic modifications, such as DNA methylation and histone modification, can alter gene expression without changing the DNA sequence. These modifications can potentially contribute to Bt resistance by regulating the expression of genes involved in toxin binding and activation. Although the provided papers did not explicitly discuss epigenetic modifications in the context of Bt resistance, this area remains an important avenue for future research to fully understand the complexity of resistance mechanisms.