IDENTIFICATION OF MUTATION IN THE *BEMISIA TABACI* (GENN.) *PARA* SODIUM CHANNEL GENE ASSOCIATED WITH RESISTANCE TO PYRETHROIDS

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Abstract

Mechanism of resistance to lambda-cyhalothrin in highly resistant *Bemisia tabaci* strain can be explained by the voltage-gated sodium channel which is the primary target site of pyrethroid insecticides. The super knockdown resistance (super-kdr) to pyrethroids is caused by changes at specific sites on *the para*-type sodium channel protein domain II (IIS4-6). The *B. tabaci para*-type sodium channel gene was RT-PCR amplified from lab-strain (reference), Parent (first generation after treatment with the insecticide) and generation thirteen after treatment was considered. The mechanisms of resistance to Lambda-cyhalothrin (Karat 20% EC) in a Q biotype, highly resistant *Bemisia tabaci* strain. Analysis of the sequence of the IIS4-IIS6 region of *the para* sodium channel gene of lab-strain, Parent and resistant strain (G13) revealed two amino acid replacements compared to that of the SUD-S susceptible strain. One is the leucine to isoleucine substitution at position 925 (L925I) and allele r1-Q1 (GenBank accession no. DQ205206) has identical intron sequences with samples of parent and lab-strain, other is a novel kdr resistant mutation for *B. tabaci*, a threonine to valine substitution at position (T929V) and alleles r2-Q1 (GenBank accession no DQ205207) has identical intron sequence with G_{13}..

Keywords: *Bemisia tabaci*, sodium channel gene, insecticide resistance, pyrethroids