Effect of the Mutation Val1016Ile in the Voltage Gated Sodium Channel Gene of *Aedes aegypti*

Brito, L.P.[1]; Belinato, T.A.[1]; Martins, A.J.[1]; Lima, J.B.P.[1]; Peixoto, A.A.[2]; Valle, D.[1]

[1]Laboratório de Fisiologia e Controle de Artrópodes Vetores; [2] Laboratório de Biologia Molecular de Insetos - FIOCRUZ

Control of *Aedes aegypti* has been impaired mostly due to insecticide selection of resistant populations. Metabolic resistance is characterized by the increased activity of naturally detoxifying enzymes, such as multi function Oxidases (MFO), Glutathione-S Transferases (GST) and Esterases. Another important mechanism, known as target-site resistance, occurs when the insecticide target molecule in the insect central nervous system is altered, losing affinity. It has been recently suggested that a mutation in the *Ae. aegypti* voltage gated sodium channel gene ($AaNa_V$) at position 1016 (Val1016Ile) plays an important role in pyrethroid resistance. Pyrethroids are presently used against *Aedes* adults in the country. To assess this effect, we selected a homozygous $AaNa_V$1016 Ile/Ile lineage from a natural population. It was obtained after crossing and allelic specific PCR genotyping of various individual couples. Dose-response bioassays with deltamethrin-impregnated papers confirmed a high level of resistance of the selected lineage when compared to the susceptible strain Rockefeller. The F1 of Rockefeller and $AaNa_V$1016 Ile/Ile lineage crosses is susceptible to pyrethroid, indicating the mutation is recessive. Since the mutant lineage also exhibits increased GST and Esterases activities, pyrethroid resistance can not be entirely attributed to the target site mutation. Purification of the mutant lineage is in course and both the role of the mutation itself to pyrethroid resistance, and its interference in the insect fitness will be evaluated.

Keywords: *Aedes aegypti, kdr* mutation, pyrethroid resistance

Supported by: FIOCRUZ, CNPq, SVS-MS and HHMI

Excluído: ¶
¶

Excluído: ¶

Excluído: Quebra de página
Frequency of Val1016Ile mutation in the voltage gated sodium channel gene of *Aedes aegypti* Brazilian populations¶
Martins, A.J.[1]; Linss, J.G.B.[1], Peixoto, A.A.[2]; Valle, D.[1]¶
[1]Laboratório de Fisiologia e Controle de Artrópodes Vetores; [2] Laboratório de Biologia Molecular de Insetos – FIOCRUZ¶
¶
One of the major insect pyrethroid resistance mechanisms affects its target site, the voltage gated sodium channel (Na_v). In *Aedes aegypti*, the Val1016Ile substitution of the $AaNa_v$ gene is associated to resistance in several Latin American countries. Genotyping of susceptible and resistant mosquitoes from 7 Brazilian localities detected the Ile1016 mutation in 5 populations, and a higher frequency of this substitution in resistant specimens in all cases. Additionally, 10 out of 16 vector populations presented the Ile1016 mutation. Our data suggest involvement of this substitution with pyrethroid resistance in Brazil.¶
¶