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EVOLUTION OF INSECTICIDE RESISTANCE

Helio Schechtman[1], Max O. Souza[2]*

[1]Fundao Oswvaldo Cruz, Brazil
[2]Universidade Federal Fluminense

maxsouza@id.uff.br(*corresponding author), h.schechtman@fiocruz.br

ABSTRACT

We are witnessing a global re-emergence of many vector-borne diseases such as malaria, dengue and chikungunya disease [1], for which there is neither aetiological treatment nor chemoprophylaxis [2] nor licensed vaccine available. In this scenario, control of the vector population is possibly the best alternative, and lack of such an adequate control might lead to recurrent outbreaks–cf. [3, 4]. Such a control is typically achieved by use of chemical insecticides that target them at a particular stage of their life-cycle, such as larvicides or adulticides [5]. However development of resistance has been routinely observed. In most cases, resistance is likely to be genetically mediated, and due to mutations in one or more genes. Specifically, in the case of pyrethroid-based insecticides the mechanism for resistance is target- site alteration [6, 7], .i.e. a genetic mutation also known as Knock-Down ResistanceKDR. Even though, most resistance mechanisms incur on fitness costs, and KDR is no exception, once a mutation occurs it can spread very fast with slow reversal in the absence of insecticide pressure [8]. Particularly in Brazil, it has been documented in field populations a large increase in these genes frequency and even fixation [9].

With this picture in mind, we employed an in silico model adapted from [4], and parametrised it for Aedes aegypti–which is a highly competent vector for dengue and the most important one [1]. The persistence of the resistance gene, once it is prevalent in the population, was then investigated by identifying the reversal time for susceptibility as a key quantity [10].

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