BSc Research: Abstract

Insecticide resistance of the major malaria vector *Anopheles Culicifacies* in Sri Lanka

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Malaria is one of the mosquito born diseases that has been causing significant problems for the past 40 years in Sri Lanka, where *Anopheles culicifacies* is the primary vector. This main vector is a species complex consisting five different sibling species, designated as A, B, C, D and E (Surendran et al., 2000 & De Silva et al., 2005). Sibling species E and B are primarily responsible for the disease on the island. (Surendran et al., 2003). Transmission of the disease is reduced by controlling the vector population. Indoor residual spraying, insecticide treated nets and laviciding are the techniques used to control the vector (WHO-2005). One of the major problems that has arisen as a result of the continuous use of insecticides is the development of a resistance in the mosquito population to insecticides, this resistance of which changes overtime. Careful monitoring of insecticide resistance and an understanding the resistance on a molecular level is essential to preventative measures for this resistance. It is also important for the formation of new insecticides. The quickest way for identifying this insecticide resistance is by carrying out a susceptibility test. The *An. culicifacies* collected from the malaria endemic area showed resistance to organophosphates, and the *An. annularis* on the secondary vectors from the study area also showed resistance to organophosphates.

Insensitive acetylcholinesterase (AChE) is a common, organophosphate and carbamate insecticide resistance mechanism. In *Anopheles gambia, Culex pipens* and *Anopheles albimanus*, the high AChE resistance is due to a single point mutation G119S of the ace-1 gene (Weill et al., 2004). Therefore, this investigation was carried out to find out whether the organophosphate and carbamate insecticide resistances among the Sri Lankan *An. culicifacies* population is caused by iAChE.To achieve this aim, the mosquito population was tested for the presence of the G119S point mutation.