Resistensi aedes aegypti terhadap insektisida malation di Jawa Tengah pengaruhnya pada aktivitas enzim asetilkolinesterase = Resistance of the aedes aegypti to malathion insecticide interference on the activity of the acetylcholinesterase enzyme

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Abstrak

Pencegahan penyakit tular vektor nyamuk kini dipersulit dengan munculnya resistensi vektor terhadap insektisida. Insektisida organofosfat (OP)-malation merupakan salah satu insektisida yang masih digunakan di Indonesia, oleh karena itu pengawasan status resistensi vektor terhadap insektisida tersebut perlu dilakukan. Dua mekanisme utama yang mendasari resistensi vektor terhadap malation adalah peningkatan enzim metabolik esterase dan insensitif enzim asetilkolinesterase (AChE). Penelitian sebelumnya di Indonesia telah melaporkan keterlibatan enzim esterase pada resistensi vektor terhadap malation, namun peran insensitif AChE belum diketahui jelas.

Penelitian ini merupakan penelitian deskriptif menggunakan nyamuk Aedes aegypti dari Jawa Tengah. Penelitian dilakukan pada bulan April-Oktober 2013 di Lembaga Eijkman. Aedes aegypti sensitif dan resistan malation hasil bioassay dianalisis secara molekuler untuk mengetahui aktivitas enzim AChE yang tersisa setelah dihambat oleh malation. Selain itu, tiga mutasi titik (G119S, F290V, dan F455W) pada gen Ace1 juga dideteksi untuk melihat pengaruh ada tidaknya ketiga mutasi tersebut terhadap aktivitas enzim AChE setelah dihambat oleh malation. Aktivitas enzim AChE ditentukan berdasarkan metode Ellman, sedangkan deteksi mutasi G119S dengan metode PCR-RFLP, dan mutasi F290V-F455W dengan metode PCR-Sequencing.

Tidak ada perbedaan "aktivitas sisa" enzim AChE yang bermakna dan tidak ditemukan mutasi G119S, F290V, dan F455W pada Ae. aegypti resistan. Hasil ini menandakan bahwa mekanisme insensitif AChE tidak mendasari resistensi Ae. aegypti terhadap malation di Jawa Tengah. Walaupun demikian, terdapat peningkatan "aktivitas sisa" AChE yang tidak bermakna pada Ae. aegypti resistan dibanding Ae. aegypti sensitif. Hasil ini menandakan bahwa kemungkinan terdapat peran enzim lain yang dapat memetabolisme malation lebih cepat atau terjadi peningkatan produksi AChE pada nyamuk resistan sehingga AChE tetap dapat menghidrolisis substratnya (asetilkolin). Mekanisme insensitif AChE belum terlibat penuh dalam mendasari resistensi Ae. aegypti terhadap malation di Jawa Tengah, namun kemungkinan mekanisme ini terlibat dapat diteliti lebih lanjut dengan menganalisis peningkatan produksi enzim AChE yang juga dapat memengaruhi aktivitas AChE selain mutasi gen Ace1.
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The prevention of mosquito-borne diseases becomes difficult to overcome since the vectors have developed resistance to insecticides. The molecular basis of resistance to insecticides therefore need to be explored to determine the resistance status earlier. In Indonesia, organophosphate (OP)-malathion insecticide has been widely used to control vector population and therefore the resistance status to this insecticide should be under control. Two main mechanisms have known to be associated with resistance to malathion, previous

studies in Indonesia reported that esterase responsible in resistance to malathion, however the insensitive AChE-based mechanism remain to be determined.

Descriptive study was conducted at Eijkman Institute during April to October 2013 using Aedes aegypti from Central Java. Malathion sensitive and resistant Ae. aegypti from bioassay were subjected to molecular analysis to compare the remaining activitiy of AChE between those mosquitoes after inhibited by malathion. The presence of three point mutations (G119S, F290V, and F455W) in the Ace1 gene associated with resistance to malathion were also detected to see the effect of the absence or presence of those mutations to AChE activity.

The results showed that AChE remaining activities in the resistant Ae. aegypti have no significantly different compare to those in the sensitive Ae. aegypti. No associated mutations found in the Ace1 gene (G119S, F290V, or F455W) as well. These results indicated that insensitive AChE-based mechanism is not involved in Ae. aegypti resistance to malathion in Central Java. However, we noticed that the remaining activities of AChE are increased insignificantly in resistant Ae. aegypti, suggesting the possibilities of metabolic enzyme which can degrade insecticide faster or could be due to overproduction of AChE enzyme which may increase the hydrolizing process of acetylcholine (ACh). Insensitive AChE-based mechanism is still not fully involved in Ae. aegypti resistance to malathion in Central Java, however the potency of its involvement should be further analyzed by considering the overproduction of AChE enzyme itself which could contribute in AChE activity enhancement other than Ace1 gene mutation.