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# A glutathione S-transferase metabolic resistance to insecticides is associated with higher Plasmodium infection in the African malaria vector Anopheles funestus

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Metabolic resistance to insecticides is threatening malaria control in Africa. However, the extent to which it impacts malaria transmission remains unclear. Here, we investigated the impact of a glutathione S-transferase metabolic resistance on Plasmodium infection in field Anopheles funestus s.s. compared to A296S-RDL. The 119F-GSTe2 resistant allele was present in Southern (Obout) (56%) and Centre (Mibellon) (25%) regions of Cameroon whereas 296S-RDL resistant allele was at 98.5% and 15%, respectively. The whole mosquito Plasmodium and sporozoite infection rate was, respectively 57% and 14.8% in Obout (n=508) and 19.7% and 5% in Mibellon (n=360). No association was found between L119F-GSTe2 and whole mosquito infection status. However, when analyzing oocyst and sporozoite infection separately, the resistant homozygous 119F/F genotype was significantly more infected in Obout than both heterozygote (OR=2.5; P=0.012) and homozygote susceptible (L/L119) genotypes (OR=2.10; P=0.013). In contrast, homozygote RDL susceptible mosquitoes were more infected than other genotypes (OR=4; P=0.03). No additive interaction was found between L119F and A296S. Sequencing of the GSTe2 gene showed no association between the polymorphism of this gene and Plasmodium infection. Glutathione S-transferase metabolic resistance potentially increases the vectorial capacity of resistant An. funestus mosquitoes suggesting a potential exacerbation of malaria transmission in areas of high GSTe2-resistance.

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