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Genotyping for point mutations in selected codons of pfcr1 and pfmdr1 genes of Plasmodium falciparum among patients with uncomplicated malaria in Mbita district Kenya

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Abstract

Background
Malaria remains a leading cause of morbidity and mortality in Kenya, especially in young children and pregnant women. Due to widespread resistance of Plasmodium falciparum to drugs such as Chloroquine (CQ) and Sulfadoxine-Pyrimethamine (SP), Artemisinin Combination Therapy (ACT) was adopted in Africa as a means of improving treatment efficacy and slowing the spread of resistance. The development of drug resistance by the parasites for the various malaria drug regimens that have been in use before has been attributed to point mutations within the parasite genome. Therefore
this study investigated the prevalence of point mutations in selected codons of the \textit{pfcrt} and \textit{pfmdr-1} genes of \textit{Plasmodium falciparum}. It is however unclear whether ACT will be effective in preventing the selection of resistant parasites in Africa, where parasite transmission rates are generally much higher with parts of Asia and Africa already reporting a reduction in sensitivity to ACT.

\textbf{Methods}

The dot-blot/probe hybridization technique was used to identify point mutations in codons 74, 75 and 76 of the \textit{pfcrt} gene and codons 1034, 1042 and 1246 of the \textit{pfmdr-1} gene in Mbita a malaria holoendemic site in Kenya. In the \textit{pfcrt} gene, 76T mutation was found to be in 91 (79.83\% CI 63.1-88.5) of 114 samples while the, the wild type allele 76K was present in 23 (20.17\% CI 9.0-22.0) samples. Codons 1246 showed allelic variation with 1246D the wild type allele being 72.8\% (CI 52.0-89.1). This was a significant increase in the 76K allele ($p$=0.001) in comparison to the year 2005 where prevalence of 76K was 6\%.

\textbf{Conclusion}

There’s an expansion of the wild-type allele 76K of the \textit{pfcrt} gene and no significant difference in the 1246D allele of the \textit{pfmdr1} gene, moreover the prevalence of 76T allele is still high in Mbita hence it’s beneficial to continue using AL as treatment for uncomplicated malaria.

\textbf{Keywords:} Malaria, Drug Resistance, Point Mutations

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