KEMRI | Wellcome
POLICY BRIEF
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Antimalarial drug resistance (AMDR) repository from 1987–2022

Summary

1. The majority of data was generated from Kilifi, Coastal Kenya and Kisumu, Western Kenya
2. The main AMDR molecular markers with data were the chloroquine, sulphadoxine-pyrimethamine and artemisinin
3. The earliest identification a WHO-validated k13 mutation, P553L, was in 2006 in Kisumu
4. More data is required from across the country to fill in the AMDR data gaps and comprehensively map the genetic markers nationally.

Background

Antimalarial drug resistance (AMDR) refers to the parasites reduced susceptibility to standard antimalarial treatments and prolonged or incomplete parasite clearance that can lead to treatment failure [1]. The malaria parasite has historically evolved to avoid drug action with mutations emerging to all existing and new antimalarial treatments. Over 60 years ago, the sulphadoxine-pyrimethamine (SP) genetic resistance markers [3,4] provided supporting evidence to therapeutic efficacy studies of the urgent need to change national antimalarial treatment policy from SP to artemisinin-based combination therapies.

Since the earliest identification of the artemisinin resistance mutations in the kelch 13 gene in Rwanda, in 2014, we are now >10 years late in ramping up surveillance for these mutations in Kenya. Data is available though, as malaria molecular surveillance (MMS) is conducted by national and international academic research groups. A scoping review of AMDR mutations in Kenya was undertaken to assemble historical and contemporary data on mutations to previously and currently used antimalarial drugs. The intention is to provide a MMS database that can be used by the National Malaria Control Programme (NMCP) to better understand the past, present and possible future of parasite mutations and initiate better future coordination and submission of data assembled by research partners.

Findings

The national analysis showed that by the time drug policy changed from chloroquine to SP in 1999, the dihydropteroate synthase (dhps) mutant genotype was already rising from 1996 in the Coast and 1998 in Western Kenya (Figure 1), while the dihydrofolate reductase (dhfr) codon 108 shift to mutant genotype occurred as early as 1988 in the Coastal parasite populations. The WHO-validated k13 mutations were first described, P553L, in 2006 in Kisumu (Table 1).

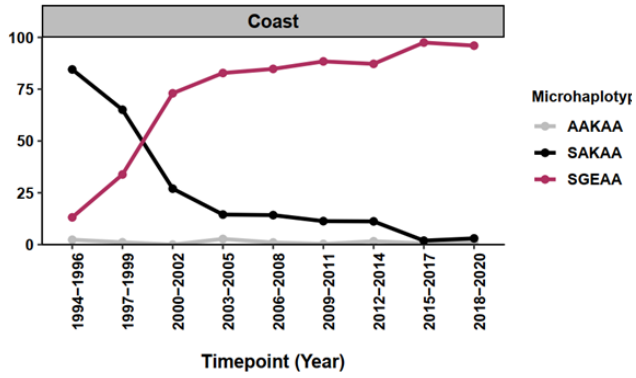

Table 1. List of WHO validated K13 mutations in Kenya

year_sample	study_county	sample_size	C469Y	P553L	A675V	reference
2006	Kisumu	50	-	2	-	Taylor et al. 2015
2018-2022	Kisumu, Kisii, Kakamega, Homa Bay	775	-	-	0.4	Jeang et al. 2024
2022	Bungoma	74	5.9*	-	-	Osoti et al. 2025
2022	Busia	322	2.8*	-	-	Osoti et al. 2025
2022	Busia	226	4.8	-	-	Makau et al. 2024
2022	Kakamega	145	-	4.3*	-	Osoti et al. 2025
2022	Kisumu	161	10.7*	3.6*	7.1*	Osoti et al. 2025
2022	Migori	275	2.1*	-	-	Osoti et al. 2025
2022	Siaya	337	6.9*	-	-	Osoti et al. 2025
2022	Turkana	92	4.3*	-	-	Osoti et al. 2025
2022	Vihiga	68	2.1*	-	-	Osoti et al. 2025
2022	West Pokot	32	33.3*	14.3*	50*	Osoti et al. 2025

An asterisk (*) denotes mixed genotype frequencies, where both wild-type and mutant alleles were detected within the same sample.

Recommendations

The foci of AMDR work are from research institutions in Western Kenya and the Coast. Together the consolidated database highlights the essential variables to support data sharing (Table 2). The compilation and standardization of over 100 studies provided a high-level, structured overview of when and where resistance markers have been surveyed. This establishes a foundational national repository to support strategic surveillance planning by the Kenya NMCP. It further highlights the urgent need for a national representation of data to fill in critical data gaps.

A centralized MMS repository will allow for the necessary resources and technical support to be mobilized; expertise to be shared by developing a network of laboratories for genomics and bioinformatics; standardized protocols to generate reproducible data and enable reagents sourcing at scale; and importantly coordinated sample referral systems [54]. For malaria this is important as control interventions can be targeted to regions where resistance is emerging as evidenced from the current data in Western Kenya that also highlights the need for border control interventions. This policy brief should strengthen the foundation for a nationally representative surveillance framework.

Table 2. List of standard variables extracted from 110 studies.

VARIABLE
PMID
Year of publication
Study county
Study town
Year of sample collection
Number of samples genotyped (sample size)
Number of samples genotyped
Target gene
Target allele
Number of wildtype genotypes
Number of mutant genotypes
Number of mixed genotypes
Codons
Microhaplotype
Microhaplotype frequency
Genotyping assay
Participants age
Participants age unit (years or months)
Participants clinical status (asymptomatic, symptomatic or severe)
Study design (health facility-based or community survey)
Data extraction comments

References

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