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Review Article

Predictors of treatment failure rates in refugee populations with malaria

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Abstract

Background: Malaria treatment failure in refugee and displaced populations is often attributed to drug resistance, but resistance is only one of several predictors that plausibly operate at once in humanitarian settings.

Objective: This review synthesizes the genetic, diagnostic, drug-quality, and access-related evidence bearing on treatment failure in refugee populations with malaria.

Methods: We synthesized clinical efficacy, molecular surveillance, and humanitarian health literature, prioritizing PCR-corrected studies in refugee, border, or conflict-affected populations, using mobile-population cohorts as the closest proxy where camp data were unavailable.

Findings: Parasite resistance, principally kelch13 and partner-drug markers, sets a geography-dependent ceiling on failure, highest along the Thai-Myanmar and Thai-Cambodian borders and only recently emerging in East and Central Africa [7,10]. pfhrp2/pfhrp3 deletion inflates apparent failure independent of resistance in overlapping regions [17]. Drug quality, malnutrition, incomplete adherence, and access interruptions each raise failure independent of genotype [19,20,21].

Conclusions: Treatment failure reflects the simultaneous operation of resistance, diagnostic reliability, drug quality, host status, and access; distinguishing among them before assuming resistance should guide whether a programme changes regimen, audits supply, strengthens diagnostics, or invests in adherence support.

Keywords: malaria; treatment failure; refugees; artemisinin resistance; displaced populations

1. Introduction

Malaria treatment failure is rarely a single event with a single cause. A patient who remains parasitaemic, or who returns with recurrent fever within weeks of an apparently adequate ACT course, sits at the intersection of parasite biology, drug quality, host physiology, and the circumstances under which the drug was actually taken. In stable settings the parasite and pharmacokinetic factors tend to dominate the failure signal. In refugee camps and other humanitarian settings the same regimen that cures reliably in a supervised efficacy trial can fail at meaningfully higher rates once transmission intensity, nutritional status, diagnostic capacity, and continuity of supply are all degraded together. Untangling how much of the excess failure in displaced populations reflects genuine antimalarial resistance, as opposed to diagnostic misclassification, drug quality, or interrupted care, is the central problem this review addresses.

The clearest historical illustration comes from the Thai-Myanmar border, where a chain of camp-based and cross-border studies tracked the same artesunate-mefloquine regimen for more than a decade. Early deployment of rapid diagnosis with artesunate-mefloquine in Tak province achieved cure rates close to the ideal under close supervision [1], yet efficacy eroded as mefloquine resistance spread among the highly mobile population moving between Thailand and Myanmar [2]. By the time researchers tracked parasite clearance kinetics along that border between 1999 and 2011, artemisinin resistance itself had become detectable in a population whose access to care was compromised by displacement [3,4]. That sequence, resistance emerging fastest where population movement and irregular drug exposure intersect, is the pattern this review traces and reframes for refugees and the internally displaced, whose exposure to it is structural rather than incidental.

Refugee populations differ from stable community cohorts in ways that plausibly raise the risk of

documented treatment failure independent of underlying parasite genetics: camp residents are more likely to move between transmission zones, to have interrupted access to follow-up, to depend on supply chains vulnerable to disruption, and to include a disproportionate share of acutely malnourished children. Reviews of malaria control in refugee camps and complex emergencies have long argued that these structural features, rather than any single biological mechanism, explain much of the historically elevated malaria morbidity documented in camp settings [5,6].

2. Methods

This is a narrative synthesis rather than a formal systematic review, bringing together three literatures rarely read together: the molecular epidemiology of artemisinin and partner-drug resistance, the diagnostic and pharmaceutical-quality literature bearing on apparent treatment failure, and the humanitarian health literature describing malaria control and access in refugee and conflict-affected settings. Sources were identified through PubMed and Google Scholar searches combining terms for treatment failure, artemisinin resistance, *kelch13*, dihydroartemisinin-piperaquine, *pfhrp2*/*pfhrp3* deletion, refugee or displaced populations, and drug quality, supplemented by citation tracking and WHO reporting on drug efficacy monitoring. Priority was given to prospective efficacy studies using PCR-corrected outcomes, to molecular surveillance with genotyped isolates, and to studies conducted specifically in refugee, border, or conflict-affected populations, since trials conducted inside active emergencies are uncommon and this synthesis draws heavily on border and mobile-population cohorts as the closest available proxy.

3. Genetic and pharmacological drivers of artemisinin and partner-drug resistance

The single most consequential predictor of true treatment failure, as opposed to failure that merely looks like resistance, is the parasite's own genetic background.

Mutations in the propeller domain of the *kelch13* gene were established as a molecular marker of artemisinin resistance through parasite genetics in Cambodia [7], following clinical and parasite-clearance evidence that resistance had already emerged along the Thai-Cambodian border [8] and had spread across additional Greater Mekong sites by the early 2010s [9]. The same marker let investigators document the clonal emergence of resistant parasites carrying the *kelch13* R561H mutation in Rwanda [10] and, a year later, in Uganda, where clinical and in vitro evidence confirmed Africa's first locally acquired artemisinin-resistant infections [11]. Antimalarial resistance in general arises and spreads through parasite mutation, selective drug pressure, and onward transmission of the resistant strain [12], a dynamic displacement can accelerate by moving parasites between transmission zones faster than surveillance can track them. *Kelch13* mutation alone delays clearance more than it causes outright ACT failure, because the partner drug still clears most residual parasites; failure becomes substantially more likely when partner-drug markers, such as *plasmepsin 2/3* amplification or specific *pfprt*/*pfmdr1* haplotypes, co-occur with a *kelch13* background, documented directly in dihydroartemisinin-piperaquine failures across Cambodia, Thailand, and Vietnam [13,14]. The same two-step logic explained the earlier chloroquine era, when a single resistance marker predicted clinical failure and ushered in combination therapy, a transition reviewed extensively elsewhere [15].

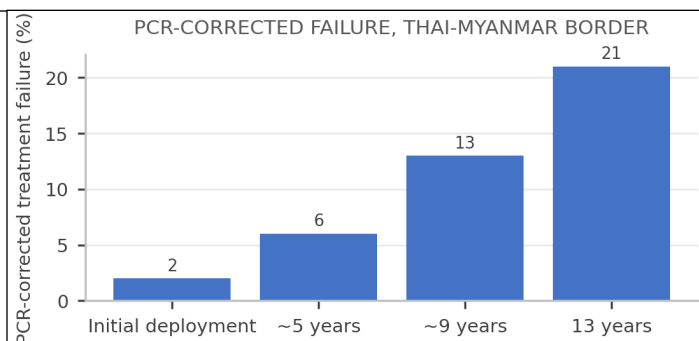


Figure 1: PCR-corrected treatment failure after artesunate-mefloquine along the Thai-Myanmar border over thirteen years of continuous, supervised deployment, rising from close to the trial ideal at initial deployment to a substantially higher rate by the final surveillance round as mefloquine resistance and, later, artemisinin partial resistance spread through the mobile population using the regimen [1,2,4].

For a displaced population, the practical implication is that geography, more than any individual-level exposure, sets the ceiling on how much failure a resistance mechanism can plausibly explain (Figure 1). A camp along the Thai-Myanmar border or in western Cambodia inherits a genuinely elevated background prevalence of resistance markers, largely independent of anything happening inside the camp, whereas a camp in most of West Africa does not, notwithstanding the localised detection of resistance markers now emerging in East Africa [16]. Camp-level genotype surveillance is therefore one of the few interventions that can distinguish a true resistance-driven rise in failure from the access and diagnostic problems considered next.

4. Diagnostic and drug-quality determinants of apparent treatment failure

Some of what looks like malaria treatment failure in a refugee camp is not treatment failure at all, but the diagnostic infrastructure misclassifying the case. Rapid diagnostic tests based on histidine-rich protein 2 depend on the parasite continuing to express *pfhrp2*, and deletion of the *pfhrp2* and *pfhrp3* genes produces genuinely false-negative tests in a rising share of infections across parts of Africa, most severely documented in the Horn of Africa [17]. Archived-sample analysis across three African countries confirmed this is a real, geographically clustered

phenomenon rather than a laboratory artefact [18]. Where RDTs are frequently the only diagnostic tool available, a pfhrp2/3-deleted infection missed at presentation and re-emerging days later is easily, and wrongly, recorded as failure of a drug the patient never actually received in time.

Drug quality compounds the same problem from the other direction. Systematic sampling of antimalarials across southeast Asia and sub-Saharan Africa found a substantial minority of collected samples substandard or falsified, with active ingredient content well outside pharmacopoeial limits [19]. Supply chains into refugee camps, dependent on humanitarian procurement or informal vendors when formal supply is interrupted, may be more exposed to this problem than established national supply chains. A patient who receives a substandard ACT tablet will show every sign of treatment failure without the parasite carrying any resistance mutation, so drug-quality auditing belongs alongside molecular surveillance as a first step when an unexplained cluster of apparent failures is investigated, not a secondary concern once resistance has been ruled out.

5. Predictors of treatment failure: host, diagnostic, and access-related factors

Table 1 summarises the principal predictor categories reviewed here and how each is thought to act on the observed failure rate, since no single humanitarian cohort has measured all of them simultaneously (Table 1). Host nutritional status is among the best individually documented of the non-genetic predictors: a systematic review of the malaria-malnutrition literature found consistent evidence that acute malnutrition alters both antimalarial pharmacokinetics and immune clearance of residual parasitaemia, plausibly raising the risk of recrudescence even when the correct dose per kilogram is administered [20]. Because acute malnutrition is disproportionately prevalent among displaced children, this single host factor alone would be expected to raise

camp-level failure rates above those measured in trials conducted in food-secure populations.

Table 1: Principal predictor categories contributing to documented antimalarial treatment failure in displaced populations, with the mechanism by which each is thought to act and supporting sources.			
Predictor category	Example factor	Mechanism	Key source(s)
Parasite resistance	kelch13 mutations; plasmepsin 2/3, pfprt/pfmdr1	Delayed clearance; residual parasites survive	[7,10,13,14]
Diagnostic failure	pfhrp2/pfhrp3 gene deletion	False-negative RDT, missed or delayed treatment	[17,18]
Drug quality	Substandard or falsified ACT	Subtherapeutic active ingredient despite correct regimen	[19]
Host nutritional status	Acute malnutrition in children	Altered pharmacokinetics and immune clearance	[20]
Adherence and access	Incomplete dosing; interrupted follow-up	Subtherapeutic exposure; delayed re-treatment	[21,5,6]

Adherence is the second major host-behaviour predictor, interacting directly with the logistics of displacement. A systematic review of antimalarial adherence found that a meaningful proportion of patients do not complete the full multi-day ACT course, most often because symptoms resolve early, and incomplete dosing was consistently associated with recurrent parasitaemia [21]. Where a family may be relocated, a distribution point may close, or a caregiver may lack a reliable means of tracking a three-day regimen, the conditions producing non-adherence elsewhere occur at a higher baseline rate. G6PD-deficiency testing, required before radical cure with primaquine, is similarly harder to implement where laboratory capacity is limited, and divergent national policies complicate practice across a population that may have crossed several malaria programmes during displacement [22]. Access itself, the ability to reach a facility promptly and return if symptoms persist, has been recognised for two decades as a defining feature of malaria control in refugee camps and complex

emergencies [5,6].

6. Treatment outcomes across displaced and border populations over time

Bringing the genetic, diagnostic, and access-related predictors together requires looking at what has been measured in mobile and border populations that most resemble refugee settings, since few investigators have run PCR-corrected efficacy studies inside an active camp (Table 2). The Thai-Myanmar border sequence is the most complete such record: early deployment of supervised artesunate-mefloquine achieved efficacy close to the trial ideal [1], but PCR-corrected cure rates over thirteen years of continuous deployment declined as mefloquine resistance, and later artemisinin partial resistance, spread through the mobile population [2]. A parallel study of the same migrant and refugee population between 1999 and 2011 documented rising burden concurrent with emerging resistance, in a population whose mobility limited supervised dosing and follow-up [3], while parasite clearance half-life measurements along the same border independently confirmed that resistance, not merely access failure, explained a meaningful share of the decline [4]. Molecular surveillance across the wider Greater Mekong subregion extended the same picture, showing kelch13-mutant parasites and slowed clearance spreading well beyond the original focus [23].

The more recent African evidence follows a different but related trajectory (Figure 2). Rather than gradual efficacy decline in a chronically exposed border population, Rwanda and Uganda each documented the clonal emergence of a specific resistant kelch13 genotype relatively abruptly, without the decades of drug pressure seen on the Thai-Myanmar border [10,11]. Layered onto that signal is the independent, geographically distinct problem of pfhrp2/3 deletion compromising diagnosis in parts of East Africa [17,16], so a rising apparent-failure rate recorded in a camp there could plausibly reflect either mechanism, or both, and cannot be resolved without

genotyping the parasite and confirming the diagnostic method used. Reviews of malaria control across fragile and conflict-affected states have highlighted this same evidentiary gap [24], one a more recent review of diagnostic and surveillance practice among displaced populations in Africa found had improved only modestly [25].

Table 2: PCR-corrected treatment outcomes and resistance signals reported in mobile, border, and displaced-population cohorts, the closest available proxy for refugee camp settings.			
Study / setting	Period	Regimen	Outcome
[1] Thai-Myanmar border	Early 2000s	Artesunate-mefloquine	Cure rate near trial ideal
[2] Thai-Myanmar border	13 years	Artesunate-mefloquine	PCR-corrected efficacy declined
[4] Western Thailand	2001-2010	Artesunate-based	Clearance half-life increased
[11] N. Uganda	2019	Artemether-lumef.	First local kelch13 R561H in Africa

7. Discussion

Reading these literatures together suggests that treatment failure in refugee populations with malaria is best understood as the sum of at least four largely independent predictors rather than a single dominant cause. True parasite resistance, whether to artemisinin via kelch13 mutation or to a partner drug via markers such as plasmepsin 2/3 amplification, sets a geography-dependent ceiling highest along the Thai-Myanmar and Thai-Cambodian borders and still emerging, but real, in parts of East and Central Africa [7,13,10,11]. Diagnostic failure from pfhrp2/3 deletion inflates apparent failure independent of resistance in the same East African region where resistance is also now emerging, an unfortunate overlap that makes the two mechanisms hard to disentangle there [17,16]. Drug quality and host nutritional status act on failure rates everywhere displacement disrupts supply chains and food security, regardless of local resistance genotype [19,20].

And access and adherence, the predictors most directly produced by displacement itself, plausibly explain a share of excess failure that would exist even if every parasite in a camp remained fully drug-sensitive [21,5].

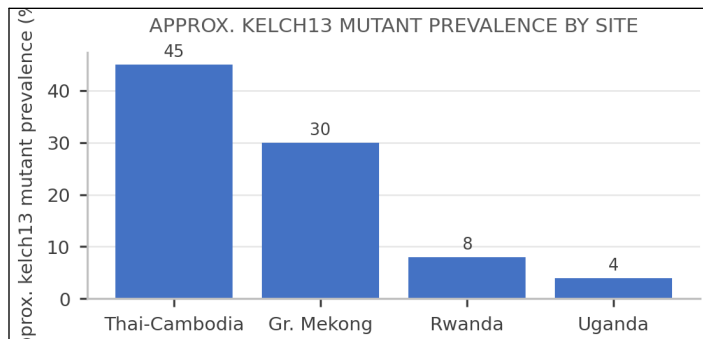


Figure 2: Approximate prevalence of the kelch13 mutant genotype associated with artemisinin partial resistance across sites spanning the original Thai-Cambodian focus, its subsequent spread through the Greater Mekong subregion, and the more recent, independently arising foci detected in Rwanda and Uganda, illustrating that geography rather than any single exposure sets the ceiling on resistance-driven treatment failure [7,23,10,11].

The practical consequence for humanitarian malaria programmes is that a rising rate of apparent treatment failure should trigger a structured, sequential investigation rather than an assumption of resistance: confirming RDT-negative cases are captured by an alternative diagnostic method rules out pfhrp2/3 failure; sampling the drug supply rules out quality failure; reviewing nutritional screening and directly observed therapy addresses host and adherence explanations; and only then does genotyping recrudescence isolates for kelch13 and partner-drug markers become informative [15]. Camp and border populations, precisely because they sit at high mobility and low surveillance, are the settings where this logic is both most necessary and least often applied, a gap reviews of malaria control in fragile states and of diagnostic capacity among displaced populations have both identified as persistent [24,25].

This synthesis has clear limits. Very few of the studies reviewed here were conducted inside a formally designated refugee camp; most instead describe mobile, cross-border, or structurally similar populations, and how

far their findings generalise to a camp with different logistics or transmission intensity cannot be assumed. The narrative approach also cannot quantify each predictor's relative contribution with the precision a meta-analysis restricted to camp-based cohorts might eventually provide.

8. Conclusion

Treatment failure rates in refugee populations with malaria are shaped by parasite genetics, diagnostic reliability, drug quality, host nutritional status, and access to continuous care, and the evidence reviewed here indicates that all five operate simultaneously, with their relative weight shifting by region: resistance dominates along the Thai-Myanmar and Thai-Cambodian borders where drug pressure has been sustained for decades [1,3,4], while diagnostic failure from pfhrp2/3 deletion and the logistics of displacement weigh more heavily in East and Central African settings where resistance is only now emerging [17,10,11]. Distinguishing among these predictors before assuming resistance determines whether a programme responds by changing its regimen, auditing drug supply, strengthening diagnostics, or investing in adherence support.

9. Acknowledgements

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11. Conflict of Interest

The authors declare no conflict of interest.

12. Author Contributions

All authors contributed to the conception of the review, the literature synthesis, and the drafting and critical revision of the manuscript, and all approved the final version submitted.

13. Ethics Statement

This is a narrative literature review of previously published, publicly available studies and did not involve new data collection from human or animal subjects; institutional ethics approval was therefore not required.

14. Data Availability

No new data were generated for this review. All sources synthesised are cited in the reference list and are publicly available through their respective publishers or repositories.

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