Opinion
Volume 11:3, 2022

Journal of Malaria Control and Elimination

ISSN: 2470-6965 Open Access

Cyclic Resistance to Antimalarial Monotherapy

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Introduction

A dangerous parasite ailment called malaria is spread by anopheline mosquitoes. There are four species of Plasmodium that often infect people, but only Plasmodium falciparum (*P. falciparum*) is responsible for the majority of morbidity and fatality cases. Unsettlingly, drug resistance has developed across all antimalarial medication classes and it is largely to blame for the high death rate associated with malaria, especially in Africa. The World Health Organization (WHO) has recommended artemisinin-based combination therapies (ACTs) as the first-line treatment for P. falciparum malaria in nations where drug resistance has hampered the effectiveness of other medications. Combination therapy of antimalarials is one method of partially alleviating drug resistance [1-5].

Description

Antimalarial medications work in different ways and at different points in the Plasmodium life cycle. Particularly, quinine, chloroquine, amodiaquine, mefloquine, piperaquine and halofantrine interfere with the biocrystallization of heme, causing toxic heme accumulation that impairs membrane function and causes cell lysis. Similar to how dihydroartemisinin binds to hemozoin and releases harmful free radicals, artemsinin, artesunate and artemether are prodrugs of this medication. Additionally, mefloquine may specifically target P. falciparum's 80S ribosome, which would prevent protein synthesis and have schizonticidal effects.

In contrast to sulfadoxine and pyrimethamine, doxycycline prevents *P. falciparum* from synthesising nucleotides and deoxynucleotides as well as folate. Specifically inhibiting the mitochondrial electron transport chain, atovaquone is a competitive inhibitor of ubiquinol. For these antimalarials, it's noteworthy that a number of other modes of action have been suggested. Quinine, for instance, also affects P. falciparum's purine nucleoside phosphorylase. There has been a link between periodic rhythms and antibiotic medication resistance. In a single university medical facility in Germany during a ten-year period, Pseudomonas aeruginosa drug resistance levels fluctuated.

A cyclic function controls the majority of antimalarial resistance. This observation supports a combination therapy using two or more antimalarial medications and is therapeutically useful. Combination therapy (i.e., primaquine and tafenoquine) are required to accomplish a "radical cure" and to get rid of the parasite as solo therapies fail. Cyclic drug resistance to antimalarials is consistent with Darwin's theory of natural selection. Antimalarial resistance is initially a rare and intermittent characteristic. The survival of parasites that are resistant is then encouraged when the antimalarial medicine has been used extensively globally, which in turn results in resistance. The medicine is then used less as resistance rises. Finally, natural selection weakens and resistance weakens as drug usage reduces.

Additionally, our findings show that using antimalarial medications before the resistance peak and after it has been achieved is not recommended (i.e., artemisinin and dihydroartemisinin). The majority of antimalarial medications appear to be long-lasting, according to these findings. Importantly, the scientific community should not be discouraged by these findings as they work to create new antimalarial medications and bolster our existing pharmacological library to combat this dreadful illness. The fact that the normalised citations also correlate strongly with non-cyclical functions, such as polynomial functions of order 3 and higher, as well as the annual citations of antimalarial drug resistance, should be recognised as a potential drawback to this study. It's important to note that our data has to be normalised in order to take pointwise mutual information into consideration. (PMI)

Antimalarial medications should be less heavily weighted if their incidence with resistance is over-cited in a particular year. Similarly, if drug resistance is mentioned frequently over the course of a year, its link to malaria should be given less weight. Thus, pointwise mutual information has been taken into consideration *via* normalisation. Antimalarial resistance is treated as being globally uniform in this study since it does not differentiate between the resistance of the various strains of the Plasmodium agent (*P. falciparum, P. vivax*, etc.) and their geographic regions. However, the regional experimental data reported by Singhasivanon and the global clinical data released by WWARN are consistent with our theoretical predictions of antimalarial resistance.

Conclusion

As may be seen from the PubMed citations, antimalarial resistance is cyclical. Notably, the projected resistance is in line with experimental and clinical findings, which implies that despite the fact that most antimalarial monotherapies are long-lasting, it is still best to avoid them-especially when resistance is significant. Last but not least, the cyclic resistance favours antimalarial medication combinations, especially those with low resistance.

Conflict of interest

None.

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Received: 03 May, 2022, Manuscript No. mcce-22-68245; **Editor assigned:** 04 May, 2022, PreQC No. P-68245; **Reviewed:** 10 May, 2022, QC No. Q-68245; **Revised:** 16 May, 2022, Manuscript No. R-68245; **Published:** 24 May, 2022, DOI: 10.37421/2470-6965.2022.11.184

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How to cite this article: Talisuna, Ambrose Otau. "Cyclic Resistance to Antimalarial Monotherapy." Malar Contr Elimination 11 (2022):184.