Malaria Control and Elimination: How Far we are: An Opinion Article

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Malaria is caused by four Plasmodium species in humans (P. falciparum, P. vivax, P. malariae and P. ovale) which spread from one person to another via the bite of female Anopheles mosquito. P. falciparum causes most deaths from malaria and is most prevalent on the African continent whereas P. vivax has a wider geographical distribution. According to the latest WHO estimates, released in December 2015, there were 214 million cases of malaria in 2015 and 438,000 deaths. Existing strategies to control malaria include vector control, chemoprevention and case management [1]. There are currently no licensed vaccines against malaria. RTS, S/AS01 is the only ongoing research vaccine against P. falciparum, which might still take 3 to 5 years to come out, if safety and effectiveness are considered acceptable. Thus, in absence of a successful vaccine, malaria control relies on the use of anti-malarial drugs. Artemisinin-based combination therapies (ACTs) are the best available treatment for P. falciparum malaria. The impact of the use of ACTs for malaria treatment is proven by the fact that Tu Youyou was awarded the 2015 Nobel Prize for Medicine for the discovery of the malaria drug, Artemisinin. But to the dismay of malaria research community all over the world, parasite resistance to artemisinins has been detected in 5 countries of the Greater Mekong subregion: Cambodia, Lao People’s Democratic Republic, Myanmar, Thailand and Viet Nam as observed in case of all previous anti-malarial drugs. The major concern now is the spread of multi-drug resistance to other regions with dire public health consequences. The most critical benchmark in the study of ART resistance was the identification of single point mutations in the propeller region of P. falciparum kelch protein gene on chromosome 13 (PfK13) as a molecular marker associated with delayed parasite clearance in vitro and in vivo. Numerous studies have been reported to determine the levels of polymorphisms of K13 in this region in order to map the spread and evolution of ART resistance [2-4]. Very interestingly there have been some reports of slow parasite clearance rates even in absence of K13 mutant alleles suggesting the role of additional molecules in development of ART resistance in P. falciparum. It would be crucial to identify additional genetic loci involved in ART resistance.

The pace at which the geographical extent of artemisinin resistance is spreading is faster than the rate at which control and elimination measures are being developed and introduced. This emphasizes the fact that apart from understanding the current state and mechanisms of antimalarial drug resistance it is extremely essential at the same time to expand the current arsenal used against the parasite. This would include the identification and development of novel vaccine candidates and the anti-malarial drug targets for malaria [5-7].

References