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A REVIEW OF GENETIC DIVERSITY OF PLASMODIUM INFECTIONS IN HUMANS

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Abstract

The human has been regarded as the natural hosts of the four species of malaria viz., *Plasmodium vivax, Plasmodium falciparum, plasmodium ovale, plasmodium malariae* but genus *Plasmodia* is very wide and it involves more than 200 parasite species that infects a variety of hosts such as reptiles, birds, rodents, primates and other mammals. The Plasmodium genus belongs to the large phylum of apicomplexans that also includes parasites responsible for other important human and animal diseases such as malaria parasites are members of the order coccidian, sub order Haemosporidiidea, family plasmodiidae, genus plasmodium. Malaria results from an infection with plasmodium parasites. All four species of *Plasmodium(i.e.,Plasmodiumvivax, Plasmodium falciparum, Plasmodium ovale, Plasmodium malariae*) infect humans by a bite of a female mosquito of the genus Anopheles. Of these four parasites, *Plasmodium* falciparum and *Plasmodiumvivax* are responsible for the greatest morbidity than *Plasmodium*ovaleand*Plasmodium*malariae. The *Plasmodiumfalciparum* causes more mortality than *Plasmodiumvivax*. Of these four species, the most widely distributed species of *Plasmodiumvivax*, which is a threat to about 40% of the world's population, infecting an estimated 70 million to 390 million people annually.Theburden of malaria in Southeast Asia has been underappreciated, despite recent evidence suggesting that the continent contributes almost malaria). India contributes 77% of the total malaria in Southeast Asia and about 95% of the population of moderate to high risk of malaria in south East Asia region is living in India.

1. INTRODUCTION

Plasmodium vivax is a protozoan parasite and a human pathogen. This parasite is most frequent and widely distributed cause of recurring malaria. *Plasmodium vivax* is carried by the female Anopheles mosquito. It is most widespread human malaria parasite species found mainly in Asia, Latin America and some parts of Africa (except sub Saharan Africa).

Plasmodium vivax is believed to have originated in Asia. But recent studies have shown that wild chimpanzees and gorillas throughout central Africa are endemically infected with parasites that are closely related to human *Plasmodium vivax*. These findings indicate that human *Plasmodium vivax* is of African origin. *Plasmodium vivax* accounts for 65% of malaria cases in Asia and South America. Malaria is the disease as old as humanity itself, and often called as the king millennia, malaria has played a major role in the history of mankind. Malaria is the fifth cause of death from infectious diseases worldwide (after respiratory) infections, HIV/AIDS, diarrheal diseases, and tuberculosis. It is a most serious vector - born disease and is one of the major causes of illness and death in tropical and subtropical region of the world. Inaddition to the direct effects it exerts by increasing premature mortality and morbidity. It is responsible for considerable economic worldwide prevalence of malaria is estimated to be approximately 300 million to 500 million clinical cases each year and an estimated mortality of 1 million per year. It has placed a heavy burden on tropical and subtropical countries for centuries. In December, 2014, WHO reported an estimated 5,84,000 deaths. Malaria mortality rates have fallen by 47% globally since 2000 and by 54% in the WHO region.

Unlike *Plasmodium falciparum*, *Plasmodium vivax* is capable of undergoing sporogonic development in the mosquito to lower temperatures. Nearly 2.15 billion people are at risk of *Plasmodium vivax* infection in 94 countries in the world and 16 million clinical cases occurs annually. The highest burden of *Plasmodiumvivax*infection is seen in Southeast Asia and South America. In most parts of Africa *Plasmodium vivax* infections absent because of the inherited lack of "Duffy antigen receptor" for chemokine on the surface of Red Blood corpuscles (RBCs) in the majority of people. However, there are few reports describing prevalence of submicroscopic *Plasmodium vivax* infection in certain parts of Africa, suggesting that either the parasite is evolving of use alternative receptors for erythrocyte invasionsonpopulation in those regions express low levels of Duffy antigen receptors.

2. GENETIC DIVERSITY OF PLASMODIUM FALCIPARUM

The intricate life cycle of the parasite needs a sophisticated array of proteins encoded by a genome of 23 Mb spread over 14 chromosomes and comprised of 5300 genes. Even within the same location, the parasite's genetic structure varies based on local epidemiological and demographic conditions.

Several proteins produced by P falciparum throughout different phases of its life cycle are polymorphic, which contributes to the parasite's varied genetic structure. The polymorphism is generally produced by change in sequence of the short tandem repetitions of the antigen which may generate immunodominant epitopes. There are seven variable blocks in MSP-1, a typical molecular marker for measuring genetic diversity, each separated by either conserved or semi-conserved areas (Fig 1). In each PfMSP-1 variable block, the isolates MAD20 and K1 represent two different variants. Recombination between these two typical kinds of MSP-1 polymorphism may be responsible for the majority of Plasmodium falciparum MSP-1 polymorphism (blocks 3, 4, 5). In Block 2, three allele families of K1, MAD20, and RO33 have been identified14. K1 and MAD20 type block 2 alleles differ in their quantity, sequence, organization, and point mutation polymorphism of tripeptide repeats and the flanking areas, respectively. There are no point mutations in the non-repetitive RO33 allele. A recombination event involving alleles from the Mad20 and RO33 families, resulting in the MR allele family, has also been discovered in Block 2.

To avoid immune responses triggered by previous exposure to different kinds of antigen, parasites may employ polymorphisms in repeats. Tandem repetitions may also induce T-cell-independent B cell responses that fail to create memory B cells or somatic hypermutation, leading to antibody affinity maturation, as a result. Genetic variety of Plasmodium falciparum (MSPs) is intensively investigated to assess the genetic diversity of Pf malaria in different transmission zones, since genetic diversity is an indicator of the dynamics of malaria transmission, complexity of infection and host immune response.

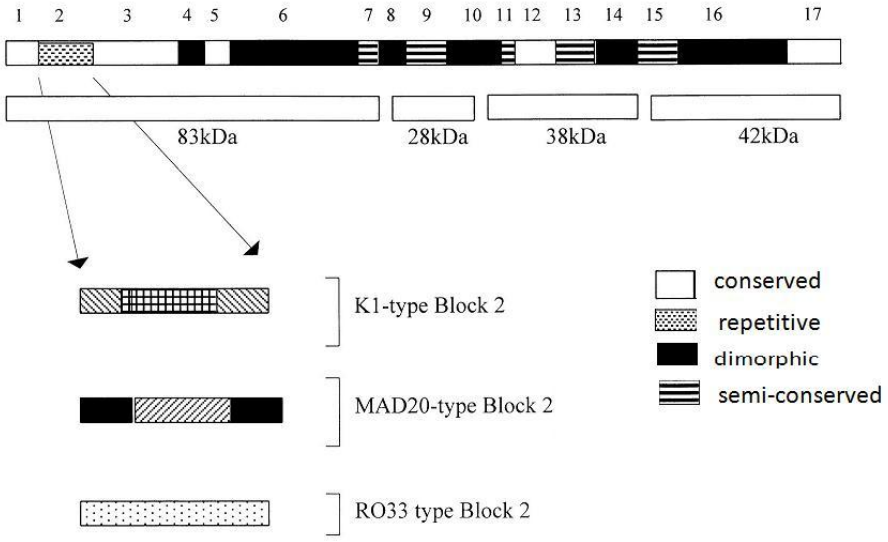

Fig 1: Schematic representation of MSP-1 of *P. falciparum.*

3. GENETIC DIVERSITY AND TRANSMISSION INTENSITY

Plasmodium falciparum genotypes from places with different endemicity and transmission characteristics have been shown to be linked.

Different malaria-endemic regions in the Southwestern Brazilian Amazon, southern Vietnam, and northern Tanzania were analyzed for MSP-1 locus allelic diversity. When compared to mesoendemic Vietnam, Tanzania's holoendemic population had a lower MSP-1 variety in human hosts. More than twice as many people in the Senegalese communities of Dielmo and Ndiop were infected with parasites than in Ndiop, where malaria was mesoendemic, in a study of parasite diversity. It was discovered that the number of genotypes per person with P. falciparum infection was greater than expected in a hypoendemic area along the Thai-Burma border. While a small number of PfMSP-1 have also been found in a similar endemic region. More variation was found in Myanmar's hypoendemic zone than in Thailand and Iran, but less than in other holoendemic regions of Senegal, Uganda, and Gabon.53 Similarly, the MSP-1 gene revealed substantial diversity in field isolates from India's eastern and north-eastern areas, similar to that found in South-East Asia, Papua New Guinea, and Latin America, places with lower mesoendemicity than those in Africa with higher holoendemicity. Hyperendemic Solomon Islands, compared to the mesoendemic region of Thailand in South East Asia, has also been shown to have a limited multiplicity of infection. Since transmission strength correlates with the number of genotypes discovered, a link exists, although it is unlikely to be linear. In lower endemicity areas like Brazil and Vietnam, the MSP-1 gene

type was shown to be distributed independently in the human host, but not in high endemicity areas like northern Tanzania and the Gambia. Parasite diversity is low in the Venezuelan Amazon, according to a recent research, although this might have more than one cause, such as the bottleneck effect. The greater the number of alleles present in the local parasite population and the greater the frequency of infections, the greater the allelic diversity.

4. GENETIC DIVERSITY AND DISEASE OUTCOME

Certain MSP-1 genotypes have been linked to increased risk of clinical illness as well as disease severity. In children with more clones, the risk of developing clinical malaria was greater. A lower chance of developing clinical malaria was shown to be associated with increased infection multiplicity in one research. In Tanzanian children with asymptomatic diseases, there was a larger average number of genotypes than in those with symptomatic disorders. Complexity of infections was observed to be lower in severe malaria isolates as compared to uncomplicated malaria isolates.

msp-1 allele class distributions and their connection to clinical malaria outcomes have been studied. Alleles of the MAD20 family were found to be overrepresented in severe malaria cases in Orissa, India, where malaria was highly endemic. There have been reports of a link between MSP-1 genotypes and the severity of the disease, where severe malaria was found to have no MSP-1 allele, or studies that failed to find any such association. Clinical disease is more likely when the MAD20 allele is present. As a result, those who had the K1 and MAD20 alleles were considerably less likely to get the symptomatic form of malaria. The RO33 variant was shown to be associated with illness severity and the K1 allelic family was the most prevalent in isolates from symptomatic patients, according to research.

5. GENETIC DIVERSITY AND HOST FACTORS

Differential age dependence on multiplicity of infection (MOI) has been shown in some molecular epidemiological field research, however this was not seen in other studies. Adults had the most complicated infections in Ndiop village, Senegal, while children had the fewest. Children beyond the age of two were also shown to have a greater degree of infection complexity than those under the age of one. However, in adults, the multiplicity of infection is dramatically reduced with age as assessed. Malaria endemicity has a significant impact on age-related changes in multiplicity of infection (MOI). MSP-1 fragments decreased in older

age groups in Dielmo, a highly endemic location, but there was no effect on infection complexity in Ndiop, a mesoendemic zone.

Additionally, the importance of the genetic makeup of a person's body has become widely recognized. Haemoglobin variant types, a+ thalassemia, and other genetic features may have an impact on the spread of P. falciparum and the existence of various genotypes. Two ethnically distinct African groups have different parasite multiplicity, which may be due to genetic variations. Host factors have a significant part in immunity, as evidenced by the Fulani tribes of Africa being less likely to clear infection by resistant parasites than Dogon or Malinke groups, showing a critical role for host factors.

6. CONCLUSION

Vivax malaria, which represents a relatively low level of parasitemia in peripheral blood, is a dangerous illness. Except for parasitemia, the infection's well-documented course has all the hallmarks of a pernicious disease associated with falciparum malaria, including severe illness and a deadly result. Regardless of how dangerous this parasite is, new research has shown that it is connected with a high level of morbidity and death.

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