Opinion
Volume 11:2, 2022

Journal of Malaria Control and Elimination

ISSN: 2470-6965 Open Access

Malaria Vaccines and Adjuvants for *Plasmodium falciparum*

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Introduction

Malaria, caused by the apicomplexan Plasmodium spp., is still one of the world's most dangerous infections for people and other animals, with high morbidity and fatality rates =. The World Health Organization (WHO) reported 228 million illnesses and 405,000 fatalities worldwide in 2018. Although eight Plasmodium species may infect people, the majority of malaria infections are caused by *P. falciparum* or *P. vivax*, although falciparum malaria is the leading cause of mortality. According to 2017 WHO data, African nations bear the lion's share of the worldwide malaria burden (90 percent). Malaria is a severe health hazard in Asian nations such as India, which accounts for 4% of the worldwide burden.

Malaria is still a severe health hazard in Asian nations, such as India, which accounts for 4% of the worldwide burden. As a result, the WHO-sponsored Special Programme for Research and Training in Tropical Diseases (TDR), the US National Institutes of Health, the UK Department for International Development, the Bill & Melinda Gates Foundation and other organisations have increased funding for research and development as well as other control measures (such as vaccination) to reduce malaria cases. WHO has also made greater efforts to deploy critical malaria commodities such as quick diagnostic tests, insecticide-treated mosquito nets, vector control, artemisinin-based combination therapy and pesticide resistance in malaria vectors [1].

Description

The life cycle of the malaria parasite is complicated. The legacy of Ronald Ross and Giovanni Battista Grassi set the groundwork for understanding the life cycle of the malaria parasite. Humans have pre-erythrocytic and erythrocytic phases, while the mosquito vector has a sexual life cycle. The pre-erythrocytic stage When an infected mosquito bites the host for a blood meal, the sporozoite is injected primarily into the dermis and occasionally straight into the blood vessels. Once in the liver, the sporozoite invades and infects the hepatocytes. Sporozoites proliferate rapidly inside the hepatocytes, producing 10–30,000 merozoites. Stage of erythrocytic differentiation. Merozoites penetrate red blood cells, where they develop into trophozoites and schizonts.

Each schizont produces 6–12 merozoites, which are discharged into the circulation (last one minute before infecting another RBC) and infect new RBCs. A few parasites grow into male and female gametocytes during the process. Gametocytes are produced and develop initially in the bone marrow (I–IV stages), then in the spleen (V stage). Mosquito life cycle/sexual stage A limited proportion of parasites divert from asexual reproduction and develop into male and female gametocytes, which enter the Anopheles mosquito's stomach after a blood meal [2].

Within the mosquito's midgut, male and female gametocytes grow into flagellated microgametes (eight) and a single macrogamete, respectively. A zygote formed by the union of a macrogamete and a microgamete develops into an ookinete through meiosis and enters the mosquito's gut wall to create oocysts and spawn sporozoites. After oocysts break, these sporozoites enter salivary glands and infect a new host following a mosquito bite [3].

P. falciparum transmission can be controlled by vector control methods such as pesticide spraying and/or the use of chemically treated mosquito nets, as well as antimalarial medications for prophylaxis and radical cure. The global strategy for combating P. falciparum include early detection, the use of bed nets and timely treatment with anti-malarial drugs. However, the parasite has gained resistance to all current anti-malarial medications. Chloroquine was a critical tool in several nations' efforts to eliminate malaria throughout the previous century. Resistance to chloroquine monotherapy arose in the 1970s and chloroquine-resistant P. falciparum parasites are now found worldwide.

Other medications, such as pyrimethamine, mefloquine, or artemisinin derivatives, were also created and gradually supplanted chloroquine. Because resistance to these medications evolved, combination treatments were created to treat *P. falciparum* infections. Combination therapy based on artemisinin have been significant in reducing the mortality toll. However, resistance to artemisinin and its companion medications has been a major worry in the recent decade. As a result, there is a need for innovative treatments and vaccines, notably for controlling and perhaps eradicating P. falciparum malaria. The purpose of this article is to discuss the present state of malaria vaccine research, with a focus on the use of adjuvants and their mechanisms of action [4].

Antibodies mediate protective immune responses in malaria infection through a variety of mechanisms, including inhibition of parasite motility, invasion, egress, adhesion and hepatocyte traversal ability, promotion of antibody-dependent complement-mediated sporozoite/merozoite lysis, phagocytosis, antibody-dependent cellular cytotoxicity, transmission-blocking activity and others. Surprisingly, ethnicity influences the formation of natural immunity against *P. falciparum*. In contrast to endemic inhabitants, who require two infections, the majority of non-immune Western tourists, if not all, only a single infection to elicit invasion inhibitory antibodies against *P. falciparum*.

Similarly, protection against malaria in newborns born in endemic areas correlates with pre-existing antibodies (i.e., maternal antibodies). Furthermore, youngsters (5 years) who are regularly exposed to the natural infection are more resistant to the severe clinical signs of malaria. The development of *P. falciparum* antigen-specific B cells to generate monoclonal antibodies and the passive transfer of immunoglobulins have been appealing tactics in malaria research. Many malaria candidate vaccines have been developed to elicit an effective antibody response. Antibodies against sporozoites or neo-antigens expressed on the surface of infected hepatocytes mediate protection by blocking or restricting pre-erythrocytic infection and development. Anti-sporozoite antibodies have been shown to inhibit sporozoite motility in the dermis and liver, destroy sporozoites in the skin, aid opsonization and phagocytosis by monocytes or macrophages in the spleen or liver, inhibit sporozoite invasion into hepatocytes and inhibit sporozoite development within hepatocytes [5].

Conclusion

To summarize, there is a need to coordinate research in multiple directions to develop an efficient malaria vaccine, such as a clear understanding of the malaria parasite's life cycle, identification and characterization of targets of interest with a broad range of strains covered, identification of a suitable

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Received: 01 March, 2022, Manuscript No. mcce-22-66942; **Editor assigned:** 02 March, 2022, PreQC No. P-66942; **Reviewed:** 09 March, 2022, QC No. Q- 66942; **Revised:** 17 March, 2022, Manuscript No. R-66942; **Published:** 24 March, 2022, DOI: 10.37421/2470-6965.2022.11.179

adjuvant, which could enhance the magnitude and quality of the immunogenicity of antigen(s) without age factor and characterization and selection of a suitable 'permissive' adjuvant.

Acknowledgement

None.

Conflict of Interest

No potential conflict of interest was reported by the authors.

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How to cite this article: Nicoletta, Basilico. “Malaria Vaccines and Adjuvants for Plasmodium falciparum.” Malar Contr Elimination 11 (2022):179.