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Immune Responses during Pre-erythrocytic Stages of Malaria

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Introduction

Mosquitoes carrying *Plasmodium spp.* inject sporozoites into a mammalian host's skin during a blood meal. When they enter the host's bloodstream, they infect the liver and spread disease. After undergoing a peaceful metamorphosis, merozoites enter the bloodstream and cause malaria symptoms and transmission. Chemotherapeutic and immunoprophylactic treatments can halt the illness's progression to clinical malaria during the silent pre-erythrocytic stage, which is a disease bottleneck. Despite its modest efficacy, RTS,S/AS01, the only malaria vaccine on the cusp of approval, inhibits sporozoite invasion primarily by blocking antibodies to the CSP protein, a crucial component of the sporozoite pellicle.

Description

Through immunisation with radiation-attenuated sporozoites, genetically attenuated sporozoites, or chemoprophylaxis with infectious sporozoites, animals and people can receive sterile protection against malaria; nevertheless, sporozoite-based live vaccines have substantial deployment difficulties. The protection brought on by sporozoites in the pre-erythrocytic stages is mediated by antibodies against the sporozoite and CD8+ T lymphocytes against peptides presented by MHC class I molecules in infected hepatocytes. Because of this, the identification of malaria antigens produced in the sporozoite and liver stages may result in the creation of new vaccine candidates that can be utilised as standalone or in combination recombinant protein-based virus-like particles or sub-unit virally-vectored vaccines [1].

Invertebrates, predominantly Anopheles mosquitos, and vertebrate mammals are the two very different hosts that Plasmodium sp. parasitizes. They are polymorphic, obligate intracellular parasites with a complex life cycle that includes both an asexual and sexual stage. Although infections with these pathogens have been seen in other primate species, P. falciparum, P. vivax, P. ovale, P. malariae, and P. knowlesi are the five species that are known to cause naturally occurring infections in humans [2].

Female Anopheles mosquitoes of diverse kinds that are Plasmodium-infected spread malaria. A female infected mosquito injects about 100 sporozoites into the skin of a mammalian host during a blood meal. The sporozoites scan the epidermis for capillaries, enter the bloodstream of the host within minutes, and eventually infect liver cells. Before successfully infecting one hepatocyte and residing in a parasitophorous vacuole with specific activities, the sporozoites in the liver transit through numerous hepatocytes. The hepatic infection caused by P. falciparum lasts around 7 days and is asymptomatic. The hypnozoite, a latent form that P. vivax and P. ovale can produce, can remain in the liver and cause relapses months or years later by infecting the bloodstream. Following asexual schizogony, parasites produce tens of thousands of merozoites in hepatocytes. Parasites called exo-erythrocytic forms reside inside hepatocytes (EEFs). The liver stage and sporozoite invasion of the mammalian host are both parts of the pre-erythrocytic stage, commonly referred to as the exo-erythrocytic cycle. To stop the disease from advancing to clinical malaria, chemotherapeutic and immunological prophylactic therapy are appropriate at this stage of the illness [3].

The parasites enter the bloodstream and infect erythrocytes, where they proliferate asexually after merozoite egress from the afflicted liver cell. This is the symptomatic and extensively researched erythrocytic cycle of malaria. Merozoites rupture the host cell and infect fresh erythrocytes after infecting them and evolving into trophozoites, which eventually evolve into schizonts. On the other hand, some trophozoites develop into gametocytes. The parasite restarts sexual development when a female mosquito ingests a blood meal from an infected mammalian host that contains at least one female and one male gametocyte. Inside the mosquito, during the sexual stage, or sporogonic cycle, gametes develop into ookinetes, which subsequently pass through the midgut wall to form oocysts. Oocysts produce a large number of sporozoites [4].

Malaria-Related Immune Response

The regulation and causation of disease are influenced by molecular elements of the innate and adaptive immune systems. There are three ways to develop clinical immunity to malaria: through sickness, symptomatic infection, and partial parasitemia. In malaria-endemic regions and in people who have experienced several infections over the years (8–15 years), premunition (absence of fever with infection and lower parasitemia density) is observed, leading to innate immune responses that lessen the chance of clinical disease. The term was developed in the early 1900s during epidemiological studies with people from endemic regions who can control parasitemia and develop a subclinical sickness. While the immune response evoked is strong, it is not sterilising immunity. It has a slow acquisition rate, is only present in holo- or hyperendemic regions, is soon lost, is strain and IgG dependent, and is directed towards blood-stage parasites. Although the exact mechanism of protection is unknown, evidence points to cytophilic antibodies and memory cells produced after repeated infections with different Plasmodium strains as potential contributors [5].

An innate immune response, which serves as the body's initial line of defence against Plasmodium infection, is produced. This is followed by an adaptive immunological response, which consists of T-cells, B-cells, and antibodies. Spz is injected into the skin by a mosquito bite and can remain there for up to 6 hours. This retention has an impact on the site of antigen presentation, as well as the location and kind of response elicited.

Conclusion

RBC The parasite's main protein source is haemoglobin. Heme, a lipophilic prosthetic group that is very deadly to the parasite, is released by the hydrolysis of haemoglobin. As a result, heme must be detoxified, which is done by turning it into the crystalline, insoluble compound hemozoin (Hz). When P. falciparum infects a host cell, Hz binds DNA in the phagolysosomes and cytosol, activating TLR9 in response to nucleic acids, NLRP3, AIM2, and other cytosolic sensors. For CD8+ T-cells to recognise antigens during the hepatic stage, MHC class I molecules must be produced on all nucleated cells and exposed on the surface of hepatocytes. By preventing Spz from entering hepatocytes and neutralising Spz antibodies, immunoglobulins can prevent disease progression in a number of ways. Mrz can be opsonized at

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the erythrocyte stage by specific antibodies that cause cell-mediated death or inhibit RBC invasion and block the proteins that bind to substances on the cell surface.

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