Opinion

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Immune Evasion Mechanisms used by Plasmodium and the Immunopathogenesis of Malaria

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Abstract

Malaria is a parasitic disease caused by the Plasmodium species, transmitted by the female Anopheles mosquito. It remains a significant public health issue, with an estimated 229 million cases and 409,000 deaths worldwide in 2019. The immune response to Plasmodium infection is complex, involving both innate and adaptive immune mechanisms. In this essay, we will discuss the immunology and immunopathogenesis of malaria, including the immune response to infection, the role of immune evasion strategies employed by the parasite and how these factors contribute to the development of disease.

Keywords: Malaria, Parasitic disease, Plasmodium infection, Immunopathogenesis, Immune mechanisms.

Introduction

Upon infection with Plasmodium, the innate immune system is the first line of defence. Monocytes, macrophages and dendritic cells recognize the parasite through pattern recognition receptors (PRRs) such as Toll-like receptors (TLRs) and NOD-like receptors (NLRs). This recognition leads to the production of proinflammatory cytokines such as TNF-α, IL-1β and IL-6, which activate other immune cells and contribute to the recruitment of more immune cells to the site of infection. One of the key features of the immune response to Plasmodium infection is the activation of the complement system. The complement system is a group of plasma proteins that function to eliminate invading microorganisms. Activation of the complement system results in the production of membrane attack complexes (MACs), which can directly kill the parasite or infected host cells [1, 2].

The adaptive immune response is also critical in controlling Plasmodium infection. T cells, B cells and antibodies all play important roles in the immune response to the parasite. T cells, particularly CD4+ T cells, are important in providing help to B cells and in activating other immune cells. CD8+ T cells are also involved in controlling the infection by directly killing infected cells. Antibodies, which are produced by B cells, can directly neutralize the parasite, preventing it from invading host cells [3]. Despite the immune system's best efforts, Plasmodium has developed several strategies to evade host immunity. One such strategy is antigenic variation. Plasmodium has a large number of surface proteins that it can switch between during the course of infection. By changing the surface antigens it expresses, the parasite can evade immune recognition and continue to proliferate within the host [4].

Another strategy employed by Plasmodium is the sequestration of infected red blood cells (RBCs) in the microvasculature of organs such as the brain, where they can avoid detection by immune cells. The parasite expresses surface proteins on infected RBCs that allow them to adhere to endothelial cells, preventing their clearance by the spleen. The immune response to Plasmodium infection can also contribute to the development of disease. For example, the production of proinflammatory cytokines during the innate immune response can lead to the development of fever and other symptoms associated with malaria. Additionally, the sequestration of infected RBCs in the microvasculature can lead to the development of organ-specific complications such as cerebral malaria, which is characterized by the sequestration of infected RBCs in the brain [5].

Conclusion

The immune response to Plasmodium can also contribute to the development of anaemia. The destruction of infected RBCs and the production of proinflammatory cytokines can lead to a decrease in the production of new RBCs, leading to anaemia.

In conclusion, the immunology and immunopathogenesis of malaria is a complex topic. The immune response to Plasmodium infection involves both innate and adaptive immune mechanisms and the parasite has developed several strategies to evade.

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