

Immunology and Infection by Protozoan Parasites

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Introduction

When protozoa enter the blood stream or tissues they can often survive and replicate because they adapt to the resisting natural host defences. The interaction of immune system with infectious organisms is a dynamic interplay of host mechanisms aimed at eliminating infections and microbial strategies designed to permit survival in the face of powerful effector mechanisms. Protozoa cause chronic and persistent infections, because natural immunity against them is weak and because protozoa have evolved multiple mechanisms for evading and resisting specific immunity [1].

The protozoan parasites, that cause necessary diseases moving millions of individuals worldwide particularly within the tropical and climatic zone areas, area unit chargeable for high mortality and morbidity. Most of those parasites area unit transmitted by insect vectors and invade a spread of various tissues in their class hosts [2]. Prophylactic and therapeutic methods area unit so much from satisfactory. Indeed, though important progress has been created in our understanding of the immune reaction to parasites, no definitive step has nonetheless been with success done in terms of operational vaccines against parasitic diseases. Moreover, some medicine area unit offered, however there are a unit issues over their effectiveness, toxicity, and emergence of resistant strains [3].

Natural and specific immune response to protozoa

Different protozoa vary greatly in their structural and biochemical properties and stimulate distinct patterns of immune responses and have evolved unique mechanisms for evading specific immunity [4]. Protozoa activate quite distinct specific immune responses, which are different from the responses to fungi, bacteria and viruses. Protozoa may be phagocytosed by macrophages, but many are resistant to phagocytic killing and may even replicate within macrophages. *T. brucei gambiense* is the best example of protozoa which can induce humoral immune response because of its extra-cellular location. In *Leishmania sp.* infections, cellular defense mechanisms depend upon CD4+ T-lymphocytes and activate macrophages as effector cells that are regulated by cytokines of Th1 subset. *Plasmodium sp.* is a protozoa which show the diversity of defence mechanisms which can be cellular or humoral, depending on Ag and protozoa's location [5].

Immune evasion mechanisms of protozoa

Different protozoa have developed remarkably effective ways of resisting specific immunity: anatomic sequestration is commonly observed with protozoa *Plasmodium* and *T. gondii*; some protozoa can become resistant to immune effector mechanisms [6,7]: *Trypanosoma*, *Leishmania* and *T. gondii*; some protozoa have developed effective mechanisms for varying their surface antigens: *Plasmodium* and *Trypanosoma*; some protozoa shed their antigen coats, either spontaneously or after binding with specific antibodies: *E. histolytica*; some protozoa alter host immune response by nonspecific and generalized immunosuppression (abnormalities in cytokine production, deficient T cell activation): *Trypanosoma*, *Leishmania*, *Toxoplasma*, *Entamoeba* [8].

Conclusion

Protozoa activate numerous, different immune mechanisms in human body. Evolution, progression and outcome of diseases depend upon these mechanisms. Resent progresses in research have defined and selected Ag as candidates for new vaccines. Better definitions regarding the role of cytokines in protozoan infections will facilitate rational development of cytokines and cytokine antagonists and their use as immunotherapeutic agents.

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Conflicts of Interest

The author has no known conflicts of interested associated with this paper.

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