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Immunology and Infection by Protozoan Parasites

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In od c ion

When protozoa enter the blood stream or tissues they can o en survive and replicate because they adapt to the resisting natural host defences. e 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 e ector 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 speci c immunity [1].

e 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 de nitive step has nonetheless been with success done in terms of operational vaccines against parasitic diseases. Moreover, some medicine area unit o ered, however there are a unit issues over their e ectiveness, toxicity, and emergence of resistant strains [3].

Na al and peci c imm ne e pon e o p o o oa

Di erent protozoa vary greatly in their structural and biochemical properties and stimulate distinct patterns of immune responses and have evolved unique mechanisms for evading speci c immunity [4]. Protozoa activate quite distinct speci c immune responses, which are di erent from the responses to fungi, bacteria and viruses. Protozoa may be phagocytozed 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 e ector cells that are regulated by cytokines of 1 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].

Imm ne e a ion mechani m of p o o oa

Di erent protozoa have developed remarkably e ective ways of resisting speci c immunity: anatomic sequestration is commonly observed with protozoa Plasmodium and T. gondii; some protozoa can become resistant to immune e ector mechanisms [6,7]: Trypanosoma, Leishmania and T. gondii; some protozoa have developed e ective mechanisms for varying their surface antigens: Plasmodium and Trypanosoma; some protozoa shed their antigen coats, either spontaneously or a er binding with speci c antibodies: E. histolytica; some protozoa alter host immune response by nonspeci c and generalized immunosuppression (abnormalities in cytokine production, de cient T cell activation): Trypanosoma, Leishmania, Toxoplasma, Entamoeba [8].

Concl ion

Protozoa activate numerous, di erent immune mechanisms in human body. Evolution, progression and outcome of diseases depend upon these mechanisms. Resent progresses in research have de ned and selected Ag as candidates for new vaccines. Better de nitions 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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Con ic of In e e

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