ISBN: 978-93-91216-76-4

Chapter - 5

Host Immune Responses and Drug Interaction against *T. evansi*

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Abstract

All the parasites enter into body through various pathways invading the host immune system and complete the life-cycle and trypanosomes are no different from this. To ensure the survival and propagation, the parasites exploit and manipulate the host defence factors to restore normal body function during infection. The manipulation by parasite causes evasion of host immune response and at the same time the host ends up sustaining some self-inflicted collateral tissue damage. When there is trypanosoma infection, the activation of effector and suppressor cells occurs, and thus balancing between two contradictory branches of immunity that will determine the degree of resilience of host to the agent. The trypanosomes invade to the host immunity directly or indirectly. Parasite related factors like antigenic variation is the direct factor whereas, indirectly the suppressor cells are inducted following infection. Cell like suppressive macrophage, regulatory T cell and myeloid derived suppressor cell are known to cause suppression of host immunity in trypanosomiasis. During infection, both the innate and adaptive immunity fight against the protozoa invasions. Innate immunity is mainly contributed by macrophages. During the infection with trypanosomes, activation of both alternative and classical pathway occurs. The outcome of infection is regulated by both helper T cells and cytotoxic-T cells play important. The parasite escape from the host immune response through antigenic variation. Apart from this, enormous VSG of trypanosomes are shedded in the circulation that forms immune complexes with antibodies, thereby deposition of “membrane attack complexes” are prevented the on the parasitic cell membrane that ultimately avoids lysis of *Trypanosoma* mediated by complement. Be that as it may, examination into new trypanocides has stayed lacking for quite a long time, prompting a circumstance where the couple of mixtures accessible are losing viability because of the rise of drug resistant parasites.