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Commentary

Strategies of *Trypanosoma Cruzi* to Disrupt the Host Immune System

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DESCRIPTION

Microorganisms have developed a wide variety of tactics to trick the host immune system. *Trypanosoma cruzi*, the protozoan parasite that causes Chagas disease, is a notable example of such modifications. Both peripheral and central lymphoid tissues are among the host tissues that this parasite attacks. The parasite must resist a systemic acute reaction that results from rapid colonization of the host. In actuality, the parasite impairs both innate and adaptive immunity. It acts on host sialic acid-binding Ig-like lectin receptors to prevent dendritic cells from performing their antigen-presenting activity. These receptors also reduce $CD4^+$ T cell responses, and we provided proof that the inhibitory effects on CD4 T cells depend on the sialylation of mucins generated by parasites. Chagas disease is brought on by the protozoan parasite *Trypanosoma cruzi* (*T. cruzi*). About 30% of patients experience cardiomyopathy, neuropathies, and dilatations of the colon or oesophagus at some point in their lifespan when the disease develops into a symptomatic chronic phase. Several theories, such as autoimmune reactions and parasite-caused tissue damage, have been taken into consideration for the pathophysiology of Chagas disease. *T. cruzi* is a hemoflagellate parasite with a convoluted life cycle that infects vertebrates by biting triatomine (reduviid) insects that feed on blood.

Epimastigotes and metacyclic trypomastigotes, which are present in the insect vector, and blood-form trypomastigotes and intracellular amastigotes, which are present in vertebrate hosts, are at different stages of the life cycle. The parasite must contend with a complex immune system in vertebrates that uses circulating cells, chemicals, and specialised tissues and organs. The trypanosome alternates between intracellular proliferative forms and nonproliferative, infectious external trypomastigotes in order to evade host defence mechanisms. The many morphological life cycle forms are connected to variations in gene expression that are adaptive. The protein-coding sequences of *T. cruzi* have been predicted by genomic analysis, and virulence factors and gene clusters implicated in subverting host cell immunity have been annotated. The vast spectrum of host cells that the parasite targets, primarily nonphagocytic cells, is caused by these factors. *T. cruzi* principally uses inhibition of the mononuclear phagocyte system and manipulation of the complement system to evade the immune system.

Other protozoan illnesses like leishmaniasis and African trypanosomiasis also exhibit downregulation of phagocytic activity, indicating evolutionary convergence in the phylogeny of the protozoan parasites. *T. cruzi*, on the other hand, avoids macrophage microbicidal activity by escaping from the phagolysosome to the host cell cytoplasm, where it replicates, in contrast to other protozoan parasites that hinder the formation of phagolysosomes. Additionally, it prevents the production of cytokines that are released by infected macrophages. The parasite only weakly activates TLRs, and a large cysteine protease limits macrophage activation by blocking the NF-κB P65 pathway and preventing IL-12, a proinflammatory cytokine, from being produced. In this circumstance, macrophage infection promotes the release of anti-inflammatory cytokines like IL-10 and TGF-β, which hinder the maturation of protective immune responses and promote the spread of infection.

T. cruzi, however, comes in a number of natural strains, and it appears that their immune modulatory effects vary by strain, a characteristic that may affect how the parasite and host interact. According to comparative RNA sequence analysis and phylogenetic reconstructions, the different strains diverged roughly 100 million years ago. In natural reservoirs, the diverse strains cohabit dynamically. Triatomine bugs from domestic and peridomestic regions have really been discovered to include various combinations of *T. cruzi* strains. Moreover, rather than occurring at the level of a single strain, immune evasion may take place at the population level. Depending on the parasite strain, the big trans-sialidase family of genes encodes different $CD8^+$ T cell immunodominant epitopes. The cytotoxic mechanisms used by T cell-mediated cytotoxic T cells to stop parasite growth inside the host differ, and $CD8^+$ T cells are essential for managing the intracellular parasite.

The animal kingdom's oldest inhabitants are protozoa, which have developed into one of the planet's most dominant life forms. The *Trypanosome cruzi*, kinetoplastid protozoa, originated from this evolutionary branch. The fact that these parasitic, unicellular

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creatures have survived to the present day is mostly due to their effective reproductive system, which results in enormous numbers of offspring through short generation durations and a quick maturation cycle. These characteristics cause severe infections that have an acute phase that vigorously stimulates the host immune system. Yet, the virus and its host engage in intricate molecular and cellular interactions during the acute phase, where the parasite can take advantage.