Editorial

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# Host-parasite Relationship is Critical to Understand Immune Response against Parasite Infection

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## Editorial

Myeloid cells are part of the innate immune system, which includes neutrophils, basophils, eosinophils, mast cells, and natural killer cells. Monocytes, macrophages and dendritic cells (DCs) are part of the mononuclear phagocyte system (MPS) and play an important role in control of disease, but can contribute to the establishment of persistent infections [1,2].

The MPS is the first line of defense against infections agents and provide an immediate defense against infections in tissues. The MPS respond to parasites through germline-encoded host receptors that recognize pathogen patterns (PRRs) and their respective pathogen-associated molecular patterns (PAMPs). In addition, they are responsible for confer specificity to the host innate system by activation of Toll-like receptors (TLRs). For example, different classes of DCs recognize microbial molecules via TLRs and respond to PRRs according to their TLR expression [3,4].

At the same time, parasites are able to developed mechanisms to escape the host immune response. These mechanisms allow the resistance of the parasite to immune response and lead to a successful infection process.

In *Trypanosoma cruzi* plasma membrane associated trans-sialidase enzyme is involved in parasite survival in the vertebrate host by invasion and escape from phagolysosomes. Furthermore, trans-sialidase enzymes are stage-specific molecules and it has different roles in parasite internalization. For example, GP82 induces biogenesis of the parasitophorous vacuole and host cell invasion [5].

*Trypanosoma brucei* present a system of antigenic variation that changes frequently the surface antigens and allows the parasite to evade the immune system recognition and humoral response. The antigenic variation is generate by mutation or recombination and challenges the immune system continually by new epitopes. In addition, the antigenic variation determinate whether human can be infected. For example, *T. brucei* and *T. brucei congolense* are unable to infect humans. In contrast, *T. brucei rhodesiense* and *T. brucei gambiense* are able to lead to a fatal disease due to the proliferation of parasites in the blood and tissues [6].

In the case of *Leishmania sp.*, this parasite has a great evasion strategy. This protozoan is able to suppress the microbicidal functions of macrophages by altering signaling pathways. *Leishmania* suppress macrophage activity by modulation of expression of cytokines, production of nitric oxide and reactive oxygen species and reduction of antigen presentation [7].

In order to survive, *Plasmodium falciparum* have to change its surface antigens in each phase of live cycle. This way, the expression of membrane surface proteins is stage-specific and tends to be highly polymorphic and antigenically variable [8].

The most relevant characteristic of *Entamoeba histolytica* is its cytolytic activity. This protozoan contains hydrolytic enzymes and cysteine proteases that damage host cells and tissues. The proteases interfere with the humoral immune response by degradation of IgA and IgG antibodies. The *E. histolytica* is also able to escape the lytic effect of complement and the inflammatory response by inactivation of C3 and C5a mediators [8].

In summary, more studies of parasite antigens must be done to identify specificity for each parasite and to clarify the immune response against different parasites. This is critical to development of new methods to control of disease in endemic areas.

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