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Perspective

Trypanosoma cruzi: Subvert the Host's Immune Response

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DESCRIPTION

Trypanosoma cruzi (*T. cruzi*) acts on the MAPK pathway to avoid the host's immunological response, although these actions are incompletely understood due to a lack of research. However, it is generally known that *T. cruzi* invades cells. In order to facilitate entry and survival inside host cells, cruzi starts to subvert signaling pathways and utilize host molecules. For instance, host Protein Kinase D1 (*PKD1*) and cortactin are both recruited by *T. cruzi* Extracellular Amastigotes (EAs) to cause *PKD1* autophosphorylation and cortactin activation by ERK, which then results in the recruitment of host actin and parasite entrance into HeLa cells. *T. cruzi* and *Leishmania* species can cause macrophages and dendritic cells to activate ERK1/2 but not p38 MAPK, as well as to produce more *IL-10* and less *IL-12*. To enable parasite evasion of the host immune response, these effects hinder the development of an effective Th1 inflammatory response.

In dendritic cells and macrophages, some *T. cruzi* molecules are produced, activating Toll-Like Receptors (TLRs) such *TLR2*, *TLR4*, or *TLR9*. As a result, p38 MAPK is activated and *IL-12* is produced, promoting an inflammatory reaction. The findings of who demonstrated that dendritic cells exposed to *T. cruzi* Antigens (TcAgs) and TLR ligands elicited p38 phosphorylation that was dependent on synergism between *T. cruzi* antigens and macrophage Migration Inhibitory Factor (MIF). As a result, *IL-12* production was increased, promiting a Th1-type response.

However, it is well known that various other molecules of such pathogens function against the host's infected cells and signaling pathways, subverting the host's immune response against the parasite, despite the fact that some parasite molecule activates a pro-inflammatory immune response.

Tc52 is one of the *T. cruzi* protein that disrupts the host's immune response. Tc52 is a 52 kDa protein that has both virulence and immunomodulatory activities. It has two homologous domains that are significantly homologous to glutathione S-transferases. The *Tc52*-mediated suppression of T cell proliferation is induced when a 28 kDa peptide fragment generated from the C-terminal part of *Tc52* localizes in the cytoplasm and exhibits mitogen-dependent cytokine and chemokine-like activities.

In order to boost the secretion of *IL-10* and suppress *IL-12*, this peptide modifies the genes that encode *IL-10* and *IL-12*. Furthermore, MAPKs are most likely the mediators of the processes described above.

Other proteins that influence the immune system are Glycosylphosphatidylinositol (GPI)-anchored mucins and Trans-Sialidases (TS) are involved in *T. cruzi* infection. AgC10, a 40–50 kDa GPI-anchored mucin, prevents the development of a Th1 response by inhibiting TNF and *IL-12* release in a p38 MAPK inhibition–dependent way. In macrophages treated with *T. cruzi*, researchers found that ERK1/2 activation was associated to a reduction in *IL-12* with GPI and GPI-mucins, supporting their role in regulating the host's immunological response.

CONCLUSION

They also demonstrated that GPIs and GPI-anchored mucins might activate p38 MAPK later than ERK1/2, which would increase the production of *IL-12* and have the opposite effect on the immune response's regulation by promoting a Th1 response.

Activation of ERK1/2 has been connected to *T. cruzi* TS. Recent research has supported the function of TS in the imbalance of the Th1 cell response toward a Th2 phenotype caused by *IL-10*-stimulated secretion. However, the specific processes through which *T. cruzi* subverts the host's immune response are still unknown, despite existing knowledge of its methods.

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