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Perspective

Infection and Host Lipid Metabolism of *Trypanosoma cruzi*

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DESCRIPTION

The etiological agent of Chagas Disease (CD) is *Trypanosoma cruzi* (*T. cruzi*). In the endemic regions of South and Central America, CD is spread by coming into contact with triatomine bug faeces (the kissing bug). *T. cruzi* can enter a wound or mucosal membrane by scratching when an insect feeds on the blood of a human and excretes on the skin. Disease transmission in these locations has significantly decreased as a result of successful vector control initiatives. Congenital transmission, organ transplantation, and blood transfusion are all ways that CD spreads in nonendemic areas.

The parasite interacts with a variety of host immunological and metabolic variables during the *T. cruzi* infection phase. In the last 10 years, there has been a focus on the intimate connection between host lipid metabolism and *T. cruzi* infection. The connection between *T. cruzi* and components of the host's cholesterol transport and storage system, including macrophages, adipocytes, Low Density Lipoprotein (LDL), and High Density Lipoprotein (HDL), has been revealed by a number of research groups.

The course of CD occurs in three stages: acute, indefinite, and chronic. The majority of infected people are asymptomatic while carrying the lifelong infection, but others develop severe symptoms right away. Unspecific symptoms, including fever, nausea, diarrhoea, and rash, as well as severe ones like a chagom, a swollen inflammatory lesion at the site of the parasite entry unilateral periorbital edoema (Romana's sign), lymphadenopathy, and hepatosplenomegaly may appear in infected people during the acute stage. The majority individuals make it through the acute period and reach the extended indeterminate stage, which lasts for life, without showing any overt disease signs. However, chronic CD, which includes serious symptoms like megaesophagus, megacolon, and chronic heart disease, develops in 30% of individuals.

Cruzi has a complicated life cycle and goes through a number of changes when it infects a person. In the triatomine vector, the parasite primarily lives in its epimastigote form. In the vector's hind intestine, it changes into a metacyclic trypomastigote, which is then excreted to infect a human host. Once inside the host, the metacyclic form infects a variety of phagocytic and nonphagocytic cells, including monocytes, neutrophils, mast cells, and macrophages (i.e., epithelial cells, endothelial cells, fibroblasts, and mesenchymal cells). Trypomastigotes become intracellular amastigotes after infection and divide by binary fission. Amastigotes change back into blood trypomastigotes after the division process is finished, which allows them to escape the cell and infect nearby cells or circulate in the blood.

Macrophage lipid bodies and *T. cruzi* infection

The lipid-rich organelles known as Lipid Bodies (LB), also known as lipid droplets or adiposomes, are found in practically all living things. Lipid bodies are distinctively enclosed by a monolayer of phospholipids, unlike other organelles. Neutral lipids, particularly triacylglycerol and sterol esters, as well as other potential membranous structures, are abundant in the lipid body's centre. In the past, lipid bodies were assumed to play a role in neutral lipid storage and transport, but more recent studies have revealed that they also play a crucial role in the control of host immunological responses. In cells involved in inflammatory processes, lipid bodies play a role in the synthesis of paracrine mediator eicosanoids. Numerous inflammatory diseases, including atherosclerosis and mycobacterial infections, cause leukocytes to accumulate more lipid bodies. Host macrophages are highly active during acute *T. cruzi* infection and will prevent parasite replication.

Host adipose tissue and *T. cruzi* infection

One of the largest organs in the host is adipose tissue. Adipocytes, pericytes, monocytes, macrophages, and endothelial cells are only a few of the diverse cell types that make up this tissue. Adipose tissue has long been thought to have the purpose of storing energy. Triglycerides and cholesterol esters are stored in lipid droplets, which make up more than 95% of the mass of adipocyte cells. However, it has recently come to light that adipose tissue also serves to regulate metabolism, the

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neuroendocrine system, and the immune system in addition to storing energy. Adipokines, which are important regulators of lipid homeostasis and immunological processes, such as adiponectin, leptin, and resistin, are found in abundance in adipose tissue.

The discovery that individuals with *T. cruzi* infection had higher occurrences of diabetes suggests that metabolic dysfunction contributes to CD development. Higher parasitemia and mortality following *T. cruzi* infection were seen in chemically produced diabetes mice as well as genetically predisposed diabetic animals with faulty leptin receptors, suggesting that the dysregulation of host metabolism may be advantageous for parasite survival in the host. The main cell type involved in metabolic dysregulations such as diabetes is the adipocyte.

CONCLUSION

The host lipid metabolism is a complex mechanism involving numerous variables. Both the immune system and other energy metabolism systems interact with it. Growing interest is being paid to how the host's lipid metabolism affects how it reacts to infectious pathogens. This review may help with a more thorough understanding of how *T. cruzi* interacts with the host's lipid metabolism as well as the function of lipids in *T. cruzi* pathogenesis. We have convincingly demonstrated how *T. cruzi* interacts with a number of distinct elements involved in host lipid metabolism. Future studies on these interactions and the function of lipids in the pathogenesis of *T. cruzi* will be very beneficial.