MedicalXpress

Carbohydrate-binding proteins mitigate parasitic infection in heart tissue

October 9 2015

Chagas disease is the main cause of infectious heart disease in Latin America. Researchers from the INGEBI and IBYME Institutes in Argentina explored the effect of glycan binding protein interactions between the human host and *Typanosoma cruzi* parasite. They found that a glycan binding protein expressed in humans modified the infection in cells of the heart muscle, showing the importance of galectins in the response to parasite infection.

Galectins are a group of carbohydrate-binding proteins which are used both inside and outside the cell for signalling and regulation. Previous research has indicated their involvement in parasite infections, and the galectin Gal-1 has been shown to be up-regulated in heart cells exposed to low oxygen or undergoing inflammation. Gal-1 is also up-regulated in heart tissue from patients with severe chronic Chagas cardiomyopathy.

In this study, the researchers examined 28 patients, 19 with cardiac symptoms and 9 who were in the asymptomatic phase of the disease. Comparing them with 42 non-infected individuals demonstrated that the patients with Chagas disease had higher Gal-1 levels whether symptomatic or not. Studies of infected heart tissue cells showed that *T. cruzi* increased Gal-1 release outside of the cells, without affecting the intracellular expression. Gal-1 was demonstrated to mitigate cellular parasite infection and prevent early signs of heart cell apoptosis but did not bind to the parasite itself. The researchers also examined mice lacking the gene for Gal-1 and found that these mice had increased parasitemia and decreased survival rate following *T. cruzi* (Tulahuén

strain) infection compared with wild type mice. This work also highlights the relevance of parasite genetic background in Gal-1-mediated control of *T. cruzi* infection in vivo.

Based on these results, the researchers hypothesize that Gal-1 may recognize and bind to glycans on the heart cell surface used for parasite invasion, preventing parasite attachment and probably activating the immune response to the parasite by cytokine release. Conversely, *T. cruzi* may alter surface glycophenotype of cardiac cells, restricting Gal-1 interaction. The role of galectins in parasite-host cell interactions is a fascinating but under-explored area, and more research should provide a clearer pattern of the importance of these glycan binding proteins in mitigating parasitic infections.

More information: Alejandro F. Benatar et al. Galectin-1 Prevents Infection and Damage Induced by Trypanosoma cruzi on Cardiac Cells, *PLOS Neglected Tropical Diseases* (2015). DOI: 10.1371/journal.pntd.0004148