Studies of the participation of lectins in the progress of infection of mammalian cells by *Trypanosoma cruzi* reveal a pivotal role for galectin-3.

Souza, R.F.O.; Do Amaral, M.J.; Henriques, D.G.; Fernandes, V.C.; Carvalho, M.A.; De-Melo, L.D.B.

Dep. de Biotecnologia, IFRJ-Campus Rio de Janeiro, RJ, Brazil.

**Introduction:** The etiologic agent of Chagas disease is the protozoan *Trypanosoma cruzi*, in humans the pathogenesis progresses with intracellular development in several tissues. The early stages of infection involve adhesion, recognition, signaling and invasion, through interactions between receptors and surface molecules of the host with the parasite. Surface lectins in the host cells, as galectin-3, can mediate host-parasite interactions, as previously seen in dendritic and smooth muscle cells. **Objective:** Our experiments aim to investigate the role of galectin-3 for successful host infection by the parasite. **Methods:** To analyze the infection by *T. cruzi*, tissue-culture trypomastigotes obtained from Vero cells were used to infect Hela cells transduced with viral vectors for RNAi, which express a shRNA Gal-3 or shRNA scramble. Quantifications of the infections were estimated by Giemsa staining. Another approach involves the investigation of the infection in galetin-3 knock out (gal-3 KO) mice, estimating the levels of blood trypomastigotes by real time PCR. **Results and Conclusions:** For the *in vitro* assay, a considerable reduction in the infection of Hela/gal3 RNAi was observed, compared to Hela/scramble or Hela cells at 24, 48, and 72 hours. For *in vivo* assay, absolute quantification of DNA of blood parasites will be performed by real-time PCR to estimate the parasitemia of infected gal-3 KO mice. Researches on the role of lectins may contribute to a better knowledge of the pathophysiology, and provide new targets for the control of parasitemia in Chagas’ disease.

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