PROTEINS FROM TRYPANOSOMA CRUZI *TRANS*-SIALIDASE FAMILY PLAY DISTINCT ROLES DURING PARASITE INFECTION.

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A dramatic aspect of Chagas disease is the cardiac damage. The study of immunogenic surface molecules might help to understand the nature of the myocarditis. We demonstrated that trafficking of T cells during *T. cruzi* infection can be regulated by a family of sugar binding *trans*-sialidase (TS) proteins. The parasite uses an active TS (aTS) to sialylate its surface glycoproteins and an inactive TS (iTS) acts as a sialic acid-binding lectin. Here we studied the effect of both enzymes on T cells during parasite infection. Balb/c mice were injected with 30 µg of aTS or iTS one hour before infection and at days 2 and 3 post-infection (pi). Parasitemias were evaluated at days 6-10 pi, and the hearts were examined at day 15 pi. In agreement with the increase in parasitemia, we observed an increase in the number of amastigote nests in the cardiac tissue from the aTS treated group, when compared with the control groups. Analyses by flow cytometry, histopathology and immunohistochemistry showed a reduction in the number of leukocytes in the cardiac tissue from infected and iTS treated mice. In agreement, there was a reduction in creatine kinase activity from infected and iTS treated mice. These indicate that, although the enzymes share conserved sugar binding sites, these molecules might play distinct roles during the pathogenesis.Supported by: CNPq,CAPES,FAPERJ