

Trypanosoma cruzi calreticulin: A possible role in Chagas' disease autoimmunity

Ribeiro, Carolina Hager

López, Nandy C.

Ramírez, Galia A.

Valck, Carolina E.

Molina, María Carmen

Aguilar, Lorena

Rodríguez, Margarita

Maldonado, Ismael

Martínez, Ramón

González, Carlos

Troncoso, Rodrigo

Lavandero, Sergio

Gingras, Alexandre R.

Schwaeble,

Trypanosoma cruzi (*T. cruzi*) is the causative agent of Chagas' disease, an endemic and chronic illness that affects 18 million people in Latin America. The mechanisms underlying its pathogenesis are controversial. There is a growing body of evidence supporting the view that *T. cruzi* infection elicits severe autoimmune responses in the host, which significantly contribute to the pathogenesis of Chagas' disease, and several recent studies have reported the presence of autoantibodies and effector T lymphocytes against parasite and self antigens in infected patients and experimentally infected animals. *T. cruzi* calreticulin (TcCRT) is a 45 kDa protein, immunogenic in humans, rabbits and mice. It has a high degree of homology with human (HuCRT) and mouse calreticulin (MoCRT), which would explain why an immune response to TcCRT could contribute to autoimmune reactions in Chagas' disease. Anti-TcCRT antibodies generated in A/J mice immunized with recombinant

TcCRT (rTcCRT) reacted with rHuCRT