Soluble mediators in trypanosoma cruzi infection could trigger mechanisms of pathogenesis in chagas disease [Mediadores solubles liberados por la infección con trypanosoma cruzi podrían desencadenar mecanismos de fisiopatogenia en la enfermedad de chagas experimental]

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Resumen

Trypanosoma cruzi, ethiologic agent of Chagas disease, affects about 2.500.000 people in our country and 18 millon in Latin America. The parasite presents two stages of medical importance in the host, the amastigote, intracellular replicating form and the extracellular trypomastigote, the infective form. That is why the control of infection requires a strong humoral and cellular immune response, hence, it is very important the outcome of host-parasite interaction in the early stages of infection. 30% of infected people, develop some degree of pathology, cardiac or digestive in the chronic period of infection, attribute this to the direct action of the parasite, or to autoimmune reactions induced by Trypanosoma cruzi. The aim of this work was to study at local level, in a murine experimental model some mediators in the early infections with T. cruzi and the adaptive response at the end of the acute phase. The results revealed increased respiratory burst and synthesis of IL-6 since the first hours. Nitric oxide concentration increased with the progression of the infection, while the activation of arginase remained regulated and it was important the specific immunoglobulin production. These mediators would be involved in mechanisms of resistence to T. cruzi, but also in the pathogenia of the infection. These findings stimulate to go further in the knowledge of immunological early events directed to the development to therapeutic approaches in Chagas disease.

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