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BALB/c MICE DEFICIENT IN IL-4 PRODUCTION PRESENT SEVERE CARDITIS AND LOW PARASITISM UPON INFECTION WITH *TRYPANOSOMA CRUZI* COLOMBIAN STRAIN. Silva KN[1], Lima RS[1], Soares MBP[1], Ribeiro dos Santos R[1]. [1]Centro de Pesquisas Gonçalo Moniz, FIOCRUZ-BA.

To investigate the role of Interleukin-4 (IL-4) in the pathogenesis of Chagas' disease, we analyzed the experimental infection of BALB/c IL-4 -/- mice with the Colombian strain of *T.cruzi*. BALB/c IL-4 +/+ mice infected with Colombian strain present high parasitemia in the acute phase, peaking around the 30th day post-infection (dpi), and a mortality rate of 40%. Infected BALB/c IL-4 -/- mice showed significantly lower parasitemia and mortality (5x lower) than wild type mice. Histopathology of hearts from mice in the acute phase of infection revealed the presence of intense inflammatory infiltrate and tissue parasitism in IL-4 +/+. In contrast, hearts of IL-4 -/- mice presented carditis 2-3 fold more intense and lower tissue parasitism than wild-type mice. Although a significant splenic hyperplasia was observed in both groups of mice at 30dpi, spleens of IL-4 +/+ mice were twice as larger as spleens obtained from IL-4 -/- mice. At the end of acute phase, both groups of mice presented a decrease in the intensity of carditis compared to day 30th post-infection, although heart sections of IL-4 -/- still showed a higher inflammatory response than hearts of wild-type mice. More importantly, hearts obtained from IL-4 +/+ mice after 3 months post-infection presented discrete and focal carditis, whereas in IL-4 -/- mice carditis was intense. Parasitemia and tissue parasitism in the heart sections examined were negative in both groups at this timepoint of infection. These results show an important role of IL-4 in the modulation of *T.cruzi*-induced carditis.