Rev. Inst. Med. trop. S. Paulo
43 (6):316, November-December, 2001.

SUMMARY OF THESIS*

BILATE, Angelina Morand Bianchi – **Hamster sírio infectado por *Trypanosoma cruzi* como modelo para cardiomiopatia chagásica crônica: avaliação quantitativa de parâmetros ecocardiográficos, histopatológicos e imunológicos**. São Paulo, 2001. (Dissertação de Mestrado – Instituto de Ciências Biomédicas da Universidade de São Paulo).

Trypanosoma cruzi INFECTED SYRIAN HAMSTER AS A MODEL FOR CHRONIC CHAGASIC CARDIOMYOPATHY: QUANTITATIVE EVALUATION OF ECHOCARDIOGRAPHICAL, HISTOPATHOLOGICAL AND IMMUNOLOGICAL PARAMETRS

An animal model that reproduces the main features of human chronic Chagas´ cardiomyopathy (CCC) could allow the study of its pathogenesis. The aim of this work was characterize the Syrian hamster as a model for human CCC. Female Syrian hamsters were infected with 35,000 (G2) or 100,000 (G3) blood trypomastigotes from *T. cruzi* Y strain. Control animals were injected with saline solution. The ventricular function of each animal was analyzed by echocardiography after 4, 8 and 12 months post-infection (PI). All surviving animals were euthanized after 12 months PI and hearts submitted to histopathological analysis where prevalence and intensity of heart inflammation and *T. cruzi* antigens were analyzed and fibrosis was measured by morphometric analysis; sera were analyzed by ELISA for the presence of antibodies against *T. cruzi* extract, *T. cruzi* B13 protein and porcine cardiac myosin and IgG reactivity against cardiac myosin was also analyzed by Western blot. The proliferative response of splenic cells against cardiac myosin, *T. cruzi* B13 protein and B13- derived peptides was also analyzed. Infected animals displayed high levels of serum antibodies against *T. cruzi* whole extract. Deaths occurred in the acute and chronic phases of infection but late phase deaths were more frequent among G3 than G2. Both infected groups (G2 and G3) developed ventricular dysfunction 12 months PI but it occurred earlier and was more severe in most of animals from G3 than G2. Myocardium from infected animals displayed difuse or muti-focal myocarditis, mild to severe interstitial fibrosis and a virtual absence of parasite antigens. Animals from G3 developed a more severe heart disease 12 months PI as demonstrated by more intense inflammation, higher fibrosis and mortality. The levels of anti-B13 antibodies in G2 and G3 showed statistically significant correlation with interstitial fibrosis. Western blot analysis revealed that serum pool from G1, G2 and G3 all recognized heavy and light chains of cardiac myosin but serum pool displayed differential recognition of cardiac myosin fragments or other cardiac proteins. Splenic cells from infected animals failed to proliferate in response to cardiac myosin, B13 protein as well as B13- derived peptides 12 months PI. The data suggest that this rodent develops a *T. cruzi*- induced cardiomyopathy similar to human CCC. The quantitative analysis of echocardiographical, histopathological and immunological parameters may allow the understanding of pathogenic mechanisms, disease evolution and searching for therapeutic strategies for CCC.

Angelina Morand Bianchi Bilate
ambilate@usp.br

*This thesis is available at the Library of the Instituto de Medicina Tropical de São Paulo