

## SUMMARY OF THESIS\*

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FERNANDES, Margareth – **Interação entre plaquetas de camundongos e *Trypanosoma cruzi***. São Paulo, 1995.  
(Dissertação de Mestrado - Instituto de Ciências Biomédicas da Universidade de São Paulo). 68p.

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Host defense mechanisms against *T. cruzi* infection are complex and many of their aspects have not been well clarified. Platelets seem to be involved in some defense mechanisms of several parasitic diseases, including Chagasic infection.

We have studied some aspects of the influence of platelets on *T. cruzi*.

We confirmed a pronounced thrombocytopenia in BALB/C mice infected with *T. cruzi* strain Y. We investigated the effect of platelet depletion obtained by inoculating rabbit anti platelet IgG during the course of parasitemia in these animals. The number of bloodstream parasites was significantly higher in the treated animals than in those receiving IgG of a non-immunized rabbit. However, cumulative mortality did not differ significantly among these two groups.

A/Sn mice, known to be deficient in the C5 component of complement, were studied. We found that platelet aggregation is not induced in this mouse strain when collagen is employed as an inducer, contrary to what happens in BALB/C mice.

It is known that platelet aggregation in humans is inhibited by anti-aggregating drugs, such as: Salicylic Acid, Indometacin, Ticlid (Ticlofidin Chloridrate), Persantin (Dipiridamol), Persantin S (an association of Dipiridamol and Salicylic Acid). We found that the only

drug with anti-aggregating activity in A/Sn mice was Persantin S.

Platelets from A/Sn mice induced lysis of trypomastigotes "in vitro" in the presence of antibody and complement. However, parasite lysis was significantly lower when platelets from mice treated with Persantin S were employed.

Five minutes after i.v. inoculation of A/Sn mice with trypanosomes obtained from immunosuppressed animals, followed by anti-*T. cruzi* mouse IgG, parasites had been removed from circulation, contrary to what happens in controls. However, when platelet aggregation was inhibited by treating animals with Persantin S, the removal of parasites from circulation after anti *T. cruzi* IgG inoculation, was slowed down.

The supernatant of platelets centrifuged at speeds above those normally used to purify these cells, induced lysis of trypanosomes "in vitro" when antibody and complement were added. Therefore mediators that induce this lysis are easily liberated by platelets, without specific activation.

One of the major component of lysed platelets, of 97Kd, is practically the only fraction visualised in the supernatant of high speed centrifugation submitted to SDS polyacrilamide electrophoresis.

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