

Suramin Effects On *Trypanosoma cruzi* Trypomastigotes

D.F.R Bisaggio, R.C.V. Pinto and T. Souto-Padrón

Departamento de Microbiologia Geral, Instituto de Microbiologia Professor Paulo de Góes, CCS, Bloco I, Universidade Federal do Rio de Janeiro, Ilha do Fundão, 21949-590, Rio de Janeiro, RJ, Brasil – e-mail: souto.padron@micro.ufrj.br

Suramin, a symmetrical polysulfonated naphthylamine derivative of urea, is a potent antagonist to some P₂ receptors (purine and pyrimidine receptors) and is able to inhibit a large number of cellular enzymes, such as ecto-ATPases, retrovirus reverse transcriptase, DNA polymerase, among others. It had initially been used in the treatment of sleeping sickness and cancer chemotherapy. Suramin treatment may cause several morphological changes, like axonal degeneration or axon atrophy, which may lead neurons to apoptotic cell death; alterations in cellular organization and disruption of plasma membrane of chick embryo neural retina and rat sertoli cells. Suramin also caused a dilatation of the rough endoplasmic reticulum of *Trypanosoma rhodesiense*.

We have shown on previous studies that suramin inhibits *T. cruzi* epimastigotes and trypomastigotes Mg²⁺-ecto-ATPase activity. The incubation of both evolutive forms in the presence of the drug inhibits the adhesion and internalization of epimastigotes and trypomastigotes by resident macrophages. It was also observed that suramin inhibits the growth of epimastigotes, and may cause reservosomes swelling if epimastigotes are maintained in the presence of the drug for at least 1 hour. In this study we analyzed the effects of suramin on Y strain trypomastigotes. Parasites were cultivated in LLCMK₂ cells, in RPMI medium containing 2% of fetal calf serum and 500 μM of suramin. The presence of the drug did not affect the duration of *T. cruzi* intracellular cycle, although some of the LLCMK₂ presented a premature disruption, leaving some amastigote and epimastigote forms in the supernatant.

Preliminary results show, by scanning electron microscopy, that trypomastigotes cultivated in the presence of suramin are shorter and broader when compared to control cells, presenting an average size of 11 x 1,4 nm, while control trypomastigotes present an average size of 14 x 1,1 nm. The video microscopy technique showed that some of the suramin treated trypomastigotes present slower movements when compared to control cells. We also observed that some of the obtained trypomastigotes present parts of the flagellum or the whole flagellum detached from cell body.

Supported by: CNPq, Pronex 0885, Faperj, FUJB/UFRJ.

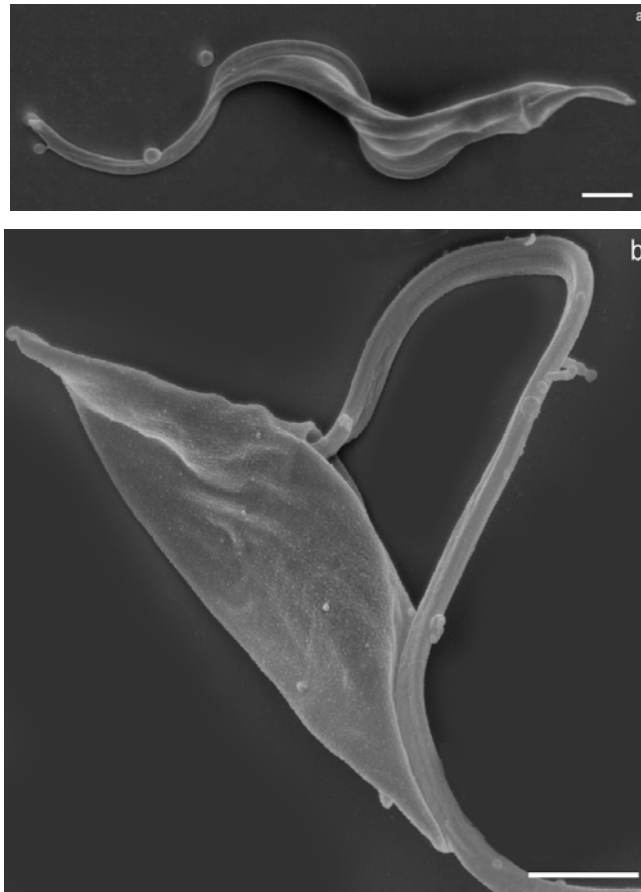


Figure 1 - *T. cruzi* trypomastigotes scanning electron micrograph. (a) control; (b) trypomastigotes cultivated in the presence of 0.5 mM suramin. Part of the flagellum is detached from cell body in drug treated cell. Bars = 1 μ M

Figure 2 - *T. cruzi* trypomastigotes transmission electron microscopy. (a) control; (b) trypomastigote cultivated in the presence of 0.5 mM suramin. In drug treated cell the flagellum is detached from cell body and the round kinetoplast is beside the nucleus. (F) flagellum, (K) kinetoplast, (N) nucleus. Bars = 1 μ M

