

RESEARCH NOTE

Trypanosoma rangeli and *Trypanosoma cruzi*: Cross-reaction among their Immunogenic Components

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It has been demonstrated that immuno-modulating mechanisms induced by epimastigotes of *Trypanosoma rangeli*, an assumed harmless human parasite, may have relevance to protection against *Trypanosoma cruzi*, the causative organism of Chagas' disease (B Basso et al. 1991 *Am J Trop Med Hyg* 44: 413-419). Evidently, this finding is related to the previously demonstrated antigenic similarity, between these parasites (D Afchain et al. 1979 *J Parasitol* 65: 507-514, B Basso et al. 1989 *Rev Lat-amer Microbiol* 31: 141-146, M Grögl, RE Kuhn 1984 *J Parasitol* 70: 822-824, F Guhl, CJ Marinkelle 1982 *Ana Trop Med Parasitol* 76: 361). However, at the present time it is not known which *T. rangeli* antigens can elicit antibodies capable to recognize *T. cruzi* components. In order to clarify this matter, we used the immunoblotting technique to delineate the cross-reactivity among the immunogenic components of these parasites.

The parasites used were isolated from humans in central Panama. Clones derived from single cell isolates (JA Dvorak 1985, *Rev Soc Bras Med Trop* 18 (Suppl): 29-38) of *T. rangeli* (LMCL2) and *T. cruzi* (MA-081A) were characterized by studies of morphology, intracellular multiplication measured *in vitro* and infectivity for vector salivary glands. Flagellates were cultivated and lyophilized following procedures designed previously (NH Vattuone, JF Yanovsky 1971 *Exp Parasitol* 30: 349-355, A Saldaña 1990 *Immunoparasitological Studies of Trypanosoma cruzi clones from Panama*, MSc Thesis, I. Karolinska-Stockholm, 90 pp). The lyophilized *T. rangeli* and *T. cruzi* epimastigotes

were used in the production of mouse antibodies as ascitic fluid (AS Tung et al. 1976 *J Immunol* 116: 676-681, AE Horna 1992 *Biochemical and Immunological Characterization of Trypanosoma rangeli* (Tejera 1920) strains affecting rural populations of Central and South America. MSc Thesis, I. Karolinska-Stockholm, 100 pp). To demonstrate the antibody cross-reactivity we followed procedures described earlier (EC Rostjord et al. 1990 *J Parasitol* 76: 698-702).

Previous reports suggested that exposure to *T. rangeli* antigens might modify the pathology due to *T. cruzi* (Grögl, Kuhn *loc. cit.*, F Guhl et al. 1987 *Parasitol* 94: 475-484, L Hudson et al. 1988 *Parasitol* 96: 449-460). Nevertheless, as far as we know, the first work that demonstrated a partial resistance in *T. rangeli*-immunized mice against *T. cruzi* infection was done by Basso et al. (*loc. cit.*). However, the studies of *T. rangeli* immunogenic components seems to be just beginning.

Our results (Figs 1, 2) revealed that there are several common epitopes among *T. rangeli* antigens and between *T. rangeli* and *T. cruzi* polypeptides. The antibodies eluted from the regions A, B, C, D, E and F recognized additional bands to the bands from which they were separated either in *T. rangeli* or *T. cruzi* immunoblotting profiles. Most of them cross-reacted with antigens in region B (81, 76 and 71 Kda). However, when the antibodies eluted from regions of 34 (G), 29 (H) and 24 Kda (I) were used, more specificity was found. The antibodies from the bands of 34 and 29 Kda recognized, without differences, antigens with similar molecular weights in the *T. rangeli* or *T. cruzi* profiles. This may, therefore, represent the expression of two antigens, highly conserved, which apparently are a second group of immunodominant determinants responsible for the observed cross-reactivity.

An additional finding was the specific recognition pattern noted with the antibodies from the 24 Kda region, apparently this antigen shows a third restrictive group of immunodominant epitopes, expressed either in the 24 Kda polypeptide of *T. rangeli* and in the 23 Kda region of *T. cruzi*.

Even with these cross-reactions, it should be kept in mind, that it is possible to find specific epitopes in each one of these molecules. The identification of specific epitopes recognized by monoclonal antibodies should facilitate studies which aim at settling this possibility.

Finally, research on purification and immunochemistry of these *T. rangeli* antigens, which cross-reacted with *T. cruzi* components, could be important on the diagnostic and management of Chagas' disease.

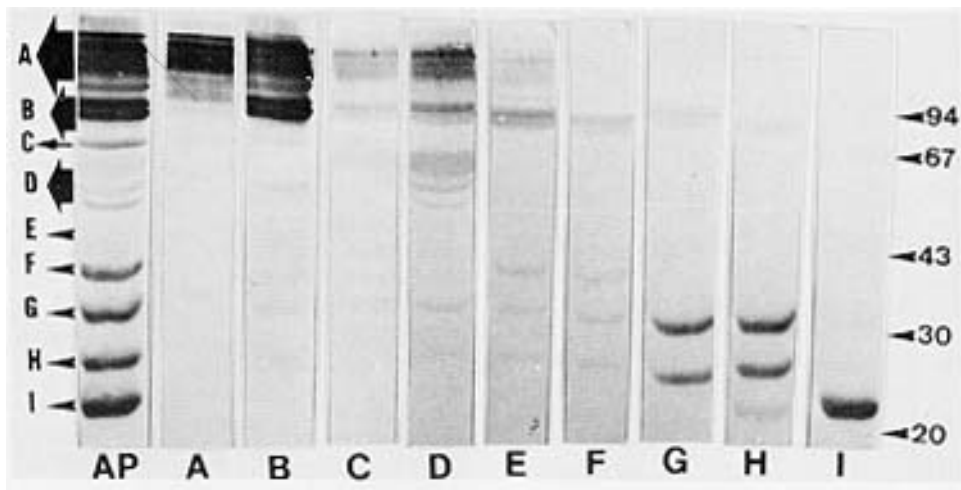


Fig. 1: immunoblot profiles of *Trypanosoma rangeli* antigens. Anti-*T. rangeli* antibodies were separated from selected areas on the *T. rangeli* antigenic profile (strip AP). The strips A-I show the patterns obtained when eluted antibodies interacted with total *T. rangeli* antigens. Right arrows indicate the position of molecular weight markers in Kda.

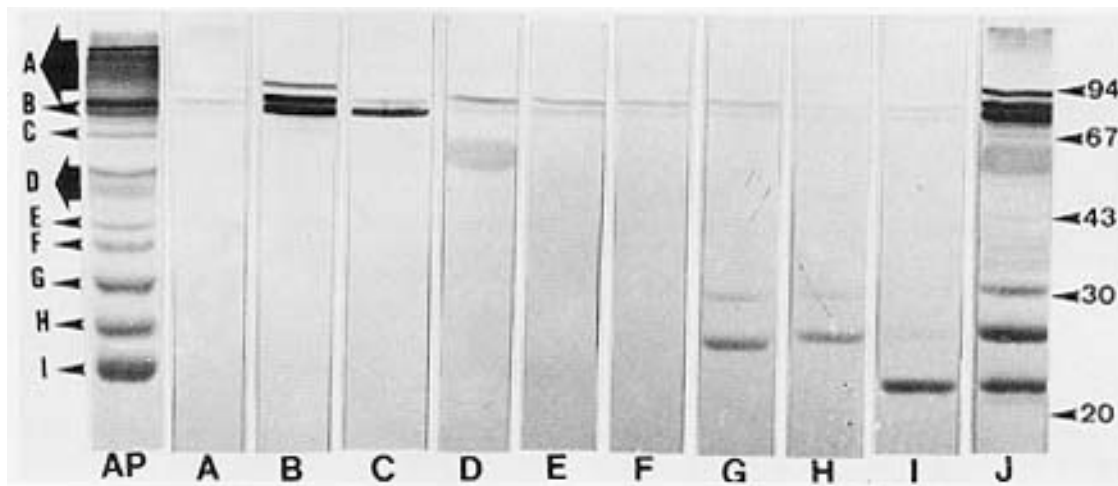


Fig. 2: immunoblot profile of *Trypanosoma cruzi* antigens. Anti-*T. rangeli* antibodies were separated as in Fig. 1. The strips A-I show the patterns obtained when eluted antibodies interacted with total *T. cruzi* antigens. The strip J show the *T. cruzi* antigens recognized by total anti-*T. rangeli* antibodies. Right arrows indicate the position of molecular weight markers in Kda.