IM-85 T.cruzi-SPECIFIC ANTIBODY-SECREETING CELLS IN SUSCEPTIBLE AND RESISTANT MICE DURING THE INFECTION.

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The rate of appearance of T.cruzi-specific antibody-secreeting cells (ASC) was analysed in resistant (C57BL/6) and susceptible (C3H/HeJ) mice infected with T.cruzi (Y strain), using ELISPOT ASSAY (Caulada & Abrahamsohn, JIM 105: 87-95,1987).

Female mice were infected subcutaneously with 5,000 blood forms of the parasite.

Spleen and lymph node cells from infected mice were assayed at various times during the infection on plates coated with tissue culture trypomastigote.

The frequencies of IgM and IgG ASC detected early in the infection (7 days of infection) were very low for both strains of mice. The number of ASC increased as infection progressed during the acute phase. The frequency of IgM ASC in lymph nodes were 2.5-2.9 times higher in B6 than in C3H mice. IgG ASC frequencies were 3.0-4.4 times higher in the resistant strain (B6).In contrast, no significant differences in the frequencies of either IgM or IgG ASC were seen in the spleens of mice from the two strains.

Furthemore, the frequency of specific ASC was always higher in the spleen than in lymph nodes for C3H mice. This pattern was not observed for B6 mice.

These results suggests an important role of lymph nodes in the development of T.cruzi-specific antibody responses.

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IM-86 VH-GENE UTILIZATION IN TRYPANOSOMA CRUZI INFECTION

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The large polyclonal activation of the three major classes of lymphocytes (B, Lyt-2+ and L3T4+ cells) in T.cruzi infection indicate that activation is due to mechanisms other than binding via parasite specific receptor. We have now approached this question by deriving Ig-secreting hybridomas from lymph nodes of acutely infected mice and analysing them for anti-T.cruzi antibody specificity and VH-gene utilization. Furthermore, normal and infected mice at various times after parasite inoculation were compared for VH-gene distribution of CFU-B produced by activated blasts recovered from spleen and lymph nodes, and for relative hybridization of total spleen RNA with each probe of the 9 known VH-gene families.

The results show that over 95% of the recovered hybridomas produce antibodies which do not react with T.cruzi antigens. Yet, while the majority of B cells activated in lymph nodes expresses VH-genes of the J558 family, activated cells in the spleen seem to use other gene families. Large increases of splenic RNA in the various homology families, and the numbers of CFU-B reflect the massive B lymphocyte response. In acute phase, all 9 families are expressed in roughly the same proportions as in normal mice, while in chronic infection, B cells expressing S107 and 7183 VH-genes might be preferentially stimulated. These results provide direct evidences for the parasite non-specific, truly polyclonal, nature ot the T.cruzi induced B cell reponse and are important to the understanding of autoreactivity and immunosuppression associated with infection.

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