S U M M A R Y

In this work we demonstrated the inhibition of lymphocyte proliferation (measured by DNA synthesis) by the immunosuppressive substance (ISS) present in the S. mansoni incubation product and released by the adult worms. The ISS was not species-specific (in rat, mouse and human), resistant to heating, dialyzable and of low molecular weight (500 - 1000) .

Our results could be summarized in the following :

1. The prepared ISS has an inhibitory activity on the lymphocyte proliferation of normal Fischer rats after being injected with it (group B) .

2. Studying the lymphocytic response of infected Fischer rats with S. mansoni (group D) showed clearly that there are two stages of lymphocytic activity;first; enhanced lymphocyte reactivity at day 10 - 14 after infection, and second; characterized by a lowered lymphocyte reactivity during the 5th week (day 35).

3. Injecting Fischer rats with the ISS after infection with S. mansoni (group C) decreased the lymphocyte proliferation at the enhanced stage (day 10 - 14) and

increased the inhibition at the unresponsive stage (day 35), if compared with control infected group D, i.e. the ISS increased the overall suppression or inhibition.

4. Purification of rat spleen cells by platting on plastic surface of Petri dish for 2 hr. then incubated in nylon wool column at the unresponsive stage did not restore the lymphocyte response to con. A or to S.mansoni antigen.

The inhibition in lymphocyte proliferation that occurred during the 5th week either due to the normal S. mansoni infection (group D) or artificially due to the injected ISS (group B & C), does not involve any specific or non-specific suppressor cells. Either inhibitory stimuli were found to act diretly on the responder lymphocytes even after removal of macrophages and T-suppressor cells.

Our work succeeded in throwing some light on the host-parasite relationship in Bilharzial infection. It also evaluated the parameters of the initial stimulation associated with infection of S.mansoni followed by a remarkable depression in lymphocytic response; this might

be due to the ISS released by the adult worms at that stage of infection and which has proved to have a direct effect on the responder lymphocytes and not through the induction of suppressor cells or by modification of macrophages inhibitory factors.

Summing up our experimental results would allow us to consider that S. mansoni could regulate the host immune response by the release of antagonistic factors having a predominantly immunosuppressive effect either directly on responsive lymphocytes or indirectly by modulation of suppressor T cell function. These observations might explain some inconsistant results in passive serum transfer, and also the failure of protective immunity induction after immunization with different crude parasite extracts.