INTRODUCTION

Over the last decade, evidence has accumulated that, following chemotherapeutic cure of Schistosoma mansoni, older individuals develop a resistance to reinfection in comparison to younger children.

This resistance has not been attributed to differences, in exposure alone and has led to the suggestion of the devlopment of an acquired immunity. Certain components-both humoral and cellular-of the immune response to human infection can be measured, but untill recently it has proved difficult to associate resistance in the older age groups with any of these.

However, in vitro studies have suggested a number of potential effector killing mechanisms involving both cells and antibodies, directed primarily at the schistomulum stage of the parasite.

Human intestinal and hepatic schistosomiasis is associated with characteristic alterations of T-cell mediated immune responses. Several mechanisms studied in experimental and human infections have been implicated in the impairment of T-cell responses. They include adherent supressor cells, T-suppressor cells, and serum factors, such as: circulating immune complexes. Soluble immune response suppressor (SIRS); its activated form was released by perioval granulomata of chemically infected mice.

In hepatosplenic schistosomiasis immunological alterations are intrensically linked to the pathogenesis of fibrosis.

In addition, experimental models of infection and vaccination have outlined the importance of CD4 T-cells in the induction of host responses to S.mansoni infection.

CD4 helper subsets in murine models are defined by their cytokine profiles : **T helper 1** producing Gamma interferon (IFN-γ), Interleukin-2 (IL-2) and lymphotoxin and **T helper 2** producing IL-4, IL-5,and IL-10.There is evidence for the induction of similar **T helper** -and-**T helper 2** -like CD4 cells.Both **T helper l** - and **T helper 2**- like cytokines responses have been identified in association with murine models of S. mansoni infection; in which **T helper l** mediators have been linked to protective responses in vaccination studies, whereas **T helper 2** responses, induced following infection and the onset of egg line, are thought to contribute to the onset of pathology.

There is recent evidence for insufficient endogenous release of interleukin- 2 (IL-2) in childen heavily infested by S.mansoni.

Due to the control role of IL-2 in lymphocyte activation, impaired production of IL-2 activity would be expected to entail further alterations, such as inappropiate production of (IFN- γ).