Specific T cell unresponsiveness in human filariasis: diversity in underlying mechanisms.

Sartono E, Kruize YC, Partono F, Kurniawan A, Maizels RM, Yazdanbakhsh M.

Department of Parasitology, Leiden University, The Netherlands.

Abstract

In an attempt to overcome T cell unresponsiveness to filarial antigens, 65 individuals belonging to the three clinical groups of elephantiasis patients, microfilaraemics, and asymptomatic amicrofilaraemics who exhibited unresponsiveness to Brugia malayi adult worm antigen (BmA) were studied. Peripheral blood mononuclear cells were cocultured with antigen and one of the following reagents that have been reported to be effective in reconstituting T cell proliferation: interleukin-2 (IL-2), interleukin-7 (IL-7), anti-interleukin-4, anti-interleukin-10, anti-CD2, anti-CD27, anti-CD28, indomethacin, phorbol myristate acetate (PMA), or calcium ionophore (A23187). We were able to overcome antigen-specific unresponsiveness in only a minority of the individuals studied. Co-culture with IL-2, IL-7, indomethacin and PMA were the only conditions which resulted in enhanced proliferation to BmA in these individuals. In general, unresponsiveness in elephantiasis patients was easier to reverse than in other clinical groups: in 50% of elephantiasis patients, in 12.5% of microfilaraemics and in 20% of asymptomatic amicrofilaraemics. The results indicate that more than one distinct immunological mechanism may account for the antigen-specific unresponsiveness in individuals exposed to and infected with brugian filariasis.

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