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Lymphocytic Filariasis immunology

Paulo Mandaric*

Department of Toxicology, Virtual University, Sau Paulo, Brazil

Editorial

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***For Correspondence:** Paulo Mandaric, Department of Toxicology, Virtual University, Sau Paulo, Brazil;

Email: Mandaric.paulo@gmail.com

EDITORIAL

Filarial parasite immune responses are made up of a complex network of innate and adaptive cells that interact with the parasite to cause a variety of clinical symptoms. An antigen-specific Th_2 response and an increase of IL-10-producing $CD4^+$T cells, followed by a muted Th_1 response, are the most prominent immunological features of lymphatic filariasis.

This antigen-specific T-cell hypo responsiveness appears to be critical for the long-term maintenance of infections with high parasite density. While the correlates of protective immunity to lymphatic filariasis are still unknown, owing to a lack of suitable animal models to examine susceptibility, it is obvious that T cells and, to a lesser extent, B cells are required for protection. Pathological symptoms of LF, including as lymphedema, hydrocele, and elephantiasis, are definitely mediated by host immunological responses, particularly $CD4^+$T-cell responses.

The failure to establish T-cell hypo responsiveness in the face of antigenic stimulation appears to be the major underlying fault in the development of clinical disease. Finally, filarial infections have a proclivity for inducing bystander effects on a range of immune responses, including responses to vaccines, allergens, and other infectious agents. The complexity of the immune response to filarial infection opens the door to a better knowledge of how immune responses to chronic infections are regulated in general.

When cell proliferation and secretion of interleukin-2 and interferon are measured in patients with lymphatic filariasis, microfilaremia is related with parasite antigen-specific hypo responsiveness. Hypo responsiveness in these people is not just parasite antigen specific, but also appears to be restricted to Th_1 responses. Th_2-mediated responses to parasite antigens, such as IL-5 secretion and IgE antibody generation, are typically substantial and similar to those reported in immunologically more reactive amicrofilaremic patients with chronic lymphatic disease.

Although the methods by which Th_1 responses are suppressed are unknown, several studies show that down-regulatory cytokines like as IL-10 may play a role. Microfilaremic individual's mononuclear cells have been reported to secrete more IL-10 both spontaneously and in response to parasite antigens.

Determining immune responses in lymphatic filariasis has been hampered by new approaches (in particular, circulating filarial antigen identification) for characterizing and categorizing filarial-infected patients, as well as fast developing understanding of new immunological mediators and efforts.

We attempted to investigate the influence of patency on antigen-driven proliferative and cytokine responses seen in peripheral blood mononuclear cells of individuals with varying clinical manifestations of lymphatic filarial infection using assays for circulating antigen in sera collected as part of the many immunological studies performed on individuals in a Wuchereria bancrofti-endemic region of South India.

In mosquito-transmitted infective larvae (L_3), some of which grow into adult worms and create micro filarial (mf) transmission stages, residents in lymphatic filariasis-endemic areas are constantly infected. As molecular vaccines against filarial parasites are being considered 1,2, the topic of whether naturally acquired resistance occurs in adult residents of endemicareashas recently become of attention. To establish acquired resistance to Filariasis in human populations, two epidemiological techniques have been used.

Brugia infection in mice has yielded a wealth of quantitative and qualitative data on the immune response generated by filarial worms at various phases of their life cycle. The immune response in the mouse and the infected human share many similarities, and in this review, we focus on areas of current interest, such as the generation of particular cytokine responses and their function in immunomodulation and protective immunity.

Lymphatic filariasis causes a variety of symptoms, ranging from microfilariaemia to severe immunopathology. Heterogeneity in genetically driven host responses has been blamed for the disease's geographical variances. Don Bundy, Bryan Grenfell, and P.K. Rajagopalan can provide a straightforward, cohesive explanation for the observed heterogeneity by modelling the disease across time.

The pathophysiology of hydrocele and elephantiasis, two primary clinical symptoms of bancroftian filariasis, is assumed to be similar. In Leogane, Haiti, the following characteristics of 121 patients with hydrocele or elephantiasis were compared: Microfilaria was found in 39 percent of 57 males with hydrocele and 3 percent of 64 people with leg lymphedema (P.001). In 15 (43%) microfilaria-negative men with hydrocele and 9 (15%) microfilaria-negative people with leg edoema, circulating filarial antigen, probably from the adult worm, was identified (P=.004).

Microfilaria-positive males exhibited lower levels of filaria-specific IgG1 and hydroceles that were significantly smaller in size and lasted significantly less time than microfilaria-negative men; hydrocele volume was inversely related to microfilarial density (P=.001). Filaria-specific IgG4 and reduced lymphocyte proliferation were linked to filarial antigen but not microfilariae. The severity of leg edoema was not linked to antigen status. Men with hydrocele are more immunologically and parasitological diverse than elephantiasis patients in this filariasis-endemic location.