ImmunoTools *special* Award 2014

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LTh17 role in modulating the immune response to infection with VIH-1 virus

The $CD4^+$ lymphocytes called T-helper (h-TL) or cooperators, mediate effector immune response, play a central role in immune protection and acquire different functional properties in response to signals generated by the innate immune system (1). They are able to stimulate B lymphocyte (BL) to secrete immunoglobulins, induces activation of microbicidal system macrophages, polymorphonuclear recruited at sites of infection and / or inflammation (1,2).Furthermore, secrete cytokines and chemokines that orchestrate the immune response effective in cooperation with the $CD8^+$-T lymphocytes (1).

The h-TL represent a heterogeneous population of cells that play an important role in adaptive immunity. Naive h-TL (n-hTL), can differentiate into memory cells or effector, according to the pattern of trigger signals received during the initial interaction with the antigen and the cytokine microenvironment in a heterogeneous population of effector cells: central memory MC-hTL and effector memory ME-hTL (1-3).

The n-hTL generate five subtypes of effector lymphocytes: Th1, Th2, Th17, and regulatory cells Th1/Th17 LT regulatory (T-reg). These subsets are distinguished by their function, the expression of surface antigens, transcription factors, and the profile of cytokines they produce (1-3). The Th1 fulfill their primary role in protection against intracellular microorganisms and organ specific autoimmune diseases; Th2 in protection against parasites in the gastrointestinal mucosa, asthma and allergic diseases; Th17 play an important role in maintaining the sterility of the intestinal mucosa in the extracellular bacteria and fungi infection in chronic inflammation in autoimmune diseases and T-reg cells regulate antigen-presenting-specific, anti-inflammatory activity and maintain tolerance of the immune system (3-5).

Infection with human immunodeficiency virus (HIV-1) is characterized by a gradual and progressive impairment of immune function, with the final result the Acquired Immune Deficiency Syndrome (AIDS) (6). Infection of HIV-1 involves interaction

between the viral envelope glycoprotein with the CD4 molecule gp120/41 located on the surface of host cells, primary target of the virus expressed the $CD4^+$-TL (7,8).

The VIH-1 pathogenesis is extremely complex, immune dysregulation affecting innate immunity, humoral adaptive and (6). The gastrointestinal tract is a key site for the HIV-1 infection, there are 40% of all lymphocytes and about 60-80% of the hTL memory cells (8,9).

The levels of the different Th subsets in HIV-1 infection remain controversial. It has been demonstrated that a polarization of Th1/Th2 response during the disease progression to AIDS (10,11). The gradual decline in peripheral blood $CD4^+$-LT trasladación induces in the lumen of microorganisms that opportunistic infections, increased proinflammatory cytokines and of viral replication that characterize the progression of the disease (8,9).

We studied the $CD4^+$-TL subpopulations in these patients HIV-1/SIDA. We found that the levels of Th1 and Th17 in naive patients are decreased since the early stages of infection, whereas Th2 maintain their levels during the course of the same. (The results were presented at the Annual Meeting of the Society of Immunology Argentina, in the Province of Córdoba Cocos, from 7 to 9 November 2013).

Our goal is to evaluate qualitatively and quantitatively the Th17 lymphocytes in relation to subtypes h-TL naive patients infected HIV-1/AIDS and correlate with clinical patient. Also determine the levels of cells: n-hTL, MC-hTL, ME-hTL, BL, cells of the innate immune system and correlate with levels of Th17.

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