SCHISTOSOMA SPP ANTIGENS INDUCE T CELL REGULATORY PROFILE IN HTLV-1-INFECTED INDIVIDUALS

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ABSTRACT

Introducton: The HTLV-1 is the causal agent of HTLV-1-associated Myelopathy/Tropical Spastic Paraparesis (HAM/TSP). The immune response in HTLV-1 infection is polarized to the Th1-type, and previous data from our group have demonstrated that the use of Schistosoma antigens to cell cultures in HTLV-1 infection resulted in down-modulation of Th1/inflammatory response. Objective: To evaluate whether Schistosoma spp antigens are able to induce *in vitro* the expression of modulatory molecules in TCD4⁺ and TCD8⁺ cells (CD25, CTLA-4 and Foxp3) in HTLV-1-infected individuals, Methods: The Schistosoma antigens Sm29, ShTSP2 and PIII were added to PBMC cultures and the frequency of TCD4⁺ and TCD8⁺ cells expressing CD25, CTLA-4 and Foxp3 were measured using flow cytometry. **Results:** The frequency of TCD4⁺CD25^{High} cells increased by the presence of *Schistosoma* antigens (without stimulation = $0.6 \pm 2\%$, stimulated with Sm29 = 1.2 ± 1 , ShTsp2 = $0.9 \pm$ 0.5 and PIII = 0.9 ± 0.6%, respectively). The frequency of TCD4⁺CD25^{High}Foxp3⁺ cell and TCD8⁺CD25^{High}Foxp3⁺ cells also increased after the addition of the *Schistosoma* antigens. Moreover, the frequency of TCD4⁺CD25^{High}CTLA-4⁺ was significantly higher in cultures stimulated with Sm29 (without stimulation = $21 \pm 10\%$, stimulated with Sm29 = $25 \pm 12\%$; p<0.05) and the frequency of TCD8⁺CD25^{High} also increased by the presence of Sm29 and ShTsp2 to the cultures (without stimulation = $0.5 \pm 0.3\%$, stimulated with Sm29 = $0.7 \pm 0.4\%$, stimulated with ShTsp2 = $0.8 \pm 0.5\%$; p<0.05). Conclusion: The Schistosoma spp antigens used in this study induced a regulatory profile in the TCD4⁺ and TCD8⁺ cells from HTLV-1infected individuals. It might act as an anti-inflammatory factor that could down-modulate the strong immune response induced by HTLV-1. Financial support- CNPQ (Universal 479417/2008 3); NIH (R01AI079238A).

Palavras-Chave: Schistosoma antigens; HTLV-1; Immunomodulation; T regs cells.