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POSTER PRESENTATION

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Loss of IL-10 secretion by regulatory B lymphocytes in multiple sclerosis patients

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Background

Recently, an alternative role of B cells has emerged. Regulatory B cells have been shown to down-regulate immune responses in mice and humans. These cells control disease progression in several models of human autoimmune diseases, including a model of Multiple Sclerosis (MS). Their regulatory function mostly relies on their capacity to produce IL-10.

Objectives

Our aim was to explore the frequency, the phenotype and the functional properties of regulatory B cells in MS patients compared to Healthy Volunteers (HV).

Methods

All patients suffered from MS and had not received immune drugs since at least 6 months. The frequency and the phenotype of B cell subsets have been analysed using different specific markers: CD19⁺CD27⁺ memory B cells, CD19⁺CD38^{dim}CD24^{dim} mature naïve B cells and CD19⁺CD24^{high}CD38^{high} transitional B cells. Their capacity to secrete IL-10 was analysed *in vitro* 48 hours after stimulation by CD40 ligand and CpG ODN.

Results

Thirty-eight MS patients (mean age: 41.55±12.8 yrs) and 21 HV (mean age: 35.4±13.4 yrs, NS) have been included. The patients had different MS forms: Clinically Isolated Syndrome (n=5), Relapsing-Remitting (n=23), Secondary Progressive (n=4) and Primary Progressive (n=6).

No significant difference was found for the frequency of the different B cell subsets in the different subgroups

of patients. Particularly, MS patients harbor the same number (in frequency and absolute value) of CD19⁺CD24^{high}CD38^{high} transitional B cells. However, the frequency of IL-10 secreting B cells was significantly decreased in MS patients (0.9±0.5%, n=9) compared to HV (2±0.95%, n=10, p<0.05, Mann Whitney test).

Discussion/perspectives

MS patients display the same frequency of CD19⁺CD24^{high}CD38^{high} transitional B cells, described as having regulatory properties. Nevertheless, B cells of MS patients present a significant decreased secretion of IL-10, supporting a defect in the B cells regulatory property. If confirmed, these results could have a considerable impact for the development of new therapeutic strategies.

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