CIRCULATING ANGIOGENIC T-CELLS ARE REDUCED IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS WITH HIGH DISEASE ACTIVITY AND WITHOUT KNOWN CARDIOVASCULAR RISK FACTORS

1S Piantoni, 1I Cavazzana, 2M Fredi, 2M Taraborelli, 3F Franceschini, 4A Tincani, 1P Airo.
1Rheumatology and Clinical Immunology, Spedali Civili and University of Brescia, Italy; 2Internal Medicine Unit, Mellino Mellini Hospital, Chiari (Brescia), Chiari, Italy

Background Recent evidences underlined the central role of T-cells in the pathogenesis of Systemic Lupus Erythematosus (SLE) and in its cardiovascular complications.1 CD3 +CD31 +CXCR4+ angiogenic T-cells (Tang) have been identified as a T-cell subtype involved in the repair of damaged endothelium cooperating with endothelial progenitor cells.2 Tang were described as selectively expanded in the circulation of systemic sclerosis patients displaying peripheral vascular complications, as a reaction to an inefficient angiogenesis.3 Not much information is available on Tang in a SLE patients: in a recent study the percentage of circulating CD8 +Tang, but not CD4 +Tang, was higher in SLE than in healthy controls.4 Not much information is available on Tang in a SLE patients: in a recent study the percentage of circulating CD8 +Tang, but not healthy controls.4

Results Peripheral B cells from SLE patients in remission or control subjects did not show any difference in IgG, IgM, and anti-dsDNA-aabs to all aforementioned stimuli. The addition of CpG and SAC to cell cultures showed a stimulatory effect on immunoglobulin, cytokine and anti-dsDNA-aab production in SLE B cells and healthy controls alike. The amount of anti-dsDNA IgG-type autoantibodies produced by peripheral B cells was negligible. However, B cells from SLE patients showed diminished capacity to produce IL-10 as compared to B cells from healthy donors (SLE Estimate =40.17, Std.error 17.21, p<0.01).

Conclusions B cells from SLE patients in remission as compared to peripheral B cells from healthy donors have comparable capacity to secrete immunoglobulin including non-IgG anti-dsDNA-aabs whereas their capacity to secrete IL-10 is impaired. This suggests a persisting intrinsic defect of B regulatory cells in SLE.

REFERENCES