Heine University hospital in Düsseldorf and from 5 healthy controls (HC). All patients were randomly collected in clinical remission state (SLEDAI 1,1±1,8). The medication consisted of prednisolone (5/13 patients), mycophenolate mofetil (3/13 patients), azathioprine (1/13 patients), hydroxychloroquine (8/13 patients) or was without immunosuppression.

After 6 days of cell culture levels of IgG, IgM, dsDNA antibodies and interleukin-10 (IL-10) were determined in the supernatants by ELISA. The effect of the stimuli alone or in combination on IgG, IgM, anti-dsDNA-aab, and IL-10 production was analysed.

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, $p<0.01$).

Conclusion 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.

PS5:95 CIRCULATING ANGIOGENIC T-CELLS ARE REDUCED IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS WITH HIGH DISEASE ACTIVITY AND WITHOUT KNOWN CARDIOVASCULAR RISK FACTORS

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

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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] However, in this study SLE patients with hypertension, dyslipidemia or smoking habit, factors which may influence Tang counts, were not excluded.

The aim of this study was to characterise Tang in a cohort of patients with SLE without known cardiovascular risk factors.

Methods Twenty female SLE patients with a recent disease onset (<5 years) and without traditional cardiovascular risk factors or previous events (age: median value=43 [25th-75th percentile=27–54] years) and 18 healthy controls (age: 40 [32–54] years) were enrolled.

Phenotypic analysis of peripheral Tang lymphocytes was made by flow-cytometry.

Disease activity was evaluated by SLEDAI-2K score.

Results SLE patients were divided in two groups according with disease activity. Patients with SLEDAI-2K equal or higher than 6 were defined as patients with high disease activity (n:5). They had a lower percentage of circulating Tang in comparison with healthy controls (10 [8–15] vs 16 [14–23]% of CD3 +T cells, $p=0.04$). The result was confirmed in absolute number (83 [60–103] vs 242 [165–328] cell/microliter, $p=0.04$). SLE patients with low disease activity had levels of Tang which were intermediate between, and not significantly different from, healthy controls and patients with high disease activity.

Conclusions Tang were reduced in our patients with active SLE, and no known cardiovascular risk factors, suggesting that this reduction was directly explained by disease activity.

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PS5:96 IL-21 DEPENDENT GRANZYME B PRODUCTION OF B-CELLS IS DECREASED IN PATIENTS WITH LUPUS NEPHRITIS

[1]M Rabani, [1]B Wilde, [1]K Hübbers, [1]A Kribben, [2]O Witzke, [2]S Dolff. *[1]Department of Nephrology, University Hospital Essen, Germany; [2]Department of Infectious Diseases, University Hospital Essen, Germany*

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Objectives B-cells play a crucial role in the pathogenesis of lupus nephritis. Recently, a separate subset has been discovered characterised by expression of Granzyme B. The aim of this study is to investigate this subset in patients with systemic lupus erythematosus especially in patients with lupus nephritis.

Methods Isolated peripheral blood mononuclear cells of patients with systemic lupus erythematosus (n=30) and healthy controls (n=21) were *in vitro* stimulated with CPG, IgG +IgM and IL-21. Patients were sub-grouped in patients with and without biopsy proven lupus nephritis. CD19 +B cells were analysed for intracellular Granzyme B expression by flow cytometry. Patients disease activity was assessed by systemic lupus erythematosus disease activity index (SLEDAI).

Results The strongest stimulus for Granzyme B secretion of CD19 +B cells was IgG +IgM in presence of IL-21. Patients with systemic lupus erythematosus had a significant decreased percentage of Granzyme B+CD19+B cells. This could be shown in particular for patients with active disease and with lupus nephritis.

Conclusions These data demonstrate that CD19 +B cells of patients with systemic lupus erythematosus are impaired to produce Granzyme B. This may contribute to an imbalanced B-cell regulation towards effector B-cells which might promote the development of lupus nephritis.