Results CD4+T cell proliferation was significantly less inhibited in cocultures with SLE-B25+ compared to cocultures with ND-B25+ (mean 51% vs 35% of proliferation of T cells cultured alone (100%), p<0.01). In cross-over-experiments ND-T cell proliferation decreased below 50% in 22 of 37 cases cocultured with SLE-B25+ but in 34 of 37 cases cocultured with ND-B25+. This effect was independent from T cell origin: SLE-T cell proliferation was similarly reduced below 50% in only 21 of 37 cases with SLE-B cells but in 32 of 37 cases with ND-B cells. Of interest, B25+ cells from patients with WG (n=37) exhibited strong inhibitory effects similar to their normal counterparts. So far, no differences in B cell activation markers, cytokine production or viability were found between ND-, WG- and SLE- B25+-cells. In addition no hints for a correlation between SLE-disease activity, treatment and Breg suppressor-function could be stated.

Conclusions B25+ from SLE patients exhibit reduced regulatory capacity towards CD4+T cells in contrast to B25+ from healthy donors or patients with WG, suggesting that suppressive defects of SLE-B cells might be rather disease specific and less representative of autoimmunity in general or chronic inflammation. Future experiments deal with the SLE-B cell specificities interfering with suppressive function and the investigation for parameters to restore it.

8 DECREASED IMMUNOREGULATORY ACTIVITY OF B CELLS DERIVED FROM PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and objectives Our group has recently generated human B cells with immunoregulatory properties in vitro from peripheral blood (PB) of healthy donors. Upon prestimulation via their B cell receptor (BCR) large, activated CD25+B cells (B25+), but not resting CD25−B-cells (B25−), induced temporary CD4+T cell energy and apoptosis. These results led us to rethink the so far pathogenic role of B cells in autoimmune disease.

Our aim was to test the immunoregulatory capabilities of B-cells from patients with systemic lupus erythematosus (SLE), as an autoimmune disease with characteristic B cell involvement. Since Treg defects are reported, at least in advanced stages of disease, it could be suspected that Breg might be affected, as well.

Materials and methods Highly purified CD19+B cells and CD4+Th-cells were separated from PBMC by magnetic cell sorting. B cells were prestimulated with SAC (*Staphylococcus aureus* Cowan I Antigen) for 3d and sorted into highly activated $FSC^{hi}CD25+$ (B25+) and small resting $FSC^{lo}CD25-$ (B25-) B cells by cytometric cell sorting. Upon 4d coculture with Th-cells and αCD3+IL-2, T cell proliferation was determined by 3H-TdR incorporation. Experiments were set up in parallel with B cells from healthy donors (ND) and patients with SLE. For better comparison the study additionally included B cells from patients with another autoimmune disease, Wegener's Granulomatosis (WG).