ANTI-C1Q AUTOANTIBODIES AS A TARGET FOR TREATMENT WITH SCFV ANTIBODY IN MRL/LPR MOUSE MODEL OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Aim: Systemic lupus erythematosus (SLE) is a chronic inflammatory autoimmune disease characterized by tissue damage in multiple organs caused by autoantibodies and the resulting immune complexes. One possible way for complement system contribution to onset of autoimmune disorder could be realized by the impairment of C1q mediated apoptotic clearance as part of human homeostasis. The capacity of C1q to bind early apoptotic cells could be decreased or even lost in the presence of anti-C1q antibodies which are specific for epitopes within gC1q.

Material and Methods: A phage-displayed library expressing single-chain recombinant antibodies was screened to select scFv specific for anti-C1q autoantibodies from different groups of lupus sera.

Two groups of MRL/lpr mice were used for *ex vivo* and *in vivo* experiments.: 7 weeks old mice that are still disease free and 16 weeks old with advanced disease manifestations. We have injected the mice with 20 μg/mouse weekly of the studied scFv antibody. Control groups were injected with PBS only. Blood samples were collected weekly and the sera were stored at -80 ºC for subsequent analyses.

Results: The data show that the scFv treatment modulates the percent of B and T cell subpopulations and splenocyte apoptosis. An increase of the proteinuria levels in the 7 weeks old MRL/lpr mice, splenocyte proliferation change and the number of plasmocytes producing anti-dsDNA antibodies in the treated group were observed also.

Conclusion: The treatment with anti-idiotypic scFv antibody has modulatory effect on lupus symptoms in MRL/lpr murine model of SLE.