the following autoantigens: RNP/Sm, Sm, SSA/Ro60, SSA/Ro-52, SSB, Scl-70, PM-Scl, Jo-1, CENP-B, PCNA, dsDNA, nucleosomes, histones, ribosomal P antigen, AMA-M2.

Results 16 SLE patients with multiple autoantibodies were investigated. SSA-antibodies were found both in sera and CIC from 12 patients. 11 out of the 12 SSA positive patients had Ro52 antibodies that showed reactivity in both serum and CIC. SSB antibodies were present in 10 sera out of which 7 also had detectable levels in CIC. RNP antibodies were detected both in sera and CIC from four out of seven patients and Sm in one. Nine patients had nucleosome and 10 histone antibodies in sera. Out of these, low amounts of nucleosome and histone antibodies were found in CIC from one patient. None out of six patients that were positive for dsDNA antibodies in sera had reactivity against dsDNA in their isolated CIC. The autoantibody specificities found were comparable in CIC isolated using both methods.

Conclusion Our results confirm our initial hypothesis that SSA and SSB antibodies are important in the formation of CIC in SLE, in contrast to anti-dsDNA which instead might form IC in situ in tissues. This implies an active role for anti-SSA/SSB antibodies in the ongoing inflammatory process, a hypothesis that the authors will investigate further. The anti-spliceosomal anti-RNP and anti-Sm antibodies might also participate in CIC formation, but the number of hitherto investigated sera is small. The authors will now continue with investigation of paired sera taken from individual SLE patients during flares and inactive disease, to test the hypothesis that some specific autoantibodies might accumulate in CIC during active SLE.

A14 SSA AND SSB ANTIBODIES ARE IMPORTANT IN THE FORMATION OF CIRCULATING IMMUNE COMPLEXES IN SLE

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Background The authors have earlier demonstrated that the amounts of circulating immune complexes (CIC) are higher in anti-SSA positive as compared to anti-SSA negative systemic lupus erythematosus (SLE) patients. As the same discrepancy was not found for anti-DNA, the authors hypothesised that anti-SSA but not anti-DNA might form CIC during active SLE. Here the authors have used two techniques to study the autoantibody content in CIC.

Methods Immune complex were either isolated from SLE sera by PEG-precipitation or bound to C1q-coated wells and thereafter eluted. Comparison was then done between autoantibody content in serum and in CIC respectively using a commercial line blot assay (Euroline ANA profile 3) containing