

(Corgenix). In addition, disease activity (DAS28 or BASDAI), CRP, IgM rheumatoid factor, anti-CCP, and lipid levels (total cholesterol, TC; LDL-C, HDL-C and triglyceride) were also assessed. Assessments were performed at baseline, as well as 6 and 12 months after treatment initiation.

**Results** Anti-TNF treatment was highly effective in both diseases, as the mean DAS28 decreased from 6.32 to 3.16 ( $p = 0.02$ ) in RA, mean BASDAI decreased from 5.87 to 1.84 ( $p < 0.001$ ) in AS. In RA, AS and the mixed arthritis population ( $n = 43$ ) baseline oxLDL/beta2gpl levels were  $0.235 \pm 0.1$ ,  $0.245 \pm 0.1$  and  $0.238 \pm 0.1$ , respectively. There were no significant differences between RA and AS patients. In RA, ETN/CZP treatment resulted in non-significant decreases in complex levels after 6 months ( $0.214 \pm 0.1$ ) and 12 months ( $0.206 \pm 0.1$ ). In AS, oxLDL/beta2gpl complex levels did not change after 6 months of ETN therapy, but significantly decreased after one year ( $0.195 \pm 0.1$ ;  $p = 0.01$ ). In the RA+AS population, anti-TNF treatment significantly decreased oxLDL/beta2gpl levels after 12 months ( $0.203 \pm 0.1$ ,  $p = 0.02$ ). In addition, baseline oxLDL/beta2gpl complex levels positively correlated with TC (RA:  $r = 0.563$ ,  $p = 0.002$ ; AS:  $r = 0.542$ ,  $p = 0.049$ ; RA+AS:  $r = 0.532$ ,  $p < 0.001$ ), and LDL-C (RA:  $r = 0.630$ ,  $p < 0.001$ ; AS:  $r = 0.756$ ,  $p = 0.004$ ; RA+AS:  $r = 0.648$ ,  $p < 0.001$ ) in both diseases. Circulating oxLDL/beta2gpl levels did not correlate with DAS28, BASDAI or CRP.

**Conclusions** In a mixed cohort of RA and AS patients, anti-TNF therapy suppressed the circulating levels of oxLDL/beta2gpl complexes, markers of atherosclerosis and vascular disease in SLE or APS. Moreover, oxLDL/beta2gpl levels correlated with TC and LDL-C in arthritides. oxLDL/beta2gpl complexes do not seem to be markers of disease activity in RA or AS.

#### A6.14 SEMAPHORIN 3A, AN IMMUNOREGULATOR AND POTENTIAL BIOMARKER FOR DISEASE SEVERITY IN SYSTEMIC SCLEROSIS

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**Introduction** Semaphorin 3A (sema3A) plays a regulatory role in immune responses, mainly affecting the activation of regulatory T cells. It has been found to correlate with disease activity in rheumatoid arthritis and systemic lupus erythematosus (SLE).

**Aim** To investigate the expression of sema3A in patients with systemic sclerosis (SSc) compared to healthy controls and SLE disease controls and to correlate its expression with clinical characteristics.

**Methods** 27 SSc patients, 42 SLE patients and 18 healthy controls were enrolled. Serum level of sema3A was measured by ELISA and expression of sema3A on regulatory T cells was evaluated by FACS analysis. SSc patients were evaluated for demographics, clinical manifestations, routine laboratory results, nailfold videocapillaroscopy patterns, pulmonary function tests, echocardiograms, modified Rodnan skin score (mRSS) and disease activity and severity scores.

**Results** Serum levels of semaphorin 3A were lower in SSc compared to healthy controls  $14.38 \pm 5.7$  ng/ml vs.  $27.14 \pm 8.4$  ng/ml,  $p < 0.0001$  and similar to SLE  $15.7 \pm 4.3$  ng/ml. The expression of semaphorin 3A on regulatory T cells was also lower in SSc compared to healthy controls  $61.7 \pm 15.7\%$  vs.  $88.7 \pm 3.7\%$  ( $p < 0.0001$ ). Semaphorin 3A serum level

inversely correlated with the duration of disease;  $r = -0.4$ ,  $p = 0.036$  and with low C4 level  $r = 0.66$   $p = 0.026$ . SCL-70 antibody positivity was associated with a lower semaphorin 3A level in serum (difference in mean of 3.44  $p = 0.06$ ).

**Conclusion** Sema3A expression is low in SSc serum and more specifically on regulatory T cells. This may help explain the reduced activation of regulatory T cells in SSc, contributes to our understanding of the pathogenesis of the disease and may serve as a future target for treatment.

#### A6.15 GENETIC SIGNATURES MAY BE ASSOCIATED WITH VASCULAR PATHOLOGY IN RHEUMATOID ARTHRITIS

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**Background and objectives** Accelerated atherosclerosis, increased cardiovascular (CV) morbidity and mortality have been associated with rheumatoid arthritis (RA). In single SNP studies, *CD40*, *HLA-DQB1*, *MTHFR*, *SMAD3* and possibly other alleles have been associated with cardiovascular disease (CVD) or vascular pathophysiology in RA. Endothelial dysfunction, carotid atherosclerosis and arterial stiffness that may predict the development of CVD are assessed by brachial artery flow-mediated vasodilation (FMD), common carotid intima-media thickness (ccIMT) and arterial pulse-wave velocity (PWV), respectively. In this study, we wished to determine expression profiles of multiple genes that may differentiate between physiological and pathological vascular function in RA.

**Patients and methods** Altogether 16 RA patients were recruited. FMD, ccIMT and PWV were assessed in all patients using standard B-mode ultrasound techniques. FMD  $< 6\%$ , ccIMT  $> 0.6$  mm and PWV  $> 9$  m/sec were considered abnormal. Peripheral blood mononuclear cell samples were obtained and used in microarray. The signature of those genes were determined by principal component analysis (PCA) and hierarchic clustering (GeneSpring software), which significantly differentiated patient subsets with normal vs. abnormal FMD, ccIMT and PWV.

**Results** Among RA patients, 11 had low (impaired) and 5 had normal FMD, 11 had high (increased) and 5 had normal ccIMT and 9 had high (increased) and 7 had normal PWV. Altogether 20 genes differentiated patients with low vs. normal FMD. Altogether 33 genes separated high vs. normal PWV. Finally, 240 genes differentiated increased vs. normal ccIMT.

**Conclusions** Using microarray, genetic signatures may differentiate RA patients with and without vascular pathology.

#### A6.16 DECTIN-2 IS ESSENTIALLY INVOLVED IN INNATE IMMUNE RESPONSE OF EXPERIMENTAL ARTHRITIS

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## A6.15 Genetic signatures may be associated with vascular pathology in rheumatoid arthritis

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