Conclusions: Cell surface markers of activation and adhesion increased in SLE monocyte subsets compared to HC. In parallel, increased inflammatory cytokines and chemokines that attract monocytes to tissues were increased in the serum of these patients. Linking a possible source of this increase in serum analytes, HC monocyte subsets were stimulated with disease-relevant ligands and evaluated in culture. Along with increased monocyte expression of CX3Crl, preliminary data demonstrates an increase in CX3CL1 expression on endothelial progenitor cells (EPCs) and immature and mature circulating endothelial cells (iCECs and mCECs respectively) in active disease.

Disclosure of Interest: None declared

**FR10292**

**DYSFUNCTION OF TFH, TREG AND TR1 CELLS IN APOE-/- FASLG LDL C57BL/6 MICE WITH LUPUS SYMPTOMS AND ATHEROSCLEROSIS**

S. Wang, G. Yao, L. Sun, Department of Rheumatology and Immunology, The Affiliated Drum Tower Hospital of Nanjing University Medical School, Nanjing, China

**Background:** Cardiovascular disease due to atherosclerosis is currently recognised as one of the leading causes of death among patients with systemic lupus erythematosus (SLE). It is well established that dysfunction of lymphocytes contribute to the pathogenesis of SLE. Recent studies also showed infiltration of several subsets of lymphocytes in atherosclerotic lesions and their various contributions to atherosclerosis were uncovered in experimental models and patients. However, the predominant and specific subsets of lymphocytes that play a critical role in the pathogenesis of SLE patients with cardiovascular complications remained to be elucidated.

**Objectives:** This study aims to define the dominant population of lymphocytes in mice with combination of lupus and atherosclerosis.

**Methods:** The mouse model of accelerated atherosclerosis in lupus (ApoE-/- Faslgld LDL C57BL/6 mice) was generated from apolipoprotein E-deficient (apoE-/-) and Faslgld C57BL/6 mice. The lupus-like autoimmunity and atherosclerotic lesions was evaluated. The lymphocytes of spleen and peripheral blood were analysed by flow cytometry.

**Results:** The results of PCR and sequencing showed that the double-mutant ApoE-/- Faslgld LDL C57BL/6 mice were generated. Spleens from 5 month-old ApoE-/- Faslgld LDL C57BL/6 mice were significantly enlarged compared with wild type mice (WT mice). ApoE-/- Faslgld LDL C57BL/6 mice displayed a pattern of glomerulonephritis typically found in SLE and showed marked C3, IgG and IgM deposits in the glomeruli. Anti-dsDNA antibody and high levels of creatinine were detected in the serum of ApoE-/- Faslgld LDL C57BL/6 mice. These results indicated that the ApoE-/- Faslgld LDL C57BL/6 mice have typical characteristics of SLE. Oil red O staining revealed that there was significantly increased atherosclerotic lesion area at the proximal aorta in ApoE-/- Faslgld LDL C57BL/6 mice compared with WT mice (figure 1 a,b). The frozen section of myocardium stained by oil red O revealed that lipid deposited in myocardial cells of ApoE-/- Faslgld LDL C57BL/6 mice (figure 1 c,d). As expected, total cholesterol, LDL cholesterol and triglyceride were significantly increased, while HDL cholesterol decreased in the double-mutant mice. These results indicated that ApoE-/- Faslgld LDL C57BL/6 mice had accelerated atherosclerosis.

**FR10293**

**IMBALANCE IN CIRCULATING SUBSETS OF INNATE LYMPHOCYTES IS LINKED TO DISEASE ACTIVITY AND TYPE I INTERFERON SIGNATURE IN PRIMARY SJÖGREN’S SYNDROME**

S.L. Blokland1,2, L.L. van den Hoogen1,2, E.F. Leiten1,2, A.A. Kruize1, T. R. Radstake1,2, J.A. van Roon1,2, 1Rheumatology and Clinical Immunology; 2Laboratory of Translational Immunology, Umc Utrecht, Utrecht, Netherlands

**Background:** Recent studies indicate an important role for innate lymphoid cells (ILCs) in the pathophysiology of rheumatic diseases. In rheumatoid arthritis and spondyloarthopathies elevated numbers of subsets of ILCs have been found at the site of inflammation producing cytokines including IFN-γ and IL-22 and in addition, group 3 ILC have been suggested to be involved in formation of ectopic lymphoid structures in rheumatic diseases. significance of type I IFN in ILC3, which produce IFN-γ and IL-22, in the pathogenesis of SLE has been suggested.

**Objectives:** The aim of this study was to assess the frequency of ILCs subsets in the peripheral blood of patients with primary Sjögren’s syndrome (pSS). We hypothesised that ILC3-like cells would be increased in pSS patients.

**Materials and Methods:** The study included 10 Sjögren’s syndrome patients (8 pSS and 2 secondary Sjögren’s syndrome) and 10 healthy individuals. The frequency of ILCs subsets was assessed by flow cytometry.

**Results:** The results of flow cytometry showed that the frequency of ILC3-like cells was significantly increased in pSS patients compared to healthy controls. The proportion of ILC3-like cells was highest in the peripheral blood of pSS patients.

**Conclusions:** The ILC3-like cells play a role in the pathogenesis of SLE and pSS. Further studies are needed to elucidate the role of ILC3-like cells in the pathogenesis of SLE and pSS.

**Disclosure of Interest:** None declared
ILC3 via increase of Fas (CD95) expression, rendering the ILC more susceptible to apoptosis. Mazzi JACI 2017, Zhang JCI 2015. Duerr Nat Immunol 2016

Objectives: In this study, we explored for the first time the frequency and phenotype of circulating ILCs in pSS and SLE and their relation to the IFN signature.

Methods: Frequencies and phenotypes of ILC subsets and pDCs were assessed by flow cytometry in peripheral blood of patients with pSS (n=20), SLE (n=20) and healthy controls (n=17). Patients were stratified by the presence or absence of an IFN signature as assessed by RT-qPCR on peripheral blood mononuclear cells as previously described. BricARD 2013

Results: ILC1 numbers were increased in peripheral blood of patients with SLE as compared to healthy controls and in pSS patients ILC1 numbers correlated with disease activity (ESSDAI score), serum IgG levels and anti-SSB auto-antibodies (all p<0.05). Numbers of ILC1, ILC2 or ILC3 did not significantly differ between patients with SLE and pSS. However, patients with a high expression of the type I IFN signature had significantly decreased numbers of ILC2 and ILC3 (p=0.04 and p=0.02, respectively). The decrease of ILC2s and ILC3s was related to increased expression of Fas (CD95) on these cells in patients with a high type I IFN signature (both p<0.01).

Conclusions: Both in SLE and pSS, a type I IFN signature is related to reduced numbers of circulating ILC2s and ILC3s in association with increased Fas expression on these cells possibly rendering them more susceptible to Fas/FasL-dependent apoptosis at peripheral sites.

Disclosure of Interest: None declared