aim of our study is to investigate the value of B cells subsets as biomarkers in patients with active LN, in patients at the onset of renal manifestation or with renal flare, and finally in nephritic patients in relation to their clinical and laboratory characteristics at the baseline and during the course of the disease.

**Methods** 50 patients with active LN at disease onset or disease flare were enrolled and evaluated every tree months. Laboratory, immunological and disease activity data were collected at the baseline and at 6(T6), 12(T12), 24(T24), 36(T36) months and at the last follow-up(FU).Number of renal flares, time to renal remission and persistent proteinuria at the last FU were considered. B cell subsets were evaluated at baseline through cytofluorimetry and classified using C27/IgD classification. The characterisation of B cells subsets was realised in 50 LN patients and 37 healthy controls.

**Results** LN patients had a lower percentage of CD19 +cells than controls(9.2% vs 10.6%; p=0.01)as well as a lower percentage of memory unswitched cells CD27 +IgD+(10.7% vs 15.3%; p<0.001) while LN patients had an higher percentage of plasmablasts and double negative memory cells CD27-IgD- (respectively 5.9% vs 1%; p<0.001 and 10,9% vs 4,1%; p=001).

No significant differences regardless B cells subsets were found between early LN patients and long ones as well as between LN patients at the onset and LN patients during renal flare. We found a correlation between an higher disease activity (assessed with SLEDAI 2K) and lower percentage of memory B cells IgD-CD27+(p=0.02).Double negative B cells CD27-IgD- tended to be correlated with an higher disease activity. Of interest the correlation between persistent proteinuria detected during the follow-up and a lower percentage of plasmablasts at the baseline (p=0.015).

**Conclusion** The alteration of B cells subsets is an early event in LN without differences regardless the timing of renal involvement (nephritic onset or later LN development).The association between persistent proteinuria and a lower percentage of plasmablasts at the baseline could be a negative prognostic factor considering the correlation between persistent proteinuria and worse renal outcome.

## PS1:10 EFFECTS OF BELIMUMAB TREATMENT ON B CELL HYPERACTIVITY AND TYPE-I INTERFERON EXPRESSION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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**Purpose** Belimumab, a monoclonal anti-BAFF antibody, has been approved for patients with active systemic lupus erythematosus (SLE) despite standard of care immunosuppressive treatment (ST). However, the interference of belimumab with pathogenetic pathways of SLE is not fully understood. B cell hyperactivity and overexpression of type-I interferons (IFN) have been shown to be key elements in the pathogenesis of SLE. This study shows the effect of belimumab on biomarkers representing B cell hyperactivity and IFN expression in SLE patients.

**Methods** 20 SLE patients treated with belimumab (BT), 82 SLE patients with ST and 30 matched healthy controls (HC) were recruited. Siglec-1 expression on monocytes representing IFN signature, BCMA expression on different B cell subsets and the frequency of activated naive B cells (aNB) in PBMCs were analysed by FACS. Serum levels of BAFF plus soluble receptors sBCMA and sTACI were determined by ELISA.

**Results** Compared to ST, BCMA expression was reduced in BT on naive B (p<0.001) and memory B cells (p<0.05) but not on aNB, plasmablasts and plasma cells. In comparison to HC, BCMA expression was similar on all B cell subsets, except on aNB where it was higher in BT (p<0.001). The frequency of aNB among total B cells was reduced in BT compared to ST (p<0.001) and was comparable to HC. Siglec-1 expression on monocytes did not differ significantly between BT and ST; both groups showed a rise compared to HC (each p<0.001). There was no significant difference after belimumab treatment. Furthermore, serum BAFF levels in ST and BT were higher than in HC (each p<0.001), but did not differ significantly between BT and ST. Serum levels of sBCMA (p<0.05) and sTACI (p<0.001) were lower in BT compared to ST and also after belimumab treatment (each p<0.05). BT's sTACI levels were lower than in HC (p=0.01).

**Conclusions** This study provides deeper insights into the impact of belimumab on several pathogenetic pathways of SLE activity. Regarding the inhibition of B cell hyperactivity, one key pathogenetic element of SLE, belimumab treatment showed distinct advantages. Furthermore, these results suggested that belimumab treatment did not impair the type-I IFN pathway.

## PS1:11 THE INTERFERON BIOMARKER SIGLEC1 REFLECTS DISEASE ACTIVITY IN PAEDIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS

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**Introduction** SIGLEC1 (sialic acid-binding Ig-like lectin 1, CD169) is a monocytic adhesion molecule induced by interferon – α. In adult systemic lupus erythematosus (SLE), SIGLEC1 correlates cross-sectionally and longitudinally with disease activity. The aim of this work was to examine whether SIGLEC1 also reflects the disease activity in paediatric SLE.

**Methods** Over a period of 29 months the disease activity was clinically evaluated using SLEDAI (SLE-Disease Activity Index-2000). In 28 consecutive paediatric SLE patients (mean age 16 years, range 3–38 years, 86% female, 14% male), the number of SIGLEC1 molecules per CD14 +on blood monocyte was quantified using flow cytometry. At the same time, the level of anti-ds DNA-antibody titer (ELISA) and the concentration of complement factors C3 and C4 (nephelometry) were determined. The association between SIGLEC1, C3, C4 and ds DNA-antibody with SLEDAI was estimated using a mixed linear model to model the repeated measurement of parameters within a patient. The cut-off for the change in SIGLEC1 between two consecutive visits to predict minimal clinical improvement or worsening in SLEDAI was chosen on the maximum Youden Index.

**Results** The density of SIGLEC1 molecules on the surface of monocytes based on two visits, correlated with the SLEDAI (128 determinations, betaST 0.22, p<0.012), but not with the C3 (108 determinations, betaST 0.03, p=0,80), the C4 (106 determinations, betaST −0.06, p<0.58) and the anti-dsDNA-antibodies (104 determinations, betaST 0.06, p=0.61). SIGLEC1 is a more change-sensitive biomarker than the conventional laboratory parameters C3, C4 and ds-DNA-Ab. Patients with an increase in SIGLEC1 of >1120 molecules/monocyte between two visits show a higher probability (OR=7.0, p<0.001) of minimal clinical worsening (SLEDAI more/equal 2 points). Patients who show a decrease >2902 SIGLEC1 molecules/monocyte have a higher chance (OR=6.3, p=0.004) to have a clinical improvement (SLEDAI more/equal 2 points).
**Conclusion** This prospective cohort study showed for the first time the significant relationship between the routinely measured interferon biomarker SIGLEC1 and the disease activity in paediatric SLE patients. Thus, SIGLEC1 represents a potential marker for activity monitoring in this disease.

## PS1:13 SYSTEMIC LUPUS ERITHEMATOSUS WITH POSITIVE ANTICENTROMERE ANTIBODIES – A DIFFERENT CLINICAL SUBGROUP?

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**Introduction** Anticentromere antibodies (ACA) are one of the most specific systemic sclerosis (SSc)-related antibodies. The presence of ACA has also been identified in other autoimmune diseases, mainly in SSc overlap syndromes with Sjogren syndrome (SjS), primary biliary cirrhosis and rarely in patients with systemic lupus erythematosus (SLE).
**Purpose** To evaluate the prevalence and clinical significance of ACA in a cohort of SLE patients.
**Methods** Retrospective analysis of all ACA positive SLE patients (ACA +SLE), from a cohort of 270 consecutive SLE patients fulfilling the 2012 SLICC Criteria and/or 1997 ACR Criteria, of a single referral centre, between 2010–2016. Comparative analysis was made with a representative group of 63 consecutive SLE patients without ACA (ACA-veSLE). Data were obtained by medical records review.
**Results** From 270 SLE patients, 10 (3.7%) were ACA+. All ACA +SLE patients were female. The age at the time of diagnosis was not different between the groups (40.9±16.6 years ACA +SLE vs 37.8±16.2 years ACA-veSLE), but ACA +SLE patients had longer disease duration (15.2±17.3 years vs 9.5 ±8.8 years, p=0.002, respectively). ACA +SLE patients had significantly more Raynaud's phenomenon (RP) (p=0.028), but none had a capillaroscopy SSc pattern. Sicca symptoms were also more frequent in ACA +SLE (p=0.013), with only 1 patient with a positive anti-SSA antibody. None of these patients fulfilled criteria for SjS.

Prevalence of arthritis, oral ulcers, alopecia, cutaneous lupus, serositis, neurologic, renal and hematologic involvement was not significantly different between the two groups. Hypocomplementemia at any time of the disease course was more frequent in ACA-veSLE (p=0.016).

Antiphospholipid antibodies were less frequently positive in ACA+SLE patients (20% vs 46%, p=0.1), and none fulfilled criteria for antiphospholipid syndrome (APS) (21% of ACA-veSLE patients with APS).

Apart from RP, SSc-associated clinical characteristics (skin thickening, digital ulcers, telangiectasia, pulmonary arterial hypertension, interstitial lung disease, gastroesophageal reflux and calcinosis) were not present in any of the ACA+SLE patients.
**Conclusions** ACA +SLE patients do not constitute a different clinical subgroup regarding organ involvement, but can associate with a lower probability of concomitant APS. Moreover, although highly specific of SSc, ACA can be identified in SLE patients without SSc overlap, and should not hamper the diagnosis of SLE.

## PS1:14 TRPV1 RECEPTOR ACTIVITY IN LUPUS

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Objective: The etiopathogenesis of lupus is still not fully understood. According to cumulative data, immune, environmental, genetic and neruendocrine factors interact to develop SLE. TRPV1 receptors on neuronal cells are parts of an important inflammatory pathway in autoimmunity. The role of TRPV1 receptors as a calcium dependent channel receptor in pro inflammatory cell reactions is under investigation. Capsaicin an extract of capsicum and oleoresin capsicum has high affinity to TRPV1 receptors. Substance–P discharge from TRPV1 receptors lead to pain relief. The same mechanism may be responsible for resolving inflammation. It is a hypothesis that the skin reaction to Capsaicin is an estimation activity of TRPV1 receptors in autoimmune diseases.
**Method** 29 female lupus patients and 33 healthy age and sex match volunteers who passed the inclusion criteria of the disease and the study were enrolled. For each participant, a 1 × 1 cm2 blotting paper imbrued by 0.1 ml of the capsaicin solution (0.075%) from Sigma Company was put on the volar forearm and covered by a plastic band, to prevent evaporation. The test was then carried out which consisted of time to tingling, induration area (cm2), and redness area (cm2); measured after 15 min.
**Results** The mean age of patients was 30 (25.5–41.5) and controls was 35 (28–48.5) years (p=0.09, z=−1.6). Tingling sensation was sensed by 22 (75.9%) of patients and 12 (36.4%) of controls (p=0.01, x2=13). Redness was observed in 18 (62.11%) of patients and 8 (24%) (p<0.01, x2=9.07). Time to tingling in SLE and controls was 6.5 (4.75–9) and 3 (2–4) min (p=0.02, z=−2.39). Redness area after 15 min, in SLE and controls were 8.05 (0–12) $cm^2$ and 0(0-.24) $cm^2$, respectively (p<0.01, z=−3.38). In lupus group, induration area was 1.5 (0–3.25) cm2 and in controls it was 0 (0–0), (p<0.001, z=−3.38).
**Conclusion** This study suggested that skin reaction to capsaicin in lupus patients is statistically significant stronger than normal individuals. It may stem from more substance–P release from nerve endings or more active TRPV1 receptors in lupus. Studies on TRPV1 pathway in lupus are limited. We suggest that this pathway plays some role in lupus pathogenesis and more researches on this purpose should be considered.