**Background/Purpose** Anti-carbamylated protein antibodies (anti-CarP) were detected in a large cohort of patients with Systemic Lupus Erythematosus (SLE) in correlation with erosive arthritis but not with disease activity indexes. In animal models, T-cells may be activated by carbamylated epitopes playing a role in the development of arthritis. The imbalance between circulating regulatory (Treg) and CD28- effector T-cells was described in active SLE patients, explaining its involvement in disease’s pathogenesis. Actually, no data are available about the possible correlation with these T-cell subpopulations and anti-CarP levels in SLE.

**Methods** Eight SLE patients with a median (10th-90th percentile) SLEDAI-2K=0 (0–4), anti-dsDNA levels=34.1 (15.6–427.4) UI/ml (nv<7), SDI=1 (0–1.3) were enrolled. Serum anti-CarP levels were evaluated using a home-made ELISA (nv<340 AU/ml) and peripheral blood T cell immunophenotyping was done using Flow Cytometry (Beckman Coulter). Treg were defined as CD4+CD127lowCD25high T-cells.

**Results** Enrolled patients showed levels of anti-CarP=189.38±7.7 (4.8–22.5) % of CD4+, CD4+CD28-=30.3 (17.2–35.6) % of CD4+. Analyzing possible correlations among different T-cell subtypes and anti-CarP levels, a significant inverse correlation was found between these autoantibodies and CD4 +CD28- T cells (r=–0.8, p<0.01; Spearman rank correlation). No correlations were found between autoantibodies and other T-cell subpopulations or disease activity/damage indexes.

**Conclusions** In a small cohort of patients with serologically active SLE, anti-CarP autoantibodies were found as negatively correlated to circulating CD4+CD28- T-cells, which were described in association with disease damage, independently of age, gender, disease duration and activity. This suggest a potential role of anti-CarP as marker of SLE with a minor extent of T-cell activation and, consequently, with a possible better prognosis.
based immunocytochemistry assays on SH-SY5Y (human neuroblastoma) cell cultures. The association between serum positivity for AnAb by IHC and a large panel of data (demographic, serologic, SLEDAI, conventional brain MRI, treatment) was investigated by univariate analysis. Multivariate models were fitted with covariates with \( p < 0.05 \) to identify factors independently associated with serum positivity for AnAb; \( p < 0.05 \) were considered statistically significant.

**Results**

AnAb were detected in 23 (82.1%) NPSLE patients and in 16 (39.0%) SLE patients without NP involvement resulting in 82% specificity (95% CI 71%-90%) and 61% sensitivity (95% CI 48%-72%) in differentiating NPSLE from SLE without NP involvement. None of the sera from MS patients (0%) and healthy subjects (0%) showed AnAb. Serum AnAb by IHC were independently associated with NPSLE \( (p<0.01) \) and higher SLEDAI \( (p<0.01) \). No association with specific NPSLE syndrome and brain conventional MRI abnormalities was identified.

**Conclusion**

AnAb are significantly more frequent in patients with NPSLE than SLE. Further studies are needed to identify the unknown neuronal antigens targeted by AnAb in SLE patients.

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**Abstract P18 Table 1**

<table>
<thead>
<tr>
<th>Baseline characteristics of JSLE patients in the Arthritis UK Centre for Adolescent Rheumatology</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number of patients, n (%)</strong></td>
</tr>
<tr>
<td>CKD Without CKD</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>17 (39%)</td>
</tr>
<tr>
<td>14 (80%)</td>
</tr>
<tr>
<td>12 (10-11)</td>
</tr>
<tr>
<td>12 (9-14)</td>
</tr>
<tr>
<td>215 (24-644)</td>
</tr>
<tr>
<td>2 (0-4)</td>
</tr>
<tr>
<td>13 (76%)</td>
</tr>
<tr>
<td>1.5 (0-2.3)</td>
</tr>
<tr>
<td>44 (21-96)</td>
</tr>
<tr>
<td>48 (37-82)</td>
</tr>
</tbody>
</table>

*Numbers are medians (interquartile ranges) unless otherwise stated.
*\( p<0.05 \) is significant

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**Abstract P18**

**Presence of Rheumatoid Factor Was Associated with a Decreased Risk of Lupus Nephritis in Patients with Juvenile Systemic Lupus Erythematosus**

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**Background**

Estimated 10-20% of all patients with systemic lupus erythematosus (SLE) develop clinical disease before the age of 18 years and are therefore classified as juvenile-onset SLE (JSLE). JSLE is characterised by a higher prevalence of lupus nephritis, compared to adult-onset SLE. Chronic kidney disease (CKD) refers to a state of irreversible kidney damage and/or reduction of kidney function that is associated with progressive loss of function over time. Lupus nephritis does not always lead to CKD. However, when it does it is associated with increased morbidity and mortality.

**Objectives**

We aimed to identify clinical and laboratory predictors of CKD development in JSLE patients by comparing the baseline characteristics of JSLE patients with and without CKD to ascertain if there are any significant differences between the two groups.

**Methods**

This is a single-centre retrospective study, who included patients reviewed in our young adult and adolescent clinics. Mann-Whitney U or Chi-square test were performed to compare the characteristics between the patients with and without CKD. We used the Pearson’s (r) or Kendall’s t (tau) correlation to examine if there is any association between the CKD and the baseline characteristics.

**Results**

We identified 44 JSLE patients, out of which 17 (39%) fulfilled the diagnostic criteria for CKD at their last clinical review. The stages of CKD varied from 2 to 5. All patients with CKD also had lupus nephritis, while 5/44 patients (11%) had lupus nephritis without CKD. The baseline characteristics are detailed in the table 1 below. There were statistically significant differences in the treatments used for patients with and without CKD. As expected, the highest dsDNA levels were higher in patients with CKD \( (p=0.03) \). There was also a positive moderate correlation \( (\rho=0.32) \) between raised levels of dsDNA and the development of CKD \( (p=0.008) \). We also found a negative moderate correlation \( (\tau=-0.439) \) between the presence of RF and CKD \( (p=0.04) \).

**Conclusion**

Acknowledging the limitations posed by this small study, we identified a negative moderate correlation between the presence of RF and CKD, which has also been reported in the literature before. We cannot conclude that RF exerts a protective effect against renal disease in SLE, because of the many confounders that might account for a decreased RF in JSLE. Further research using a large JSLE cohort enabling multivariate logistic regression is recommended. DsDNA antibody levels are a measure of disease activity in lupus nephritis and therefore this might explain why patients who developed CKD were noted to have higher anti-dsDNA levels, in comparison with the patients who did not develop CKD.

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**Abstract P19**

**Autoantibody Profile Analysis in SLE Patients**

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**Background/Purpose**

In Systemic Lupus Erythematosus (SLE) the presence of some autoantibodies is related to specific clinical manifestations. We aimed to define SLE patient groups according to an autoantibody profile and to analyze the correlation of these profiles to clinical manifestations.

**Methods**

A cross-sectional observational study of SLE (SLICC 2012 criteria) was conducted. A clinical and analytical evaluation was performed. Clinical manifestations were described according to RELESSER study.

We selected 8 autoantibodies to classify SLE patients: anti-dsDNA, anti-Sm, anti-RNP, anticardiolipin IgG/IgM (aCL IgG/M), anti-\( \beta2 \)microglobulin IgG/IgM (a\( \beta2 \)M), lupus anticoagulant (LA), anti-Ro and anti-La. Immunological profiles