Background and aims Prolactin has been found to be associated with immune regulation in SLE. The aim of this study is to determine the correlation between high prolactin level in comparison with IL – 6 with lupus nephritis disease activity in UKMMC.

Methods In this cross-sectional study, the analysis was conducted in SLE patients who attended Nephrology clinic in UKMMC from August 2015 till February 2016

Results Out of 43 patients with lupus nephritis, 27.9% of the patients had raised serum prolactin. The median of serum prolactin level at 0 min was 19.91 ng/ml (IQR: 15.95–22.65) for active lupus nephritis that was significantly higher as compared to the median of serum prolactin level 14.34 ng/ml (IQR: 11.09–18.70) for patients in remission (p=0.014). The serum prolactin level was positively correlated to SLEDAI (rho = 0.449, p=0.003) and UPCI level in lupus nephritis patients (rho = 0.241, p=0.032). Assessment of serum IL-6 levels found that the active lupus nephritis patients were having a higher median level of 65.91 pg/ml (21.96–146.14) compared to in remission level of 15.84 pg/ml (IQR: 8.38–92.84), (p=0.039). ROC curve analysis of serum prolactin 0 min and serum prolactin 30 min and IL-6 level for prediction of SLE diseases activity provide the cutoff value of serum prolactin 0 min was 14.63 ng/ml with sensitivity 91.7% and specificity 58.1% and AUC of 0.74 (p=0.015).

Conclusions Baseline fasting serum prolactin level was found to be a sensitive biomarker for evaluation of lupus nephritis disease activity.

Background and aims Plasmacytoid dendritic cells (pDC) are potent producers of IFNα in response to CpGc stimulation, confirmed significantly decreased expression of MCP-2/CCL8, IP-10/CXCL10 and ITAC/CXCL11.

Conclusions We found that pDC depletion with JNJ-473 was able to prevent TLR9-induced production of IFNα and various other soluble proteins which are elevated in the sera of SLE patients. We propose that these soluble factors could be useful biomarkers to determine the effectiveness of pDC depletion and the modulation of IFN in SLE.
disease process and are likely to pave the way towards identifying disease-biomarkers for early-diagnosis of LN.

**300** PREDICTORS OF EARLY RESPONSE TO RITUXIMAB IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE): RESULTS FROM THE BRITISH ISLES LUPUS ASSESSMENT GROUP BIOLOGICS REGISTER (BILAG-BR)

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Background and aims The anti-CD20 agent rituximab (RTX) is generally reserved for the treatment of refractory SLE. Whist response is variable no clear predictors of early response have been confirmed. We aimed to explore factors that predict early response to RTX in a nationwide cohort of patients receiving their first RTX course.

Methods The BILAG-BR has recruited patients with refractory SLE starting RTX in the UK since Sept 2010. For this analysis we included patients who received RTX up to November 2015, had active disease at baseline, and had disease activity indices reported at baseline and 6 months. Response was defined as improvement of all active BILAG 2004 systems with no worsening in other systems or SLEDAI-2K; and no increase in glucocorticoid dose at 6 months.

Results In 197 patients (90.36% females) 99 (50.8%) responded at 6 months. In a multivariable model with imputation for missing variables, concomitant cyclophosphamide, higher glucocorticoid doses and lower baseline disease activity. Serological associations with use of concomitant cyclophosphamide, higher baseline oral glucocorticoid dose were associated with better response. A higher baseline global BILAG-2004 score was associated with lower rates of response (Table 1).

Conclusions Early response to RTX in refractory SLE was associated with use of concomitant cyclophosphamide, higher glucocorticoid doses and lower baseline disease activity. Serological and demographic factors did not predict response. Under- standing how concomitant therapy improves longer-term responses and identifying novel biomarkers of response will improve patient selection and overall outcomes for patients receiving this therapy.

Abstract 300 Table 1 Multivariable imputed model after backwards stepwise regression

<table>
<thead>
<tr>
<th>Baseline factor</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concomitant cyclophosphamide</td>
<td>4.50</td>
<td>(1.77, 11.43)</td>
<td>0.002</td>
</tr>
<tr>
<td>Global BILAG at baseline</td>
<td>0.96</td>
<td>(0.93, 0.99)</td>
<td>0.007</td>
</tr>
<tr>
<td>Baseline oral steroid dose</td>
<td>1.03*</td>
<td>(1.00, 1.06)</td>
<td>0.033</td>
</tr>
</tbody>
</table>

*per 1mg increase in daily oral steroid dose. For every 10mg daily increase, OR 1.30

**301** EVIDENCE FOR A PRO-INFLAMMATORY AS WELL AS PROTECTIVE ROLE FOR IL-17A IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background and aims The successful application of IL-17 inhibitors in a number of chronic inflammatory diseases has increased interest for the role of IL-17 in other conditions. We investigated the clinical associations for the predominant family member IL-17A in patients with Systemic Lupus Erythematosus (SLE).

Methods Cross-sectional study of SLE patients (n=102, age 49, 86% female) recruited from a regional registry. IL-17A levels were determined by immunoassay, disease activity by SLEDAI-2K and cumulative damage by SLICC-DI scores. Non-parametric techniques were used to examine the association between IL-17A and disease activity, autoantibody profiles and damage development in SLE patients and for comparisons with healthy controls (n=31).

Results SLE patients had higher IgG levels, lower T cell and B cell counts, but median IL-17A levels did not differ from controls (28.4 vs. 28.4 pg/ml, p=0.9).

In SLE patients, IL-17A levels did not correlate with SLEDAI-2K or SDI, but were inversely related with systolic blood pressure (r=-0.31, p=0.02), years smoking (r=-0.23, p<0.001), cumulative heart (r=-0.24, p=0.03) and malignancy damage (r=-0.18, p=0.06).

Serological correlations for IL-17A existed with levels of IgG (r=0.21, p=0.05), hs-CRP levels (r=0.28, p<0.01), proteinuria (r=0.64, p=0.004) and pre-albumen (r=-0.22, p=0.03).

Longitudinal data showed only modest fluctuations in 17A levels, unrelated to SLEDAI-2K.

Conclusions These data indicate that while IL-17A participates in the inflammatory process in SLE, it also seems to serve a protective purpose in reducing heart disease and cancer in SLE. This dual role is consistent with experimental and clinical data and raises concerns about inhibting IL-17 in SLE patients.

**302** SERUM CYTOKINE ANALYSIS AND TRANSCRIPTIONAL BLOOD PROFILING REVEAL AN ASSOCIATION BETWEEN IL-3 AND IFN IN HUMAN SLE

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Background and aims IFNα, produced by plasmacytoid dendritic cells (pDCs) is a major contributor to SLE pathogenesis. IL-3 promotes pDC survival, but its role in SLE has not been well characterised. This study investigated serum IL-3 and IFNα levels, and a whole blood ‘IL-3 gene signature’ in human SLE.

Methods Serum cytokine levels were measured by ELISA in n=42 SLE donors from The Royal Melbourne Hospital and n=44 healthy donors (HD). IL-3 upregulated genes were determined by RNAs Seq of HD whole blood (WB) stimulated