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 prolactin serum 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. We investigated what additional soluble factors are produced by pDCs and the effect of pDC depletion with JNJ-473 before CpG stimulation, confirmed significantly decreased expression of MCP-2/CCL8, IP-10/CXCL10 and ITAC/CXCL11.

Results TLR9 and TLR7-agonism induced the regulation of thousands of genes, many of which were different IFNα-subtypes. Transcripts of many other secreted proteins such as MCP-2/CCL8, IP-10/CXCL10, ITAC/CXCL11 and MIP-3β/CCL19 were also upregulated. Proteins of these factors were also found to be significantly increased in SLE sera compared to HD.

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.