**Results** Bulk flow sorted cell populations (CD45, epithelial) from kidney samples can separate LN from controls based on gene expression. IFN stimulated genes were differentially expressed in renal CD45 LN cells. Analysis of single cells sorted from 4 LN kidney biopsies revealed major differences in infiltrates composition, with 2 samples demonstrating a high percentage of B cells (average of 18% compared to no B cells in the other 2 samples) and CD4 T cells (18% vs 8%), and low percentage of CD8 T cells (9% vs 23%). A high transcriptomic lupus interferon signature was detected in urine CD45 cells. Distinct infiltrates and distinct expression profiles were detected across patients.

**Conclusions** The PEARL-Phase 0 project shows the feasibility of single cell isolation and transcriptomic analysis from LN kidney and urine. Analyses at a bigger scale in the two next phases of the project will accelerate discovery of new therapeutic targets and identification of biomarkers to guide therapeutic decisions in lupus nephritis and integrate the treatment effect.

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**Background and aims** Siglec-10 (sialic acid-binding immunoglobulin-like lectins-10) is a member of Siglecs family and known to be expressed on B cells to regulate immune tolerance. This study aimed to investigate the expression of Siglec-10 on B cells of SLE and evaluate anti-inflammatory and immunosuppressive drugs impact on the expression of Siglec-10 on B cells in vitro.

**Methods** Peripheral blood mononuclear cells (PBMC) were obtained from patients with SLE (n=57) and healthy donors (n=30). Flow cytometry was performed to examine the expression of Siglec-10 on B cells. The potential association of these measures with the values of SLE disease activity index (SLEDAI) score was also analysed. In vitro cultivation of PBMC with Dexamethasone sodium phosphate and chloroquine phosphate, the expression of Siglec-10 on B cells were detected by flow cytometry after 16 hour.

**Results** The expression of Siglec-10 on B cells was significantly increased in SLE patients compared with normal controls (p<0.001). Moreover, it was positively correlated with SLEDAI (OR=0.277, p<0.05) and elevated in non-treatment patient than patients after anti-rheumatic treatment (p<0.05). In vitro assay, Dexamethasone sodium phosphate and chloroquine phosphate could significantly decreased the expression of Siglec-10 on B cells respectively(p<0.05).

**Conclusions** The results from this study imply that Siglec-10 may play a potential role in progression and pathogenesis of SLE, which may represent a therapeutic target in SLE patients.