investigate and visualise characteristic marker prevalence and co-prevalence patterns.

**Results** Based on the individual marker pattern, patients can often be stratified belonging to different study subgroups. For example, for SLE we show that different reactivity groups exist including patients with different disease activity scores and organ damage patterns.

**Conclusions** We conclude that the approach of a comprehensive prevalence and signature analysis and a vivid data visualisation is useful for any multiplex omics assay.

**Objective** Renal involvement is the most important manifestation of systemic lupus erythematosus, but assessing of inflammatory response in kidneys with non-invasive methods is still challenging. In this study we aimed to define markers of active lupus nephritis (LN) using urine immune profiling.

**Methods** Levels of cytokines (18-plex array) and mRNA expression (40 immune and glomerular injury genes) were measured in urine samples of LN patients with active disease (n=17), during remission (n=16), and in healthy subjects (n=19).

**Results** Urine levels of CCL2, CCL5, CXCL10 and IL-6 were increased in active LN as compared to remission (best discrimination for CCL2), and correlated with LN activity. In the active disease, urinary cell transcriptome showed strong up-regulation of proinflammatory cytokines (e.g. TNF, CCL2, CCL5, CXCL10), Th1 related genes (e.g. CD3G, CD4, TBX21, IFNG), and markers of glomerular damage (NPHS2 [podocin]). Active pattern of gene expression was also observed in 5 patients in remission, who had moderately increased urinary leukocyte count, two patients from this group (40%) developed renal exacerbation during following 3 months. Markers of Th17 immune axis (e.g. IL-17A) were not significantly increased in active LN.

**Conclusions** Active LN patients (also patients at risk of exacerbation) were characterised by marked increase of proinflammatory mediators in the urine. We identified CCL2 chemokine as the most promising marker for monitoring of disease flare.

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**Methods** Demographics, disease information and blood samples were obtained during routine follow-up visits of patients attending Kocaeli University rheumatology outpatient clinic. There were 150 SLE patients (92% female, mean: 46 years). Control group had 85 rheumatoid arthritis patients, 16

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**Results** In our SLE patients we observed higher values of IL10, BlyS and INF1A than healthy controls (p<0.001, p=0.005 and p=0.043 respectively), showing an average values in patients of 13.39±27.73 pg/mL INF1A, 9.99±15.84 pg/mL IL10 and 1811.31±1757.81 pg/mL BlyS. The mean clinical activity measured by SLEDAI was 5.91±5.06.

**Statistical analysis** indicate that INF1A levels are correlated to IL10 levels (p=0.001) and BlyS levels (p=0.034). Due to this finding, we categorised SLE patients by low or high level of the three cytokines: 44 INF1A(-)IL10(-)BlyS(-); 61 INF1A (+)IL10(-)BlyS(-); 5 INF1A(+)IL10(-)BlyS(+); 18 INF1A(+) IL10(+)+BlyS(+); and 14 INF1A(+)IL10(+)BlyS(+). There is a high association of increased IL10-INF1A levels and the increased of clinical activity measured by SLEDAI score (p<0.0001), and to a lesser extent with increased IL10-INF1A-BlyS levels. Patients with high IL10-INF1A and IL10-INF1A-BlyS showed a significant rise in C3-C4 consumption (p<0.001 and p=0.001 respectively) and high anti-dsDNA (p=0.001 and p=0.002 respectively). Patients with increased INF1A-BlyS exhibited high anti-dsDNA (p=0.004) and ENA positivity (p<0.001). In addition, patients with increased levels of IL10-INF1A-BlyS showed ANAs (p<0.001) and antiphospholipid autoantibody positivity (p=0.004).

**Conclusions** The 69% of our SLE patients displayed almost one cytokine increased, being the INF1A the cytokine that mainly is increased. However, increased IL10 levels, irrespective of whether there is also increased levels of BlyS and/or INF1A, is the cytokine which best fits to clinical activity in SLE.

**Purpose** To analyse the association among INF1A, IL10 and BlyS levels and clinical activity in SLE.

**Methods** A cross-sectional, observational study of 142 patients diagnosed of SLE according to SLICC 2012 criteria and 34 healthy controls was performed. In patients a complete bloodtest was made, and clinical data by personal interview was collected. We analysed the serum concentration of IL10, BlyS and INF1A by colorimetric methods. SLE patients were dichotomized as high and low levels for each cytokine based on the cytokine level above 2 SD of the mean in healthy controls. Biostatistical analysis with R (3.3.2.) was performed.

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