model showed a direct negative effect of higher socioeconomic status and a positive indirect effect of higher disease activity on MetS, the latter through corticosteroid dose. MetS did not directly impact HRQOL but had an indirect negative impact on it, through depression.

Conclusions In our causal model, MetS risk factors were related to MetS components. The latter had a negative indirect impact on HRQOL, through depression. Clinicians should consider socioeconomic status and medication and seek to modify disease activity, MetS, and depression to improve the HRQOL of SLE patients.

Background and aims Prolactin has an immune stimulatory effect and may promote autoimmunity by encouraging the development of antigen presenting cells expressing MHC class II and co-stimulatory molecules and modulating IFN-γ secretion. This study aimed to evaluate the relationship between circulating prolactin level and systemic lupus erythematosus (SLE), and to establish a correlation between plasma/serum prolactin levels and SLE activity.

Methods We performed a meta-analysis comparing the plasma/serum prolactin levels in patients with SLE to controls, and examined correlation coefficients between circulating prolactin level and SLE disease activity.

Results Twenty-five studies with a total of 1056 SLE patients and 426 controls were included. Prolactin levels were significantly higher overall in the SLE group than in the control group (SMD=0.987, 95% CI=0.512–1.463, p<4.7x10⁻⁸). Stratification by ethnicity showed significantly elevated prolactin levels in the SLE group in Asian, Latin American, and mixed populations (SMD=0.813, 95% CI=0.137–1.490, p=0.018; SMD=0.981, 95% CI=0.307–1.655, p=0.004; SMD=1.469, 95% CI=0.443–2.495, p=0.005, respectively), but not in the European population. Meta-analysis of correlation coefficients showed a significantly positive correlation between circulating prolactin level and SLE activity (Correlation coefficient=0.379, 95% CI=0.262–0.487, p=4.0x10⁻⁵).

Conclusions Our meta-analysis demonstrated that circulating prolactin levels are higher in patients with SLE and that a significantly positive correlation exists between prolactin levels and SLE activity.

Background and aims Many reports suggest that saliva could be a source of biomarkers capable of detecting certain diseases. However, very few studies conducted to profile autoantibody isotypes in the saliva of autoimmune diseases. This study was performed to establish protein microarray for saliva diagnostics and to identify distinct profiles of salivary autoantibody in patients with systemic lupus erythematosus (SLE).

Methods We constructed antigen microarrays with canonical antigens of SLE as well as cytokines to characterise autoantibodies in matched saliva and serum derived from 17 SLE patients and 13 healthy controls. The autoantibody IgG and IgA isotypes were assayed. The Axon Scanner and GenePix Pro 7.0 were used to determine median fluorescence intensities (MFI) of features and background. Data were analysed using MultiExperiment Viewer and Significance Analysis of Microarray (SAM) algorithm.

Results The dynamic range of detection on the array was 1–10⁴ ng/mL for commercial Abs spiked into saliva. We observed a high degree of specificity for its target antigen, IgG Ab reactivity against specific antigens was found mainly in serum, while IgA Ab reactivity to given antigens was predominant in saliva. SAM identified 7 antigens including BAFF, Ro60, U1-A and Sm/RNP that were significantly more reactive to IgA Ab in the saliva of SLE patients than in healthy controls (false discovery rate <0.01). The hierarchical clustered heat-map successfully placed SLE patients into close subgroups.

Conclusions Protein microarrays facilitate detection of autoantibody in human saliva as well as serum. Saliva profiling revealed that elevated IgA autoantibody reactivity to several targets including BAFF was associated with SLE compared with controls.
Conclusions Using Hitales platform to set up our clinical database can extract medical information conveniently, quickly and efficiently with sufficient accuracy. So far, we only simply analyse the clinical features of SLE patients. With joint of biological specimens’ library and follow up data, the LUPUS puzzle could be learned more.

Background and aims Long noncoding RNAs (lncRNAs) have recently been identified to be tightly linked to diverse human diseases. Systemic lupus erythematosus is an autoimmune disease and renal involvement is the most frequent complication. Inflammatory cytokines produced by renal mesangial cells (RMCs) play a vital role in lupus nephritis (LN). In the present study we investigated the contribution of the lncRNA Enst00000602652 to the pathogenesis to LN.

Methods The high throughput RNAseq data between LN and healthy control was used to screen for candidate lncRNA. SYBR Green quantitative RT–PCR(RT-qPCR) was used to detect the expression of lncRNA and individual interferon-stimulated genes (ISGs). Western blotting and luciferace was used to confirm the regulatory function of lncRNA.

Results LncRNA Enst00000602652 expression was abnormally increased in LN patients and correlated to degree of renal damage. Additionally, Expression of LncRNA Enst00000602652 was induced by stimulation of type I interferon. Silencing Enst00000602652 significantly reduced the expression of a group of chemokines and cytokines, including IFIT1, oas1, etc., which were induced by type I interferon. Furthermore, LncRNA Enst00000602652 affects IFN receptor I and phosphorylation of Jak1 and Stat1.

Conclusions Long noncoding RNA Enst00000602652 is a possible regulator of the IFN signalling pathway in LN. LncRNA Enst00000602652 may contributes to the pathogenesis of LN and provides potentially novel target for therapeutic intervention.

Methods We studied CD8+ T cell differentiation in SLE patients under standard of care (n=80), from two different cohorts. The analyses included phenotyping of T cell differentiation, intracellular cytokine staining and whole blood gene expression.

Results We identified a subset of lupus patients that have elevated numbers of terminally differentiated CD8+ T cells, identified as CCR7−CD45RAint-CD28−. This increase in terminally differentiated CD8+ T cells is accompanied by an increase in perforin and granzyme B production and correlated with a whole blood gene module of cytotoxic activity (p<5x10^-8). More importantly, this phenotype was associated with lupus nephritis (p<0.02).

Conclusions We have identified a lupus endophenotype, characterised by the increase in terminally differentiated CD8+ T cells, which correlated with cytotoxic activity and renal manifestations of the disease. These findings suggest that this group of patients may benefit from therapies that block CD8+ T cell activation and differentiation.