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.

288 CIRCULATING PROLACTIN LEVEL IN SYSTEMIC LUPUS ERYTHEMATOSUS AND ITS CORRELATION WITH DISEASE ACTIVITY: A META-ANALYSIS

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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, $p=4.7x10^{-5}$). 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=0.026–0.487, $p=4.0x10^{-9}$).

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.

289 APPLICATIONS OF PROTEIN MICROARRAY FOR SALIVA DIAGNOSTICS IN AUTOIMMUNE DISEASES

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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 1–10^4 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.

290 INVESTIGATION OF DATA CAPTURE TECHNOLOGY ON CLINICAL PHENOTYPE DISTRIBUTION OF 4150 PATIENTS WITH SLE

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Background and aims To set up the data base of Systemic Lupus Erymatosus using data capture technology (Hitales platform).

Methods Using Optical Character Recognition, Artificial Intelligence, Natural language processing technology to transfer the medical records into structured data which can be easily and freely explored. The medical records are acquired from admitted medical histories of department of Shanghai Renji Hospital.

Results Totally 4150 cases of admitted SLE patients in Dept. of Rheumatology Shanghai Renji Hospital from 2010–2015 were enrolled. The clinical patterns can be easily visualised. 3729 were females and 375 males; The average age was 36.2 ±14.1, with SLEDAI scores of 6.9±5.6. The most items frequently counted in SLEDAI were proteinuria (37.6%), low complement (33.7%) and rash (29.2%). Compared to female patients, male patients were tendency to have protenuira (48.4% vs.36.6%, p<0.01), hematuria (25.8% vs.19.7%, p<0.01). Disease activity evaluated by SLEDAI were highest in summer, however the highest cost in hospital were in winter. 47.0% (1948/4150) patients with lupus nephritis did renal biopsy. The majority pathology type was type IV (27.4%), while 23.6% for type V and 12.1% for V+IV. The most common features counted AI and CI were glomerular cell proliferation (89.6%) and interstitial fibrosis (62.4%) respectively.

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 analysis the clinical features of SLE patients. With joint of biological specimens' library and follow up data, the LUPUS puzzle could be learned more.

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IDENTIFICATION OF THE LONG NONCODING RNA ENST00000602652 AS A NOVEL INFLAMMATORY REGULATOR ACTING THROUGH TYPE I INTERFERON PATHWAY IN SYSTEMIC LUPUS ERYTHEMATOSUS

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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 positive 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.

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HIGH FREQUENCY OF CIRCULATING TERMINALLY DIFFERENTIATED CD8+ T CELLS CHARACTERISE SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS WITH RENAL INVOLVEMENT

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Background and aims Systemic lupus erythematosus (SLE) is a highly heterogeneous disease with limited therapeutic options, where clinical manifestations are the result of multiple pathological mechanisms. The elucidation of these mechanisms is critical for identifying novel therapeutic targets and agents that are more likely to benefit individual patients. Here we investigated the role that CD8$^+$ T cells play in SLE.

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$^-$CD45RA$^{int\text{-}hi}$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 ($p<5x10^{-9}$). 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.

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EFFECT OF HIGH DOSE CHOLECALCIFEROL DURING SLE FLARES IN MIR-146A EXPRESSION, REGULATORY T-CELLS AND IL-17A EXPRESSION

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Background and aims A few microRNAs have known gene expression regulatory roles in innate immunity. The miR-146a, seems to be a negative regulator of innate immunity. Interestingly, miR-146a has been reported to be downregulated in PBMCs of SLE patients, being negatively correlated with clinical disease activity and with IFN levels. The ability of vitamin D to regulate miRs and their emerging relationship have been proposed through several experimental approaches. The aim of this study was to determine the Vitamin D effect in miR-146a expression and in T-Reg and TCD4$^+$ IL-17A producing cells, in SLE.

Methods An interventional study with 3 weeks follow-up of SLE patients with a high dose vitamin D supplementation (50.000 UI or 100.000 UI/Week) was done. We assessed four female patients who had a SLEDAI >6 and at least one BILAG A. At screening, relevant data were compiled: SLEDAI-2K, BILAG score, concomitant therapy, previous SLE manifestations, 25(OH)D levels, T-Reg/IL-17A ratio and miR-146a expression. At Week 3: 25(OH) D levels, T-Reg/IL-17A$^+$ ratio, miR-146a expression, SLEDAI 2 K, BILAG and concomitant therapy.

Results No significant difference were found, regarding Vitamin D levels, before and after supplementation. Regarding Tregs/IL-17A ratio before and after supplementation, no benefits, regarding enhancing of T-regs or decrease of Il 17-A producing cells. No significative differences were found in miR-146a expression between controls and SLE active patients before and after vitamin D supplementation.

Conclusions Severe SLE activity may cause resistance to Vitamin D therapeutical effects, including enhancing of Vitamin D levels and immunogenetic effects.