

42.5% of the patients. miR-146a and miR-155 showed statistically significant correlation with the diagnosis (r_s 0.363 and 0.330, respectively) and age (r_s 0.239 and r_s 0.366, respectively). But none of them correlated with SLEDAI nor with the immunological activity according to ANA, a-dsDNA, a-Sm, a-b2GPI, a-CL antibodies, C3 and C4 complement levels.

Conclusions Our data showed dysregulation of two miRNAs involved in the pathogenesis of SLE by immune cell activation. There was no correlation between the PB levels of these miRNA and the DA as a whole as well as with the immunological activity but larger study is needed to confirm these results.

318 AUTOPHAGY-RELATED PROTEIN P62 EXPRESSION IS ASSOCIATED WITH CLINICOPATHOLOGIC FEATURES AND PREDNISONE PLUS CTX INDUCTION TREATMENT EFFICACY IN LUPUS NEPHRITIS

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10.1136/lupus-2017-000215.318

Background and aims Previous studies found autophagy contributes to the pathogenesis of systemic lupus erythematosus (SLE). Whether autophagy is involved in lupus nephritis (LN) is not elucidated. P62 is a specific substrate that is degraded through autophagy-lysosomal pathway.

Methods Immunohistochemistry Staining was performed to evaluate expressions of p62 in the biopsy kidney tissue of LN patients (n=128) and normal control (n=6). One hundred and five patients were given prednisone+CTX pulse therapy as induction treatment and followed by 24 weeks. Clinicopathologic features and induction phase remission efficacy were recorded and correlated with renal p62 expression level.

Results Compared with the controls, the expression of p62 was significantly decreased in LN biopsy tissues ($p=0.0013$), suggesting increased autophagy in LN kidney. Patients with low expression of p62 had less severe nephritis, showing significantly less proteinuria, fewer interstitial fibrosis score and higher estimated creatinine clearance rates ($p=0.0122$, $p=0.0048$, $p=0.0231$, respectively). Logistic regression analysis revealed that lower renal p62 expression was an independent factor associated with CR ($p=0.025$) (Table 1). Patients with low p62 were more likely and quicker to achieve CR (Person Chi-Square test, $p=0.001$; Kaplan-Meier test, $p=0.0294$).

Conclusions Low renal p62 expression was associated with less severe nephritis and better short-time outcome. Because low p62 expression is the result of high level of autophagy, this data suggested that autophagy might play a protective role in LN kidney. More studies are needed to evaluate the role autophagy plays in multiple organs and cell subtypes in SLE.

319 ASSOCIATION OF FUNCTIONAL IRF7 VARIANTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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10.1136/lupus-2017-000215.319

Background and aims Previous study identified rs1131665 in IRF7 associated with SLE among multiple ethnic groups. This study was undertaken to investigate whether other genetic polymorphisms within KIAA1542/IRF7 confers risk for the development of SLE.

Methods Four SNPs, including rs4963128, rs702966, rs1131665 (Q412R), rs1061502 (K179E) within KIAA1542/IRF7 were genotyped in 784 Chinese SLE patients and 899 controls/IRF7 by using Taqman genotyping assay. Luciferase reporter assay, Co-IP and EMSA were used to assess the effect of K179E polymorphism on the activation of IRF7.

Results Q412R and K179E were significantly associated with SLE in Chinese Han population ($p=5.8 \times 10^{-3}$, $OR=2.33[1.26-4.33]$, $p=2.9 \times 10^{-3}$, $OR=2.82[1.38-5.76]$, respectively. IRF7 3'UTR SNP rs702966 was associated with renal involvement ($p=0.01$ $OR=0.46[0.25-0.85]$). Compared with expression of IRF7 179E, expression of IRF7 179K risk allele resulted in a 4-fold increase in ISRE transcriptional activity and stronger ISRE binding activity in EMSA ($p=0.0002$), suggesting IRF7 179K confers elevated IRF7 activity. Further study found 179K (lysine) carrying IRF7 protein showed higher acetylation compared to 179 E (glutamic acid) IRF7.

Conclusions We detected a novel association between rs1061502 (K179E) and SLE susceptibility. K179E could change the acetylation of IRF7 *in vitro*, which might contribute to the transcriptional activity of IRF7.

320 URINE TWEAK PROTEIN IS A NOVEL BIOMARKER FOR RESISTANT-TO-TREAT LUPUS NEPHRITIS

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10.1136/lupus-2017-000215.320

Background and aims Tumour necrosis factor-like weak inducer of apoptosis (TWEAK) is an inflammatory cytokine that processes via prolonged activation of the NF- κ B pathway. TWEAK plays role in autoimmune diseases like lupus nephritis (LN). TWEAK soluble form and its receptor were found in active LN. We determined whether urinary TWEAK (uTWEAK) levels predict response to standard treatment in a multi-centre clinical trial of lupus patients.

Methods Urine samples were collected at baseline, 3 and 6 month of LN patients from a multi-centre randomized-controlled study (Clinicaltrials.gov ID#NCT01015456). The uTWEAK levels were measured with commercially available enzyme-linked immunosorbent assay (ELISA) kits.

Results All subjects (n=49) were biopsy-proven proliferative class III/IV LN. Median (IQR) patient's age were 32 (29-36) years old. Urine protein creatinine ratio and serum creatinine were 6.32 (6.16-9.20) mg/mg and 0.80 (0.82-1.03) mg/dL. After 6 month treatment of either intravenous cyclophosphamide (IVCY) or mycophenolate sodium (MPS) and steroids,