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### THE RELATIONSHIP BETWEEN IL-10, IL-17, IL-23 AND VITAMIN D LEVELS, AND DISEASE ACTIVITY OF SYSTEMIC LUPUS ERYTHEMATOSUS

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10.1136/lupus-2017-000215.315

**Background and aims** Several cytokines such as IL-10, IL-17, IL-23, and vitamin D have been suspected in the pathogenesis of SLE. However, the association between these cytokines, vitamin D and disease activity is unknown. We aimed to determine the association between IL-10, IL-17, IL-23, vitamin D and SLEDAI score.

**Methods** We included 40 patients with SLE and 20 healthy controls in the study. Clinical and laboratory parameters and, SLEDAI score were evaluated. Serum IL-10, IL-17 and IL-23 were measured by nephelometry and vitamin D by HPLC. Mann-Whitney U and Kolmogorov-Smirnov test were used for statistical analysis.

**Results** The level of vitamin D was significantly lower ( $p=0003$ ), and IL-23 was significantly higher ( $p=0001$ ) in SLE patients compared to healthy controls. There was no significant difference for IL-10 and IL-17 between both group ( $p>0,05$ ). However, a significant correlation between vitamin D and disease duration ( $p=0,02$ ), and between IL-23 and vitamin D ( $p=0019$ ) were found among SLE patients. Vitamin D levels were correlated with SLEDAI score and IL-23 in patients group.

**Conclusions** Although there are studies supporting the role of IL-10 and IL-17 in the pathogenesis of SLE in the literature, there was no significant difference between patients and healthy controls in our study. IL-23 levels were significantly higher, whereas vitamin D levels were significantly lower in SLE patients than in the control group. Also vitamin D levels were negative correlated with duration of disease and IL-23. Levels of IL-23 may be used to evaluate the disease activity, or may be a promising therapeutic approach for SLE patients.

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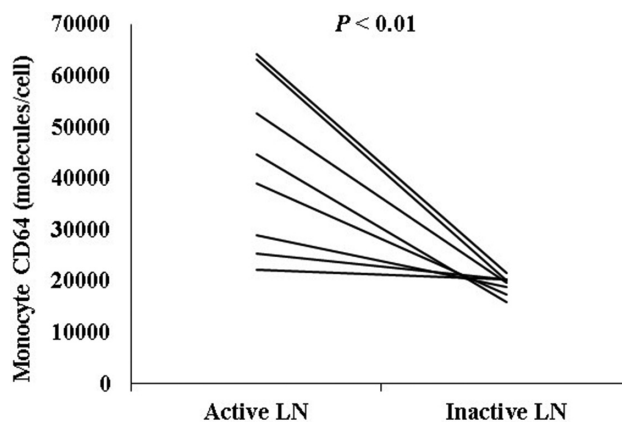
### THE LEVEL OF CD64 EXPRESSION ON MONOCYTE CORRELATES WITH THE ACTIVITY OF LUPUS NEPHRITIS

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10.1136/lupus-2017-000215.316

**Background and aims** Lupus nephritis (LN) is one of the most serious clinical manifestations of systemic lupus erythematosus (SLE). Recently, we have reported that the expression levels of CD64 on monocyte (mCD64), a high-affinity receptor for IgG (FcγRI), correlate with the disease activity of SLE (Lupus 2015;24:1076–80). However, the relation between lupus nephritis (LN) and mCD64 expression is yet to be elucidated. The aim of this study is to investigate whether or not mCD64 expression level correlates with the activity of LN.

**Methods** We quantitatively measured the mCD64 expression levels by flow cytometry in eight SLE patients with biopsy proven LN before and after treatment. All patients fulfilled



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the 1997 American College of Rheumatology classification criteria for SLE. The mCD64 expression levels of the individual patients were measured both at active (presence of proteinuria  $>0.5$  g/day and/or active urinary sediment) and inactive phase (absence of proteinuria and active urinary sediment). The changes were analysed statistically (Wilcoxon signed-rank test).

**Results** The mean  $\pm$ SD of mCD64 expression levels before and after treatment were  $42\,463 \pm 15\,466$  and  $19\,190 \pm 1696$  molecules/cell, respectively ( $p < 0.01$ , Wilcoxon signed-rank test). The mCD64 expression levels in active LN was significantly higher than in inactive LN.

**Conclusions** The mCD64 expression level correlates with the activity of LN, although a larger scale study is needed to confirm the results.

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### DYSREGULATION OF MIRNAS EXPRESSION LEVELS AND DISEASE ACTIVITY IN SLE PATIENTS

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10.1136/lupus-2017-000215.317

**Background and aims** Altered expression of miRNAs have been implicated in the pathogenesis of SLE due to their role in both the adaptive and innate immunity. miRNAs control the differentiation and immunological functions of B cells, induction pathways in T cells, activation, function and maintenance of regulatory T-cells. The aim of our study was to evaluate the peripheral blood (PB) expression of miRNAs in SLE patients and to determine their correlation with the disease activity (DA).

**Methods** 40 SLE patients were included in the study. miR-146a and miR-155 expression levels in whole PB samples were determined by PCR (SYBR Green technology).  $2^{-\Delta\Delta Ct}$  method was used for analysis. 32 healthy donors were used as controls. The DA was determined by DA index (SLEDAI) with 24 descriptors.

**Results** miR-146a was overexpressed in 62.5% of the patients. None of the patients showed underexpression of miR-146a. miR-155 was overexpressed in 50% and underexpressed in

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 PREDNISON 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- $\kappa$ 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,

# Correction: *Dysregulation of miRNAs expression levels and disease activity in SLE patients*

Shumnalieva R, Kachakova D, Monov S, *et al.* Dysregulation of miRNAs expression levels and disease activity in SLE patients. *Lupus Science & Medicine* 2017;4(Suppl 1):A142.3. doi: 10.1136/lupus-2017-000215.317

The authors want to alert readers to the following two errors identified in the published version.

One of the co-authors' name was misspelled. The correct name should have been V Shoumnalieva–Ivanova instead of V Choumnalieva–Ivanova.

At the Results section of the Abstract, the third sentence should read as: “miR-155 was overexpressed in 50% and underexpressed in 7.5% of the patients.”



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*Lupus Sci Med* 2018;0:e000215.317corr1. doi:10.1136/lupus-2017-000215.317corr1

