**INTERFERON STIMULATED LONG NONCODING RNA LINC-RNA-CMPK2 FACILITATES NEUTROPHILS INTERFERON PRODUCTION BY TLR7/8 AGONIST IN SLE**

1. Z. Xue*, Y. Tang, M. Dai, S. Chen, 2N. Shen, 1Renji Hospital- School of Medicine- Shanghai Jia Tong University, Shanghai Institute of Rheumatology, Shanghai, China; 2Cincinnati Children’s Hospital- Medical Centre, The Centre for Autoimmune Genomics and Aetiology CAGE, Cincinnati, Ohio, USA

10.1136/lupus-2017-000215.121

**Background and aims** Neutrophils are important source of high interferon in SLE, we aimed to identify Long noncoding RNAs (LncRNAs) that can be strongly induced by interferon and simultaneously show different expression in neutrophils of SLE and healthy controls. We also investigated how this LncRNA modulate neutrophils interferon production.

**Methods** RNA-seq was performed in two series of samples, interferon stimulated neutrophils samples and SLE versus healthy controls neutrophils samples. LncRNA-CMPK2 was screened out by cross-reference the two RNA-seq results. Neutrophils interferon production was measured by qPCR and ISRE report gene assay after LncRNA-CMPK2 was knocked down using antisense oligos electrotransfection.

**Results** SLE neutrophils produced more interferon when stimulated by TLR7/8 agonist R848 as compared to healthy controls. Neutrophils enhanced interferon production capacity after interferon prime. LncRNA-CMPK2 was an interferon stimulated LncRNA in neutrophils and had an expression level correlated with SLE disease activity. Knock down LncRNA-CMPK2 attenuated interferon neutrophils production.

**Conclusions** Interferon can augment neutrophils interferon production capacity in regenerative feedback. LncRNA-CMPK2 was an important interferon stimulated LncRNA and can facilitate neutrophils interferon production in SLE. Accommodate the expression of LncRNA-CMPK2 could probably supply a new thread of thought to SLE treatment.

**DENDRITIC CELLS DISPLAY ABERRANT TOLL-LIKE RECEPTOR 7/9 RESPONSES IN SYSTEMIC LUPUS ERYTHEMATOSUS**

A.L.Y. Yim, S Yan, A Chan, VSF Chan, CS Lau. LKS Faculty of Medicine- The University of Hong Kong, Department of Medicine, Hong Kong, Hong Kong S.A.R

10.1136/lupus-2017-000215.122

**Background and aims** Systemic lupus erythematosus (SLE) is a multifactorial autoimmune disease that causes multi-organ damages. Plasmacytoid dendritic cells (pDCs) are potent type I interferon (IFN) producers and myeloid dendritic cells (mDCs) are professional antigen presenting cells. Clinically, serum IFN-alpha (IFNα) level correlates with disease severity and mDCs from patients also display activated phenotypes. These observations suggested that different DCs subsets may mediate SLE pathogenesis. Therefore, the aim of this study was to evaluate whether pDCs and mDCs possessed aberrant properties that might mediate SLE progression.

**Methods** The properties of DCs from the murine lupus model New Zealand Black/White F1 (BWF1) were evaluated using flow cytometry, ELISA and qPCR.