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 neutrophils interferon 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.

Results Splenic pDCs abundance was similar before and after disease onset. The induction of CD40, CD80 and MHC II on pDCs upon Toll-like receptor (TLR) 7 or TLR9 stimulation and the level of IFN-alpha produced by pDCs in symptomatic and pre-symptomatic mice was also comparable. In contrast, splenic mDCs expanded in symptomatic mice. These mDCs decreased CD80 and MHC II expression but their ability in stimulating allogenic T cell proliferation was similar to mDCs from pre-symptomatic mice. On the other hand, TLR7 and TLR9 expressions in BWF1 mDCs were higher than mDCs from age- and sex-matched parental NZW controls. The amount of IL-10 and CXCL13 produced by mDCs from symptomatic mice upon TLR7 or TLR9 stimulation was also higher than its pre-symptomatic counterparts. Conclusions Myeloid DCs displayed heightened TLR7 and TLR9 responses in SLE. More work is needed to further dissect how mDCs promote SLE pathogenesis.