Conclusions Using Hitales platform to set up our clinical database can extract medical information conveniently, quickly and 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.

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 luciferase 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 contribute to the pathogenesis of LN and provides potentially novel target for therapeutic intervention.

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.