Over-expression of miRNA-125a could reduce serum autoantibodies and suppress autoimmune reaction in lpr mice.

**Background and aims** Treatment of LN requires an accurate assessment of disease activity.

This study aims to assess the correlation of urinary biomarkers MCP1 and NGAL, with the disease activity in LN.

**Methods** Materials and Methods: Prospective study conducted in a tertiary care centre. 60 patients with SLE were recruited and divided into 3 groups: one with Active LN (n=22), another with Inactive LN (n=20) and third formed of SLE patients with no renal involvement (n=18). Active LN patients underwent renal biopsy. For comparison another group of age and sex matched controls was taken (n=20). Disease activity was correlated with biopsy and baseline characteristics. Urinary MCP1 and NGAL were measured and its correlation with disease activity was analysed.

**Results** In patients with active LN, both UMCP1/Cr and UNGAL/Cr were significantly elevated (92.78, 76.11 pg/ml, p<0.001). In both control group and SLE without renal involvement the values of UMCP1/Cr and UNGAL/Cr were normal (24.44, 22.22 pg/ml in control and 24.3, 22.80 pg/ml in SLE without renal involvement). In patients with inactive LN the values of UMCP1/Cr and UNGAL/Cr were observed to be significantly higher than control (44.18, 38.45 pg/ml, p<0.005) and lower than those of active LN. Values of UMCP1/Cr and UNGAL/Cr were found to be in close correlation with mean rSLEDAI scores of active LN (10) and inactive LN(3.6) and disease activity as per histopathology.

**Conclusions** Levels of urinary biomarkers UMCP1 and UNGAL were significantly elevated in active LN and found to have excellent correlation with histopathological disease activity index and rSLEDAI scores.

**Background and aims** Psoriasis vulgaris (PV) is a chronic inflammatory skin disease characterised by abnormal keratinocyte proliferation and apoptosis. Evidence has showed that transcription factor WT1 plays important role in many pathophysiological processes such as organs development, tumorigenesis and cells proliferation. However, the role of WT1 in PV is still remain unclear. In this study, we will investigate the role of WT1 in the pathogenesis of lesion formation in PV.

**Methods** Skin specimens and peripheral blood mononuclear cells (PBMCs) were obtained from 25 patients with PV and 20 age- and sex-matched healthy subjects. mRNA and protein levels were detected by real-time RT-PCR and western blot. WT1 siRNA and WT1 overexpression plasmid were transfected into HaCaT cells with lipofectamine 2000 respectively. The proliferation and apoptosis of HaCaT cells were detected by CCK8 kit and Annexin V-FITC/PI Apoptosis Detection Kit.

**Results** Compared with normal controls, both the mRNA and protein level of WT1 were increased significantly in psoriatic skin and PBMCs. Transfect with WT1 siRNA inhibited the proliferation of HaCaT cells and promoted HaCaT cells apoptosis, while WT1 overexpression plasmid exhibited the opposite effect on HaCaT cells. The global DNA methylation level of psoriatic skins and PBMCs were elevated accompanied with increased DNMT1 expression. In addition, an positive correlation was observed between WT1 and DNMT1.

**Conclusions** Increased WT1 promotes the keratinocytes proliferation and inhibits the apoptosis of keratinocytes which may mediated by recruiting DNMT1 to its target genes related to cell proliferation and apoptotic pathway.

**Background and aims** To describe the familiality and concordance of manifestations among Filipino patients with systemic lupus erythematosus (SLE) and their affected first degree relatives (FDR).

**Methods** Filipino multiplex SLE families with at least 2 first degree relatives (FDRs) diagnosed as SLE were identified from University of Santo Tomas (UST) Lupus Database. Demographic and disease characteristics were described, and types of relationships within families were analysed for concordance of presenting manifestations using McNemar and Fisher’s Exact tests, with significance at p<0.05.

**Results** The prevalence of familial SLE in the UST Lupus Database (n=2474) was 7.8%. There were 192 patients (173, 90% females) from 95 families (2 families with 3 FDRs), including 25 parent-offspring pairs (23 mother-daughter) and 70 sibling pairs (56 sister-sister). Average age at SLE diagnosis was 31±11.4 SD (range 5–68) years for all affected FDRs. Among parent-offspring pairs, parents’ age averaged 44.8±9.7 SD (range 29–68) years and their offsprings averaged 23.6 +10.6 SD (range 5–35) years at SLE diagnosis, p<0.001. Average age at SLE diagnosis among sibling pairs was 28.6 +11.4 SD (range 9–55) years, with a positive linear association of age at SLE diagnosis between siblings, p<0.001. Most common presenting manifestations were malar rash (47%), oral ulcers (45%), photosensitivity (40%), hematologic (39.9%) and arthritis (39%). Concordance among related FDRs was significant for oral ulcers and hematologic manifestations, p<0.05.

**Conclusions** This study underscores the role of genetics in age onset and clinical expression of lupus.