in vitro with IL-3 for 6 or 24 hours. WB RNASeq analysis was also undertaken in n=31 SLE donors from the Monash Lupus Clinic and n=28 HDs.

**Results** Serum IL-3 levels correlated with serum IFNα (r=0.612, 95% CI 0.455–0.733, p<0.001). IL-3 stimulation in vitro altered 794 genes (−1≥logFC ≥1, FDR<0.05). Thirty-five of these genes overlapped with differentially expressed genes between SLE and HD. These 35 genes were expressed in 28/31 SLE donors, revealing the presence of an ‘IL-3 gene signature’. There was strong correlation between the IL-3 signature and an IFN signature determined by hierarchical clustering of the five hundred most variable genes in SLE donors (r=0.939, 95% CI 0.898–0.964, p<0.0001).

**Conclusions** We have previously reported a novel anti-IL-3Rα mAb (CSL362/JNJ-473), which depletes pDCs and reduces IFNα production, as well as neutralising IL-3 signalling (Oon S, JCI Insight, 2016). An association between IL-3 and IFNα was found in this study, raising the possibility that CSL362 may be especially useful for lupus patients with a dual IL-3/IFN gene signature.

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**303** **URINARY VCAM 1 AS A DISEASE ACTIVITY INDICATOR IN LUPUS NEPHRITIS**

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**Background and aims** Currently we do not have a biomarker that can closely reflect the renal disease activity. So the aim of this study is to study the utility of urinary VCAM1 (Vascular cell adhesion molecule 1) in lupus nephritis.

**Methods** It was a diagnostic case control study. The patients presenting to Rheumatology outpatient department were recruited. Patients were divided into 2 groups, SLE without active nephritis and SLE with active nephritis based on the renal SLEDAI. Urinary VCAM1 was tested in all patients using an early morning spot urine sample using ELISA. Renal biopsy was done in patients with active nephritis. VCAM1 levels were compared with the renal SLEDAI, renal biopsy disease activity (ISN/RPS) and standard of care markers. The results were analysed using SPSS software version 16. The validity and predictive value statistics was presented with 95 percent confidence interval.

**Results** Urinary VCAM1 levels had significant correlation (p=0.01) with disease activity based on renal SLEDAI. However, the correlation between the biopsy findings and VCAM1 levels was not statistically significant. Class 4 and 5 lupus nephritis had higher VCAM1 level than the lower classes. A positive correlation (r=0.38) was found between VCAM1 and double stranded DNA. There was a negative correlation between C3 value and VCAM1 (r=-0.19). The sensitivity and specificity of urinary VCAM1 is 65.22% and 75% respectively. The cut off value of VCAM1 is 23.8 pg/mg of creatinine.

**Conclusions** Urinary VCAM1 may not independently, but combined with other markers may be a promising biomarker for disease activity in lupus nephritis.
304

TNF-α PROMOTER POLYMORPHISMS (G-238A AND G-308A) ARE ASSOCIATED WITH SUSCEPTIBILITY TO SYSTEMIC LUPUS ERYTHEMATOSUS: A STUDY IN P. FALCIPARUM ENDEMIC AREA

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Background and aims Tumour necrosis factor-α (TNF-α) is a proinflammatory cytokine associated with P. falciparum malaria and autoimmune disorders. Elevated plasma TNF-α has been linked to P. falciparum malarial severity and mortality. Higher levels of TNF-α has also been reported in systemic lupus erythematosus (SLE). Two functional common polymorphisms (G-238A and G-308A) at promoter region of TNF-α gene have been linked to SLE susceptibility in different population. In the present report, we conducted a case control study to investigate association of TNF-α (G-238A and G-308A) polymorphisms with susceptibility/resistance to SLE development in a P. falciparum malaria endemic cohort.

Methods A total of 204 female SLE patients and 224 age and sex matched healthy controls were enrolled in the study. TNF-α polymorphisms (G-238A and G-308A) were typed by polymerase chain reaction and restriction length polymorphism (PCR-RFLP). Plasma level of TNF-α was quantified by enzyme linked immunosorbent assay.

Results The prevalence of heterozygous mutants and minor alleles of TNF-α (G-238A and G-308A) polymorphisms were significantly higher in SLE patients compared to healthy controls. Furthermore, heterozygous (GA) and minor allele (A) of TNF-α (G-238A) polymorphism were associated with susceptibility to lupus nephritis. SLE patients displayed higher levels of plasma TNF-α compared to healthy controls. TNF-α (G-238A and G-308A) variants were associated with higher plasma TNF-α in both SLE patients and healthy control.

Conclusions The results of the present study demonstrate that TNF-α (G-238A and G-308A) variants are associated with higher plasma TNF-α level and increased susceptibility to development of SLE in malarial endemic areas.