

**P066** Activation of TLR2 or TLR4 modulates the responses of intrinsic renal and immune cells in systemic lupus  
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Systemic lupus erythematosus (SLE) is a major autoimmune disease and the pathogenic mechanisms of this disease are still poorly understood. The experiments in this article were carried out for studying the role of TLR2 and TLR4 for contribution in lupus nephritis if any. We had administered the synthetic mimics of bacterial triacylated lipopeptides and lipopolysaccharides on every alternate day for two weeks in MRL/lpr mice. The mice were sacrificed at the end of 18 wks of age. The *in vivo* and *in vitro* data was found to show a significant contribution of glomerular endothelial cells (GENC) in recognition of Pam3Cys and LPS via TLR2 and 4 respectively, and in the renal damage by activated immune and non-immune cells in kidney. Real time RTPCR data for GENC have shown to express significant levels of TLR2 and TLR4 mRNA. Glomerular endothelial cells were also found to identify Pam3Cys and LPS and could produce the cytokines upon activation. Also the lupus nephritis was found to be aggravated in Pam3Cys and LPS injected groups. Our results focus light on the contribution of the non-immune renal cells by recognition of the bacterial pathogen mimics via TLR2 and TLR4 in the progression of lupus nephritis.