Background: Systemic lupus erythematosus (SLE) is a multi-organ autoimmune disease that is potentially fatal. There is an unmet need to improve current therapies. In patients with SLE, we observed that serum CXCL5 levels were significantly lower than healthy control subjects and negatively correlated with disease activity[1,2].

Objectives: The aim of this study is to elucidate the effect of supplemental serum CXCL5 in abrogating the pathological processes of SLE.

Methods: Ten doses of exogenous CXCL5 (3ug/kg) was administered to 16-week-old Faslpr mice weekly by intravenous injection. Mice were monitored for 10 weeks. Splenic immune profile was measured by flow cytometry. Circulating cytokine and immunoglobulin profile were detected by Luminex technology. Renal function was evaluated by urinary spot albumin creatinine ratio. In situ renal immune cell infiltration and complement 3 deposition were detected by Haematoxylin and Eosin (H&E) and immunohistochemistry staining. The molecular pathways involved were examined by RNA sequencing.

Results: In Faslpr mice, intravenous administration of exogenous CXCL5 significantly improved mouse survival with concomitant reduction of autobody secretion, proteinuria, complement 3 deposition, neutrophil infiltration and lupus nephritis (histology). Through exploratory analysis of the changes of immune profile and molecular pathways, we found that intravenous CXCL5 reduced inflammation via an orchestrated effect of regulating neutrophil trafficking and modulating helper T cell-mediated immune response. Pharmacokinetic and real-time Polymerase Chain Reaction studies further demonstrated that this orchestration was triggered by a cascade reaction - restoring vascular under-expressed CXCL5 by an exogenous stimulation, re-establishing the normal serum levels of endogenous CXCL5 and reverting the pathological processes of SLE.

Conclusion: Managing the dysregulation of CXCL5 by exogenous supplement may provide a new option for SLE therapy.

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POS0419 ABERRANT SPLICOSEOME AND ALTERED EXPRESSION OF IFN-RESPONSE RELATED GENES ARE HALLMARKS OF MONOCYTES FROM LUPUS PATIENTS WITH RENAL DISEASE
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Background: To date, novel mechanisms such as the involvement of splicing components in lupus nephropathy and its interplay with the transcriptional machinery in innate immune cells have not been evaluated.

Objectives: 1- To identify altered transcriptionic signatures associated with the immune response of monocytes from SLE patients and its association with clinical features. 2- To evaluate the role of the splicosome linked to the transcriptional profile of SLE monocytes. 3- To analyze mechanically the impact of