Background MicroRNAs play vital role in the immunopathogenesis of human and experimental lupus nephritis, but the contributions of miR-326 to renal injury in systemic lupus erythematosus (SLE) remain to be demonstrated. Here we characterize the function of the miR-326 in MRL/lpr mice of lupus nephritis.

Methods We generated MRL/lpr mice overexpression or silence in miR-326 and analyzed the clinical course of the nephritis with respect to albuminuria. In addition, renal Th17/Th17 cells and IL-17A/TGF- expression were detected by flow cytometry and immune-histochemistry respectively.

Results miR-326 overexpression did increase the development of albuminuria in MRL/lpr mice. In contrast, miR-326 silence decreased the development of albuminuria. The characterization of renal CD4 + T cells in miR-326 overexpression mice revealed high numbers of infiltrating Th17 cells and low numbers of infiltrating Tregs. IL-17A and TGF- expression respectively increased and decreased in miR-326 overexpression mice.

Conclusions miR-326 overexpression plays major role in the immunopathogenesis of lupus nephritis in MRL/lpr mice. Thus, our results suggest that miR-326 may be an intriguing new therapeutic approach for patients with lupus nephritis.

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