AUTOIMMUNE CONNECTIVE TISSUE DISEASES

THE ROLE OF MICRORNA-21 IN REGULATING THE ABERRANT DIFFERENTIATION OF T FOLLICULAR HELPER CELLS IN CD4+T CELLS OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction: Increased numbers of circulating T follicular helper cells(Tfh cells) in patients with systemic lupus erythematosus (SLE) play an important role in the development and progression of SLE. However, the mechanism of abnormal differentiation of Tfh cells in SLE is not yet clear.

Objection: To investigate the role of miRNA-21 (miR-21) in regulating the aberrant differentiation of Tfh cells in SLE patients.

Materials and Methods: This study was approved by the ethical committee of the Second Xiangya Hospital of Central South University. 24 SLE patients and 24 healthy controls were recruited. RT-qPCR was used to detect miR-21 and Tfh-related genes expression. Naive CD4+ T cells of healthy controls were transfected with miR-21 Agomir or negative control, and were stimulated with Tfh cells polarized-condition. SLE CD4+ T cells were transfected with miR-21 Antagomir or negative control, and then were stimulated by anti-CD3/CD28. The percentage of Tfh cells was detected by flow cytometry. Western blot was used to detect the protein level of BDH2. P-values < 0.05 were considered as significant.

Results: Compared with healthy controls, the expression of miR-21, CXCR5, PD1, BCL6 and IL21 in CD4+ T cells of SLE patients was significantly increased. Compared with negative control, the percentage of Tfh cells and miR-21 expression were significantly increased in naive CD4+ T cells transfected with miR-21 Agomir. Compared with negative control, the percentage of Tfh cells and miR-21 expression were significantly reduced in SLE CD4+ T cells transfected with miR-21 Antagomir. The protein level of BDH2, which was identified as a target gene of miR-21 in our previous work, was decreased in Naive T cells transfected with miR-21 Agomir and was increased in SLE CD4+T cells transfected with miR-21 Antagomir compared with respective contorls.

Conclusion: The overexpression of miR-21 promotes the aberrant differentiation of Tfh cells in SLE CD4+T cells.