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EXPRESSION OF IL-23/TH17 PATHWAY IN A MURINE MODEL OF COXSACKIE VIRUS B3-INDUCED VIRAL MYOCARDITIS

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Background The IL-23/Th17 pathway is implicated in the pathogenesis of a number of chronic inflammatory and autoimmune diseases. Whether it regulates the viral myocarditis (VMC) is unknown.

Results To examine the pathogenesis role of IL-23/Th17 axis in VMC, we used male BALB/c mice to induced VMC by Coxsackie virus B3 (CVB3) peritoneal injection. IL-23, IL-17, and signal transducer and activator of transcription 3 (STAT3) mRNA in the myocardium of VMC mice were assessed by semi-quantitative RT-PCR. IL-23 and IL-17 protein from blood serum were evaluated by ELISA. Phosphorylated-STAT3 (p-STAT3) protein expression in the myocardium was evaluated by immunohistochemical staining. Flow cytometric analysis was used to evaluate the frequencies of Th17 subsets. Isolated $CD4^+$ T cells from VMC mice were cultured with recombinant IL-23(rIL-23) in vitro. In addition, a STAT3-specific inhibitor (S3I-201) was used to test whether regulation of STAT3 could be partly responsible for Th17 diminution. Results showed that expression of IL-23, IL-17, STAT3 mRNA and protein increased in VMC mice. When purified $CD4^+$ T cells derived from VMC mice were cultured in vitro with rIL-23, the frequency of Th17 cells was dramatically increased, accompanied by significantly enhanced production of IL-17 in the supernatants of cultured $CD4^+$ T cells. S3I-201 significantly restrained Th17 cell proliferation.

Conclusions The IL-23/Th17 pathway axis is strongly expressed in murine VMC, identifying a novel pathway of potential significance in viral myocarditis.

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