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Expression of miR-98 in myocarditis and its influence on transcription of the *FAS/FASL* gene pair

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ABSTRACT. Myocarditis is a common cardiovascular disease and frequently occurs in children and teenagers. It is believed to be caused by both endogenous and exogenous factors, among which *FAS/FASL* gene pair-induced cell apoptosis is a major mechanism of myocardial cell injury. A previous study has detected low expression of microRNA (miR)-98 in myocarditis patients. Therefore, in this study we investigated the functional implications of miR-98 with respect to the disease. We carried out a case-control study including 50 myocarditis patients and 50 healthy individuals. Total RNA was extracted from peripheral blood plasma. Expression levels of miR-98 and the *FAS/FASL* gene pair were determined by real-time fluorescent quantitative polymerase chain reaction. The interaction between miR-98 and the *FAS/FASL* pair was visualized by dual-luciferase reporter assay. The expression of the *FAS/FASL* gene pair was further detected by transfecting with an miR-98 mimic or an miR-98 inhibitor. The content of miR-98 in the peripheral blood of the myocarditis patients was significantly lower than in the

healthy individuals. However, the *FAS/FASL* genes were upregulated by 1.68-fold in the myocarditis patients. miR-98 was shown to interact with the 3'-untranslated region of the *FAS/FASL* gene pair. The inhibition/facilitation of miR-98 expression in myocardial cells can modulate apoptosis. miR-98 was downregulated in the peripheral blood of myocarditis patients. It may interact with the *FAS/FASL* gene pair to further modulate cell apoptosis.

Key words: Myocarditis; MicroRNA-98; *FAS/FASL* gene pair; Cell apoptosis

