The mechanism of long-term regular exercise intervention on liver injury in patients with NAFLD based on miR-146a regulation of TLR4/NF-KB signaling pathway

Qi Peng, Jiaqin Chen, Wei Chen, ChangFeng Shao, Afang Yuan
Hunan Normal University

**Objective** To investigate the effects of long-term regular exercise on hepatic function in patients with non-alcoholic fatty liver (NAFLD) using blood biochemistry and liver fibrosis markers, and to compare the differential expression of cytokines related to TLR4/NF-KB signaling pathway. A preliminary discussion was made on its regulation mechanism.

**Methods** Forty patients with NAFLD diagnosed in the Hunan Normal University School of Medicine, according to the degree of steatosis and exercise intervention, the patients were divided into control group (NAFLD group) 20 cases and long-term regular exercise group 20 cases, and the same time in our hospital Twenty patients with physical examination were normal controls; general data of all subjects, ALT, AST, GGT, serum type III procollagen (PCIII), hyaluronan (HA), and type IV collagen (CIV) were examined; Fluorescent quantitative PCR was used to detect the differential expression of TLR4/NF-KB signaling pathway-related cytokines and miR-146a in the blood of each group of subjects, revealing the effects and possible mechanisms of long-term regular exercise on liver fibrosis.

**Results** Compared with the normal group, the levels of serum ALT, AST, GGT, PCIII, HA, and CIV in the non-alcoholic fatty liver patients were significantly lower in the long-term regular exercise group than in the control group; blood TLR4, NF-KB, MY-D88 Compared with the control group, the gene expression level was significantly downregulated in the long-term regular exercise group.

**Conclusions** Long-term regular exercise can effectively reduce nonalcoholic inflammatory liver injury and has a clear anti-fibrotic effect. Its mechanism may be related to long-term regular exercise through regulating the TLR4/NF-KB signaling pathway related factors and the regulation of molecular miR-146a, reducing inflammation and preventing the formation of fibrosis.