

The novel therapeutic mechanisms of leonurine for treating cardiac fibrosis via inhibiting FMO2

Zhaoyi Li

Macau University of Science and Technology, China

Cardiovascular diseases (CVDs) are the number 1 cause of death globally, including myocardial infarction, heart failure, atherosclerosis, and myocardial hypertrophy. Heart failure (HF) is a slow, gradual and irreversible pathological process with cardiomyocyte apoptosis, the final stage of most cardiovascular diseases. The production of reactive oxygen species (ROS) can induce cardiomyocyte apoptosis, leading to cardiac dysfunction and promoting HF. Flavin-containing monooxygenase 2 (FMO2) is an enzyme closely related to oxidative stress and cardiac fibrosis. Herba leonuri (HL) is a traditional Chinese medicine with a long history of development and application in China. Recently, modern research has found that there are various chemical components in HL. However, alkaloids are the most important biologically active compounds, especially leonurine. Therefore, this manuscript studied the novel mechanisms of leonurine on HF in vitro and in vivo. Leonurine was found for the first time to inhibit the expression of FMO2 protein, which interacted in various redox reactions. Therefore, FMO2 may reduce the accumulation of ROS though its specific mechanism needs to be further studied.

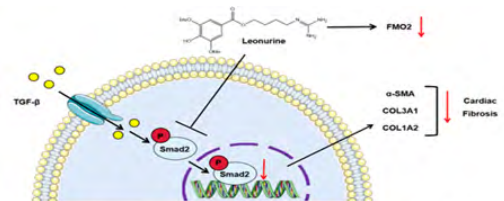


Figure 1. The novel cardioprotective effect of leonurine for treating cardiac fibrosis. Leonurine inhibits FMO2 expression and Smad2/3 phosphorylation, modulates TGF-β/SMAD signaling pathway, reduces the expression of various pro-fibrotic proteins such as α-SMA and collagen (COL3A1 and COL1A2) and ultimately treats cardiac fibrosis.

Speaker Biography

Zhaoyi Li studied at the Macau University of Science and Technology for a master's degree in Chinese Pharmacy and is engaged in cardiovascular disease and pharmacology research in the State Key Laboratory, focusing on the research of new targets for cardiovascular disease.

389965840@qq.com

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