

The role of AMPK pathway in mitigating fluoxetine- induced hepatic damage: An experimental study in rats

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Drug-induced liver injuries (DILIs) can have various clinical manifestations ranging from asymptomatic abnormal liver tests to symptomatic acute liver disease. Several studies have indicated that fluoxetine can lead to hepatotoxicity and affect liver enzyme activity. Metformin suppresses SREBP1 and reduces lipogenesis as well as fat accumulation by activating AMPK, which can have a protective effect on the liver. We investigated the role of the AMPK signaling pathway induced by metformin in preventing fluoxetine-induced hepatotoxicity in rats. Rats were divided into four groups of five at random, including Control, Fluoxetine-induced hepatotoxicity (Flux), Flux + Metformin, Flux + Metformin + Dorsomorphin. Hepatotoxicity was induced by fluoxetine and then the Serum-Specific Marker of the Liver, oxidative markers, and histopathologic were measured. Our finding demonstrated that activation of AMPK by metformin increased antioxidative activity and decreased necrosis, edema, and inflammatory cells in the liver. However, the results of the fourth group indicated that dorsomorphin administration reduced the beneficial effects of metformin by inhibiting the AMPK signaling pathway. In conclusion, the findings indicated that the AMPK signaling pathway in fluoxetine-induced hepatotoxic might be mediated through the decrease in the levels of inflammation and inhibition of oxidative stress in the liver.

Biography

Dr. Amin Hasanvand studied Veterinary Medicine at The Islamic Azad University Shahrekord Branch (IAUSHK), Iran in 2007. He then joined the research group of Prof. Dehpour at the Department of Pharmacology, Tehran University of Medical Science. He received his PhD degree in 2016 at the same University. In the same year, he was accepted as a member of the academic board of Lorestan University of Medical Sciences and is currently continuing his work with the rank of associate professor. He has published more than 40 research articles.

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