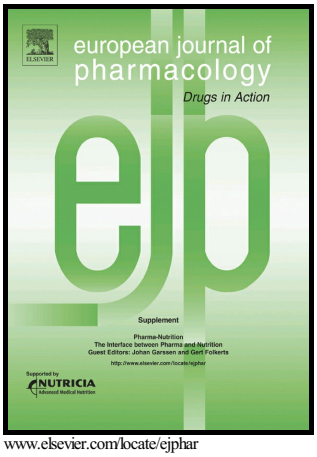

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The role of urotensin-II and its receptors in sepsis-induced lung injury under diabetic conditions

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Abstract

This study aimed to investigate the potential role of urotensin-II receptors in sepsis-induced lung injury in diabetic mice using urotensin-II receptor agonists and antagonists.

A total of 110 male CD1 mice were used in this study. Diabetes was induced by 200 mg/kg streptozotocin. One month after diabetes induction, the cecal ligation and puncture-induced polymicrobial sepsis model was applied in the diabetic and non-diabetic mice. Low and high doses of human urotensin-II agonist (HU-II) and antagonist (palosuran) were administered one hour after sepsis induction. HU-II administration was repeated in two-hour intervals. Blood and tissue samples were collected at 6 and 12 H after sepsis induction for biochemical, molecular, and histopathologic examinations.

Regarding to the lungs mRNA expression and immunohistochemistry results of TNF-α, IL1β, IL6, and NF-κB, it was observed that cytokine levels significantly increased in the diabetes group and the sepsis groups compared to the healthy group; this increase was significantly higher in the diabetes-sepsis groups. Our biochemical (superoxide dismutase, glutathione, and malondialdehyde) and histopathological findings in the lungs also supported