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Protective Effect of Linalool Against Lipopolysaccharide-Induced Acute Kidney Injury in a Rat Model

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Objectives: The retention of urea and other nitrogenous waste products and dysregulation of extracellular volume and electrolytes is the characteristic hallmark of acute kidney ischemia/reperfusion (I/R) injury. Despite the advances in therapeutic techniques, the mortality and morbidity of patients remain high and have not appreciably improved. Thus, the present study investigated the effect of Linalool (LIN) on lipopolysaccharide (LPS)-induced acute kidney injury in a rat model.

Methods: Kidney injury was induced in male Sprague-Dawley rats by injection of LPS through the tail vein. The rats were treated with 5 µg/kg body weight LIN within 12 h of the LPS administration. The urine of the rats was collected over a period of 48 h for determination of calcium and creatinine concentrations. Blood urea nitrogen in the serum was analyzed using a BC-2800 Vet Animal Auto Biochemistry Analyzer. On day 3 after treatment, the rats were sacrificed to extract the kidneys.

Results: Treatment of the endotoxemia rats with LIN caused a significant decrease in the level of kidney injury molecule-1 and blood urea nitrogen. LIN treatment significantly decreased the level of calcium in the kidney tissues compared to those of the untreated endotoxemia rats. The level of malonaldehyde (MDA) in the kidney tissues was significantly reduced in the endotoxemia rats by LIN treatment. The results from immunohistochemistry revealed a significant decrease in the expression of osteopontin (OPN) and CD44 levels. The endotoxemia rats showed significantly higher levels of TUNEL-positive stained nuclei compared to the normal controls. However, treatment of the endotoxemia rats with LIN resulted in a significant decrease in the population of TUNEL-positive cells.

Conclusions: Linalool may be a promising candidate for the treatment of acute kidney injury.