

Piceatannol Protects Against Isoproterenol-Induced Myocardial Injury in Rats

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ABSTRACT

Myocardial infarction (MI) is a critical medical emergency and an advanced stage of coronary artery disease. The disease's pathophysiology includes oxidative stress, inflammation, and cellular apoptosis. Isoproterenol (ISO), is a synthetic adrenergic agonist that mainly acts on adrenergic (β) receptors. Its administration in rats instigates significant myocardial strain, resulting in myocardial damage that resembles myocardial infarction. Piceatannol (PIC), a natural stilbene compound, exhibits free radical-scavenging, tumor-inhibiting, and chemo-preventive activities. This investigation intended to evaluate the cardioprotective benefits of piceatannol in mitigating cardiac damage provoked by ISO in rats. After 28 days of PIC treatment (5 or 10 mg kg⁻¹), rats received two doses of ISO (85 mg kg⁻¹) 24 hours apart to induce myocardial damage. The administration of ISO resulted in significant changes to ECG parameters, elevated blood levels of cardiac enzymes, and histological alterations linked to myocardial damage. It also caused oxidative damage, as evidenced by elevated malondialdehyde (MDA) levels, alongside a decline in glutathione (GSH) concentration and superoxide dismutase (SOD) function. Furthermore, ISO stimulated the synthesis of inflammatory markers such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and nuclear factor-kappa B (NF- κ B) in the myocardium, concurrently elevating the Bax/Bcl-2 ratio. Conversely, pretreatment with PIC significantly normalized ECG measurements, mitigated the increase of cardiac enzyme levels in the serum, and maintained the normal histological characteristics of the heart. PIC elevated cardiac antioxidant enzyme levels and activity, diminished lipid peroxidation products, and inhibited the expression of inflammatory markers as well as the Bax/Bcl-2 ratio. These results suggest that PIC had a cardio-protective effect in this model.

Keywords: Piceatannol; Isoproterenol; Myocardial injury; Oxidative damage; Inflammation; Apoptosis

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