





## Article

# Thymoquinone Produces Cardioprotective Effect in $\beta$ -Receptor Stimulated Myocardial Infarcted Rats via Subsiding Oxidative Stress and Inflammation

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**Abstract:** Earlier studies reported that long-term treatment with thymoquinone (TQ) at a high dose (20 mg/kg) exerts a cardioprotective effect against isoproterenol (ISO)-triggered myocardial infarction (MI) in rats. In the present study, we tested the hypothesis that TQ, as a potent molecule, can exhibit cardioprotective effects at the lower dose for a short-term regimen. The rats were administered with TQ (5 mg/kg, intraperitoneal) at the 4 h interval for 2 days. ISO (100 mg/kg/day, subcutaneous) was given for 2 days to produce MI. ISO challenge results in deformation in ECG wave front, elevated left ventricular (LV) end-diastolic pressure, and reduced LVdP/dtmax and LVdP/dtmin. The levels of the cardiac biomarker in serum, such as creatine kinase MB, alanine aminotransferase, and aspartate aminotransferase, were increased. In the myocardium, a rise in malonaldehyde and decreased superoxide dismutase, glutathione, and catalase contents were observed. Furthermore, increased levels of tumor necrotic factor- $\alpha$ , interleukin-6, and interleukin-1 $\beta$  were observed in the myocardium. TQ pretreatment significantly normalized alterations in hemodynamic parameters, strengthened the antioxidant defense system, and decreased the contents of pro-inflammatory cytokines and hepatic enzymes as compared to the ISO group. Based on the results, TQ appears to be cardioprotective at low doses, and effective even administered for a shorter duration.

**Keywords:** myocardium; glutathione; pro-inflammatory mediators; antioxidant; hemodynamic

## 1. Introduction

Globally, coronary heart disease (CHD) is the leading cause of morbidity and mortality. Myocardial infarction (MI) is the most common form of CHD [1]. Further, the most important hallmark of MI is heart failure which is caused by poor blood supply to the heart or deteriorating hemodynamics, resulting in cardiac death or severe hemodynamic impairment [2]. Heart failure is linked to elevated levels of certain biochemical parameters such as high lipid profile, and blood sugar along with several biological changes including high blood pressure, obesity, and aging [3]. The non-selective adrenoreceptor agonist isoproterenol (ISO) is known to trigger oxidative stress in the cardiac tissues, resulting in high-level tissue necrosis. Additionally, it increased chronotropic, inotropic, and Ca<sup>2+</sup> overloads. In the process, oxidative catecholamine metabolism produced an excess of free

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