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The Role of High-Intensity Interval Training in Reducing Lung Inflammation and Apoptosis Following Renal Ischemia-Reperfusion Injury

INTRODUCTION:
Renal ischemia-reperfusion (I/R) injury is a common complication in kidney, cardiac, and vascular surgeries (1). Acute kidney injury (AKI) has been linked to damage in distant organs, including the heart, lungs, liver, and brain, contributing to its high mortality rate (2). Previous studies show that a four-week high-intensity interval training (HIIT) program can reduce renal I/R injury and protect the heart. With HIIT gaining popularity for its time efficiency, fat loss, and health benefits (3), this study examines whether HIIT can also alleviate lung injury from renal I/R, potentially informing future exercise prescriptions for health promotion.
METHODS:
Thirty male Sprague-Dawley rats were assigned to three groups: Sham, Renal I/R, and HIIT. The Sham group underwent abdominal opening without further intervention, while the other two groups underwent 60-minute renal artery occlusion followed by 24-hour reperfusion. Lung injury was assessed using the wet-to-dry weight ratio and concentrations of tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6) in bronchoalveolar lavage fluid (BALF). Serum TNF-α concentrations were measured using enzyme-linked immunosorbent assay (ELISA). Lung tissues were analyzed via hematoxylin and eosin (HE) staining to assess histological damage and terminal deoxynucleotidyl transferase dUTP nick-end labeling (TUNEL) staining to evaluate apoptosis.

RESULTS:
The Renal I/R group exhibited increased lung histological damage, a higher wet-to-dry weight ratio, and enhanced apoptosis. TNF-α, IL-1β, and IL-6 concentrations were elevated in both serum and BALF, along with increased TNF-α expression in lung tissue. In contrast, serum and BALF IL-10 concentrations were reduced. HIIT intervention alleviated lung histological damage, reduced the wet-to-dry weight ratio, and inhibited apoptosis. Additionally, it mitigated the elevation of TNF-α, IL-1β, and IL-6 concentrations while attenuating the reduction of IL-10. Western blot analysis showed an upregulation of B-cell lymphoma 2 (Bcl-2)

and a downregulation of Bcl-2-associated X protein (Bax) in lung tissue, suggesting enhanced anti-apoptotic signaling.

CONCLUSION:
A four-week HIIT intervention significantly reduced renal I/R-induced lung injury, likely by modulating inflammatory cytokine production and inhibiting apoptosis. Given its efficiency and accessibility, HIIT may be a cost-effective strategy for disease prevention. Its potential lung-protective effects suggest benefits for pulmonary resilience and reduced risk of inflammation- or oxidative stress-induced lung injury. Further studies are needed to explore its broader implications for respiratory health, particularly in conditions linked to lung inflammation or ischemic injury.
Reference:
1. Snoeijs, et al., Am J Physiol Renal Physiol, 2010
2. Doi, et al., Clin Exp Nephrol, 2011
3. Atakan, et al., Int J Environ Res Public Health, 2021

apoptosis; lung injury; high intensity interval training; renal ischemia-reperfusion