## **Accepted Manuscript**

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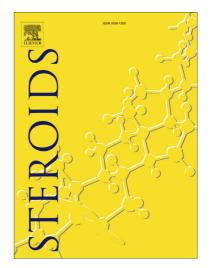
PII: S0039-128X(18)30032-1

DOI: https://doi.org/10.1016/j.steroids.2018.02.004

Reference: STE 8231

To appear in: Steroids

Received Date: 31 July 2017 Revised Date: 24 January 2018 Accepted Date: 15 February 2018



Please cite this article as: Kara, M., Ozcagli, E., Kotil, T., Alpertunga, B., Effects of Stanozolol on Apoptosis Mechanisms and Oxidative Stress in Rat Cardiac Tissue, *Steroids* (2018), doi: https://doi.org/10.1016/j.steroids. 2018.02.004

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Effects of Stanozolol on Apoptosis Mechanisms and Oxidative Stress in Rat Cardiac Tissue

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**Abstract** 

Stanozolol is a widely used 17α-alkylated anabolic androgenic steroid (AAS) derivative. Despite

stanozolol's adverse effects, its effect on oxidative stress parameters and mitochondrial apoptosis pathway is not

clearly defined. In our study, thirty four male Sprague-Dawley rats were divided into 5 groups as control (C),

vehicle control (VC), steroid (ST), vehicle control-exercise (VCE), and steroid-exercise (STE). Animals were

subcutaneously administered stanozolol 5 mg/kg in steroid groups and propylene glycol 1 ml/kg in the vehicle-

control groups. On the 28th day-after sacrification, oxidative stress (MDA, GSH, PC, SOD, CAT) and apoptosis

parameters (TUNEL, Cytochrome-c) in cardiac tissue were evaluated. Also, blood vessel morphology of cardiac

tissue was evaluated with Verhoeff-van Giesen staining. It has been demonstrated that stanozolol administration

triggers apoptosis by using TUNEL assay and cytochrome-c immunohistochemical staining intensity, while this

effect is significantly reduced in the presence of exercise. In conclusion, the present study demonstrated that

stanozolol administration induces apoptosis with increasing PC and CAT levels, while GSH, MDA and SOD

parameters do not reveal any significant change. Exercise has a protective role in stanozolol induced oxidative

stress and apoptosis. According to Verhoeff -van Giesen staining results for blood vessel morphology

assessment, it has been seen that exercise has a protective role on cardiac blood vessels. This mechanism needs

further investigations with long term exposure studies for clarifying possible pathways.

**Key Words:** Stanozolol, Oxidative stress, Cardiac tissue, Apoptosis, Immunohistochemistry

1. Introduction

Anabolic androgenic steroids (AASs) are the synthetic derivatives of male hormone, testosterone and they have

been reported as the most frequently detected doping drugs [1]. Currently, more than 100 types of AASs are

available on the market. AASs are known to be used widely at supraphysiological doses which are

approximately 10-100 times higher than therapeutic doses. In recent years, several studies have been reported

that supraphysiological doses of AASs are associated with cardiovascular, hepatic, endocrine, reproductive and

neurologic adverse effects [2,3,4]. One of the most important adverse effect of AASs is ventricular hypertrophy,

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