PUNICALAGIN MITIGATES TESTOSTERONE PROPIONATE (TP)-INDUCED BENIGN PROSTATIC HYPERPLASIA (BPH) IN CASTRATED RATS VIA OXIDO-INFLAMMATORY AND APOPTOTIC MECHANISM

Monsuru Sanni* and Oluwatosin Adaramoye*

*Drug Metabolism and Toxicology Research Laboratories, Department of Biochemistry, College of Medicine, University of Ibadan, Ibadan, Nigeria.

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Abstract

INTRODUCTION AND OBJECTIVE: Inflammation and oxidative stress plays a central role in the etiology and progression of Benign Prostatic Hyperplasia (BPH), a common urological disorder in aged men. Punicalagin (PN), a bioactive compound found in the pomegranate (*Punica granatum*), exhibits potent antioxidant and anti- inflammatory properties. Here, we are reporting the oxido-inflammatory and apoptotic effect of Punicalagin (PN) on testosterone propionate (TP)-induced benign prostatic hyperplasia (BPH) in castrated Wistar rats.

METHODS: Castrated Wistar rats weighing (180 - 240 g) had exogenous, daily administration of testosterone propionate (TP) at 3 mg/kg for four weeks, and staggered doses of PN (50 mg/kg) and Finasteride (FT) at (10 mg/kg) three times weekly for four weeks. The rats were assigned into five groups of six animals each: castrated control, castrated rats that received TP [BPH], castrated rats that received TP and PN [BPH + PN], castrated rats that received TP and FT [BPH + FT], and castrated rats that received TP, PN and FT [BPH + PN + FT]. Prostatic tissues were collected for biochemical and immunohistochemical examination. Prostatic oxidative status (SOD, CAT, GSH, GST, GPx and MDA), Inflammations (NO and MPO) were assessed biochemically and immunohistochemically (COX2 and iNOS). Testosterone level was assessed using ELISA method. Prostatic cell death regulators (p53, Bcl-2 and Bax) and prostatic receptors (AR and ER) were determined using immunohistochemistry.

RESULTS: Our data showed that PN significantly ($p<0.05$) reduced the prostatic weights and testosterone level in BPH rats. The decreased activities of SOD, CAT and GPx were significantly ($p<0.05$) increased by PN in BPH rats. The increased level of LPO and NO and activities of MPO and GST in BPH rats were significantly ($p<0.05$) reduced by PN indicating antioxidant and anti-inflammatory activities. PN also suppressed the expression of AR, ER, COX2 and iNOS which further suggest anti-inflammatory activities. PN suppressed the expressions of Bcl-2 and increased the expression of Bax and p53 suggesting anti-apoptotic effects.

CONCLUSIONS: PN mitigates testosterone propionate induced BPH via mechanism involving oxido-inflammatory and apoptotic effects in castrated rats.

Introduction

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Methods

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Results

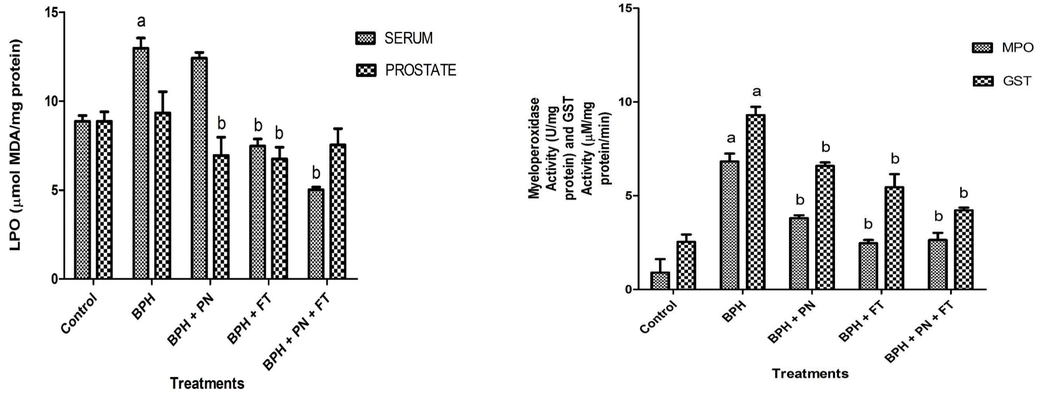

Figure 1: Effects of Punicalagin on Lipid Peroxidation (LPO), Myeloperoxidase (MPO) and Glutathione-S-transferase (GST) activities in prostate of testosterone propionate-induced benign prostatic hyperplasia (BPH) rats.
a = significantly ($p<0.05$) different from control rats.
b = significantly ($p<0.05$) different from BPH treated rats.

Results

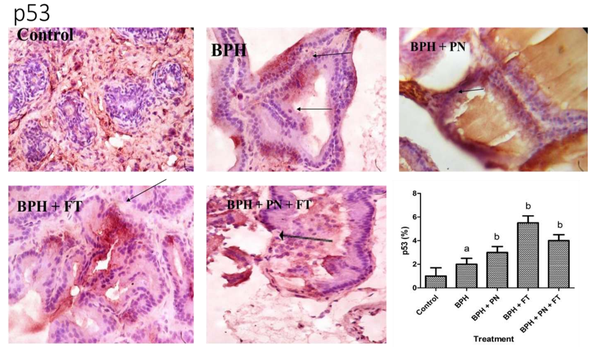

Figure 2. Photomicrograph of immunohistochemistry assay for the expression of p53 in the prostate tissues of BPH rats (x400).

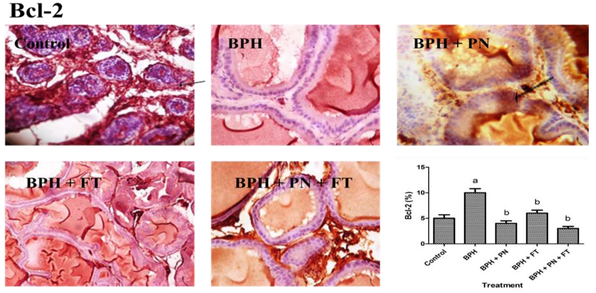

Figure 3. Photomicrograph of immunohistochemistry assay for the expression of Bcl-2 in the prostate tissues of BPH rats (x400).

Conclusions

Punicalagin (PN) mitigates testosterone propionate induced BPH via mechanism involving oxido-inflammatory and apoptotic effects in castrated rats.