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Protective Effect of *Jatropha tanjorensis* Leaves Ethanol Extract on Testosterone-Induced Benign Prostatic Hyperplasia in Albino Rats

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ABSTRACT

This study examined the protective effect of *Jatropha tanjorensis* leaves ethanol extract (JTLEE) on testosterone-induced benign prostatic hyperplasia (BPH) in albino rats. Twenty-five (25) male rats were employed for the study. Five (5) groups of five (5) rats each were created from the animals. Group 1 was neither induced nor treated (normal control), Group 2 was induced but not treated (untreated control), Group 3 was induced and treated with 1 mg/kg b.w. of finasteride (standard control). Groups 4 and 5 were induced and treated with 100 and 200 mg/kg of JTLEE (low and high dose), respectively. The BPH model was developed in the rats (groups 2 to 5) by subcutaneous injection of testosterone propionate (5 mg/kg body weight (b.w)) for 21 days. The JTLEE-treated groups showed a significant ($p < 0.05$) decrease in the weight of the prostate, bladder, and prostate index compared to the untreated control. The PSA level in the extract-treated groups (4.33 ± 0.24) showed a significant ($p < 0.05$) decrease compared to the untreated control (6.75 ± 0.32). Additionally, a dose-dependent significant ($p < 0.05$) reduction in testosterone concentrations of JTLEE-treated rats was observed. There was a significant ($p < 0.05$) elevation of HDL levels of rats treated with varying doses of the JTLEE (3.93 ± 0.16) compared to the untreated group (2.07 ± 0.23). Considering the above results, it could be concluded that JTLEE possesses an anti-BPH effect as observed in the reduction of prostate tissues and significant changes in other prostate indices.

Keywords: Benign prostatic hyperplasia, *Jatropha tanjorensis*, Antioxidants, Prostate.

Introduction

Benign prostatic hyperplasia (BPH), which has a major effect on living standards and financial burden, appears to be one of the most prevalent urological disorders in older men. Extrinsic compression of the prostatic urethra results in clinical symptoms of BPH and impaired voiding.[1] Prolonged incapacity to fully empty the bladder can result in bladder distension, leading to detrusor muscle hypertrophy and instability. The only known factors linked to the advancement of BPH are age and prostate volume, and epidemiologic research indicates that sexual hormones, age, and genetics are the main risk factors for the condition.[2] More recently, prostatic inflammation, cell-signaling issues, detrusor hyperactivity, the metabolic syndrome, and neurological, cardiac, and renal dysfunctions have all been linked to the emergence of BPH.[3]

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Despite the documented involvement of age, hormones, and genetic susceptibility, the molecular and cellular mechanisms involving the stromal and epithelial components of the prostate that contribute to BPH remain unclear.[4] BPH can also arise as a result of erectile dysfunction and hormonal imbalance.[5] Male patients with symptomatic benign prostatic hyperplasia can receive pharmacotherapy with alpha receptor antagonists (ARAs), 5alpha-reductase inhibitors (5αRIs), or a mixture of the two medications.[6] Choosing pharmaceutical medications specifically designed for BPH can support a patient-centered approach. ARAs and 5αRIs are commonly employed as initial therapy, either alone or in combination, to manage BPH when hydration and lifestyle modifications are ineffective.[7] The common weed, *Jatropha tanjorensis* (Euphorbiaceae) is found in disturbed places and roadsides. It is a perennial herb that is a hybrid of *Jatropha curcas* and *Jatropha gossypifolia*, with an intermediate phenotype. Hospital Too Far, Catholic Vegetable, and Ugwu Oyibo are a few of the popular names. Whether consumed raw or as a decoction, the entire part of the plant is utilized in conventional and folk medicine in addition to veterinary treatment. Early studies have shown that the *J. tanjorensis* plant contains high concentrations of antioxidant minerals and vitamins.[8] According to a phytochemical study, *J. tanjorensis* leaves include biologically active substances as anthraquinones, cardiac glycosides, alkaloids, flavonoids, tannins, and saponins.[9] Nigerian soups and tonics frequently use the plant's leaves, which are believed to increase blood volume. Taking *J. tanjorensis* leaves orally alleviates the signs of diabetes in southwest Nigeria.[77] The ethanolic extracts of *J. tanjorensis*, specifically the chloroform, ethylacetate, and aqueous extracts, have been assessed for their potential to prevent diabetes.[10] Novel alternative treatments to address various urological disorders have been proposed, including phytotherapies and the use of nutraceutical agents.[11] The ability of these phytotherapies to positively affect hormonal markers has been overstated in previous evaluations, and the true physiological

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impact is still unknown.[12,13] On the other hand, studies from experimental animal models using phytotherapies for the treatment of prostate illness indicate positive outcomes.[14] Extensive research is required to establish the effectiveness of various phytocompounds in the treatment and management of the BPH condition. Thus, this research aimed to assess the impact of *J. tanjorensis* leaves ethanol extract (JTLEE) on testosterone-induced BPH (TIB) in rats.

Materials and Methods

Plant Collection and Identification
Fresh *Jatropha tanjorensis* leaves were obtained from the Osara community in the Adavi Local Government of Kogi State, Nigeria. Mr. Alfred Ozioko of the Bio-resources Development and Conservation Programme (BDCP) Research Centre in Nsukka, Enugu State, Nigeria, identified the plant using the identification number Intercedd/163517. For extraction, *J. tanjorensis* leaves were cleaned, allowed to air dry at room temperature, and ground into a powder.

Extraction of Plant Materials
The powder (500 g) of plant materials was macerated in 1.5 L of absolute ethanol and allowed to stand for 72 hours at room temperature. Whatman No. 1 filter paper was employed to sift the mixture, and a rotary evaporator was utilized to concentrate the filtrate into an almost solid extract. The semi-solid concentrate was weighed and then stored for later use.

Animals
The study used twenty-five (25) male adult albino rats, and the acute toxicity of the JTLEE was examined utilizing eighteen (18) male albino mice. The animals were purchased from Dr. Wilfred Animal Farm in Nsukka, Enugu State. For seven days before the experiment, the rats were acclimated to the laboratory setting (at room temperature) using a 12-hour light-dark cycle (Animal Holding Room, Department of Biochemistry, Faculty of Science, Confluence University of Science and Technology, Osara, Kogi State, Nigeria). Ethical approval for experimental animal studies was obtained from the Directorate of Research, Innovation, and Development, with approval number, CUSTECH/DRID/AUC/EC/0001. Standard grower's mash rat pellets from Feeds Vendor, Abobo, Adavi Local Government, and water were given to the animals *ad libitum* during the experiment.

Acute Toxicity Study and Phytochemical Screening
The extract's median lethal dosage (LD50), or acute toxicity study, was determined using Lorke's[15] methodology. In this investigation, eighteen (18) albino mice were employed. There were two stages to the test. Phase one involved dividing the animals into three (3) groups of three mice each, and giving them 10, 100, and 1000 mg/kg body weight (b.w.) of the JTLEE, respectively. Phase two involved giving the animals 1600, 2900, and 5000 mg/kg body weight of the extracts. The JTLEE was administered orally. The techniques of Trease and Evans[16] and Harborne[17] were used for the JTLEE qualitative phytochemical screening.

Animal Grouping and BPH Induction
Five (5) groups of five (5) rats each were created from the animals. Group 1 was neither induced nor treated (normal control), Group 2 was induced but not treated (untreated control), and Group 3 was induced and treated with 1 mg/kg b.w. of finasteride (standard control). Groups 4 and 5 were induced and treated with 100 and 200 mg/kg b.w. of JTLEE (low and high dose), respectively. During treatment, just the vehicle was given to Groups 1 and 2. The male rats were given a subcutaneous injection of testosterone propionate (5 mg/kg body weight (b.w.)) for 21 days to generate an experimentally induced BPH model.[18]

BPH Treatment
The following day after the final day of induction, the BPH animals were treated with finasteride and various dosages of JTLEE. The treatment lasted for 21 days. Using gavage, the oral administration was carried out once a day. The vehicle used was Tween 80. The rat's weight was recorded at the start of the experiment and then weekly for six (6) weeks using an electronic weighing balance. Following treatment, the rats were denied food and water for a whole night before being sacrificed. For analysis, tissues (bladder, testis, and prostate gland) were harvested and weighed, and the blood sample was collected.

Organs Weights
The body weight of the animals was recorded on the final day of the test. Following blood collection, the rats were euthanized, prostate tissues, bladder, and testes tissues were excised and weighed individually. The prostate index was calculated as the percentage ratio of the prostate weight to the total body weight.[19]

Biochemical Analysis
According to the manufacturer's instructions, the PSA/testosterone ELISA kit (Monobind Inc., Lake Forest, CA 92630, USA) and the DHT ELISA kit (ALPCO, 26-G Keewaydin Drive, Salem, NH 03079, USA) were used to quantify the concentration of serum PSA, testosterone, and DHT, respectively. Following an overnight period of food and water deprivation, the oxidative stress indicators were determined. For the experiments, prostate tissue was extracted and homogenized. The supernatant of tissue homogenate was used. The Goldberg and Spooner[20] method was used to evaluate the glutathione (GSH) level. The Fridovich[21] method was utilized to assay the activity of superoxide dismutase (SOD). The Aebi[22] method was employed to assay for catalase (CAT) activity. Malondialdehyde (MDA), a byproduct of lipid peroxidation, was measured spectrophotometrically using the Wallin and his team method.[23] The following techniques were used to determine the concentration of the serum lipid profile: triacylglycerols,[24] high-density lipoproteins,[25] low-density lipoproteins,[26] and cholesterol.[27] Utilizing the Reitman and Frankel method,[28] the activities of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were assayed. The Babson and his colleague's method was employed to assay the alkaline phosphatase activity.[29]

Tissue Histology
The prostate tissues were examined histologically using the Drury method.[30] The tissues were preserved in 10% phosphate-buffered formalin for 48 hours. They were then processed using conventional methods for histological analysis. A Motic light microscope was used to examine the slides, and a Motic microscope camera was used to take the photomicrographs.

Statistical Analysis
The data were analyzed using the Statistical Package for the Social Sciences (SPSS, version 23), and the findings were shown as means ± standard deviation (SD). Duncan multiple tests and one-way analysis of variance (ANOVA) were used, and a difference in means was deemed significant when $p < .05$.

Results and Discussion

The present study examined the effects of *Jatropha tanjorensis* leaves ethanol extract (JTLEE) on an animal model of BPH induced by testosterone propionate (TP). Alkaloids, flavonoids, phenols, saponins, tannins, and anthraquinones were discovered in the JTLEE phytochemical screening, but reducing sugars and steroids were absent (Table 1). According to these results, *J. tanjorensis* has a wide variety of bioactive substances that may have therapeutic applications. Alkaloids have an extensive array of pharmacological properties, such as antibacterial, anti-inflammatory, and analgesic actions. Their existence in *J. tanjorensis* supports previous research by Komolafe,[31,] who found that alkaloid-rich fractions of several medicinal plants exhibited strong antibacterial activity. The main class of polyphenolic compounds, flavonoids and phenolic compounds, is well-renowned for their hepatoprotective, anti-inflammatory, and antioxidant properties. Their existence validates *J. tanjorensis* usage in traditional medicine to treat ailments linked to oxidative stress. Oboh *et al*.[32] states that flavonoids greatly increase the leafy veggies' antioxidant capability, such as *J. tanjorensis*, which are frequently consumed in Nigeria.

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Table 1: Phytochemical screening of *Jatropha tanjorensis* leaves ethanol extract

Phytochemical Constituents	Result
Saponins	+
Tannins	+
Phenols	+
Flavonoids	+
Alkaloids	+
Anthraquinones	+
Reducing Sugars	–
Steroids	–

Key: + = Present; - Absent

Additionally, there were saponins, tannins, and anthraquinones, which may be used to treat constipation and other digestive disorders as well as decrease cholesterol. It is interesting to note that the JTLEE included neither reducing sugars nor steroids. The lack of reducing sugars raises the possibility that the extract may have no direct effect on glycemic levels, which may be advantageous for diabetics.

With no death or obvious symptoms of toxicity in the experimental animals over the 24-hour observation period, the JTLEE acute toxicity evaluation, as shown in Table 2, showed a high level of safety. The lack of side effects, such as altered breathing, behavior, or movement, indicates that the JTLEE is safe at the dosages used in acute exposure scenarios. The lack of death or toxicity symptoms at a maximum dosage (often 2000 mg/kg or 5000 mg/kg) suggests that the extract has no acute toxicity effects, according to OECD recommendations for acute oral toxicity testing.[23]

Table 2: Acute toxicity of *Jatropha tanjorensis* leaves ethanol extract

PHASE 1	DOSAGE (mg/kg b.w)	MORTALITY
Group 1	10	0/3
Group 2	100	0/3
Group 3	1000	0/3
PHASE 2		
Group 1	1600	0/3
Group 2	2900	0/3
Group 3	5000	0/3

n= 3.

By suppressing endogenous gonadotropins and spermatogenesis, the subcutaneous injection of TP significantly ($p < 0.05$) decreased testicular weight compared to the normal control (Table 3), confirming the well-established negative feedback effect of exogenous androgens on testicular size.[34] This discovery is consistent with past studies showing that high testosterone can cause testicular atrophy by reducing spermatogenic activity and Leydig cell function.[35] However, when compared to the untreated group, the administration of finasteride and the various doses of JTLEE significantly ($p < 0.05$) increased testes weight, with a significant ($p < 0.05$) dose-dependent effect seen in the groups treated with JTLEE. By blocking testosterone's transformation into dihydrotestosterone (DHT), finasteride, a well-known 5α-reductase inhibitor, reduces androgenic negative feedback and permits a partial restoration of testicular growth.[36] Due to its bioactive components' anti-inflammatory and antioxidant qualities, which have been demonstrated to shield testicular tissue from androgen-induced damage, JTLEE may have restorative effects.[37] Higher concentrations of active phytochemicals appear to strengthen this protective mechanism, as indicated by the dose-dependent improvement.

However, as compared to the control, the untreated group's bladder and prostate weights, as well as the prostate index, increased significantly ($p < 0.05$). According to Miller *et al*.[38] this is in line with the hypertrophic effects of high androgens on androgen-sensitive tissues like the prostate, where increased DHT promotes gland growth and cellular proliferation, simulating the pathophysiology of BPH. Finasteride and the JTLEE were effective in reducing testosterone-induced prostatic hypertrophy since they significantly ($p < 0.05$) decreased bladder and prostate weights as well as the prostate index as compared to the untreated group. According to Traish,[39] this validates finasteride's function in clinical BPH therapy by inhibiting DHT synthesis, but the JTLEE effect might be connected to its modification of androgen receptor activation and inflammatory pathways.[40] Interestingly, there were no significant ($p > 0.05$) variations in bladder and prostate weights or prostate index between the high and low doses of the extract. This suggests that the extract's therapeutic efficacy plateaus after a specific dose threshold. This could help with dosage optimization in pharmacological research in the future.

Table 3: Effect of *Jatropha tanjorensis* leaves ethanol extract on organ weight

Group	Testes (g)	Bladder (g)	Prostate (g)	Prostate Index (mg/g)
1	2.44 ± 0.21^{d}	0.07 ± 0.01^{a}	0.13 ± 0.02^{a}	0.06 ± 0.01a
2	1.26 ± 0.04^{a}	0.14 ± 0.01^{d}	0.52 ± 0.11^{c}	0.32 ± 0.07^{c}
3	1.75 ± 0.17^{b}	0.10 ± 0.01^{b}	0.38 ± 0.08^{b}	0.21 ± 0.02^{b}
4	1.71 ± 0.09^{b}	$0.12 \pm 0.02b^{c}$	0.42 ± 0.02^{b}	0.21 ± 0.02^{b}
5	2.06 ± 0.25^{c}	0.12 ± 0.01^{c}	0.35 ± 0.02^{b}	0.17 ± 0.02^{b}

Notes: Values are expressed as mean ± SD, (n = 5). Values in the same column having different superscripts differ significantly ($p < .05$).

Given that testosterone is known to stimulate prostate growth and PSA generation,[41] the subcutaneous injection of TP significantly ($p < 0.05$) elevated PSA levels in the untreated group when compared to the control (Table 4). In cases of prostatic hyperplasia and cancer, PSA, a marker specific to the prostate, is frequently elevated, suggesting an androgen-dependent process.[42] By blocking the conversion of testosterone to dihydrotestosterone (DHT), a stronger androgen that promotes prostatic growth, finasteride treatment significantly ($p < 0.05$) reduced PSA levels.[43] Likewise, JTLEE therapy led to a dose-dependent, significant ($p < 0.05$) reduction in PSA levels, indicating that it may be a viable alternative treatment option for BPH. The JTLEE dose-dependent effectiveness validates the function of its bioactive constituents in regulating androgenic pathways.

The untreated group's testosterone level was significantly ($p < 0.05$) higher, which is consistent with testosterone-induced BPH. In relation to the group that was not treated, finasteride and the JTLEE-treated groups significantly ($p < 0.05$) decreased testosterone levels, with JTLEE-treated animals showing levels that were comparable to those of normal control. More mechanistic research is warranted since this normalization may suggest that the extract can either accelerate clearance or alter androgen production, or it may even downregulate androgen receptor expression. Curiously, serum testosterone levels show no significant ($p > 0.05$) change when the extract dose was increased to the high dose. This could indicate a threshold effect or

saturation point for the extract's activity. Given that DHT is a key modulator of prostate enlargement, the significant ($p < 0.05$) rise in DHT levels after TP injection validates the increased conversion of testosterone to DHT in the pathophysiology of BPH.[44] Finasteride and the JTLEE both significantly ($p < 0.05$) decreased DHT levels, with the JTLEE-treated groups showing a dose-dependent effect. The restoration of DHT levels to levels comparable to normal controls at higher extract doses provides more evidence for JTLEE's possible 5α-reductase inhibitory action, which is comparable to finasteride but may have fewer adverse effects. This result is consistent with earlier research showing the effectiveness of natural substances in reducing 5α-reductase activity and easing the symptoms of BPH.[45]

Table 4: Effect of *Jatropha tanjorensis* leaves ethanol extract on prostate hormones in testosterone-induced benign prostatic hyperplasia

Group	PSA (ng/ml)	Testosterone (ng/ml)	DHT (ng/ml)
1	3.09 ± 0.17^{a}	6.09 ± 0.72^{a}	2.45 ± 0.41^{a}
2	6.75 ± 0.32^{e}	16.39 ± 1.68^{c}	5.47 ± 0.44^{c}
3	5.35 ± 0.20^{d}	10.01 ± 0.91^{b}	3.35 ± 0.38^{b}
4	4.33 ± 0.24^{b}	7.48 ± 1.25^{a}	3.13 ± 0.59^{b}
5	4.80 ± 0.23^{c}	6.10 ± 0.55^{a}	2.43 ± 0.56^{a}

Notes: Values are expressed as mean ± SD, (n = 5). Values in the same column having different superscripts differ significantly ($p < .05$). PSA = prostatic specific antigen; DHT = Dihydrotestosterone.

Compared to the normal control group, the subcutaneous injection of TP significantly ($p < 0.05$) decreased high-density lipoprotein (HDL) and significantly ($p < 0.05$) raised serum levels of triglycerides (TAG), total cholesterol (TC), and low-density lipoprotein (LDL) (Table 5). The literature showing that exogenous androgen administration can dysregulate lipid metabolism is in line with the observed rise in TAG, TC, and LDL after TP injection.[46] According to Zhao *et al*., [47] testosterone may promote atherogenic lipid profiles via influencing hepatic lipid production and clearance mechanisms. Since elevated LDL and TAG levels are known risk factors for cardiovascular disease (CVD), especially when combined with hormonal disorders, such dyslipidemic changes are crucial.[48] Finasteride and varying doses of the JTLEE, however, significantly ($p < 0.05$) lowered the increases in TAG, TC, and LDL levels brought on by TP in rats with BPH. Although finasteride, a 5α-reductase inhibitor, is well recognized for its effects on prostate tissue, new research indicates that it may also have positive benefits on systemic lipid profiles, perhaps through enhancing insulin sensitivity and modifying androgen metabolism.[49] Its bioactive phytochemicals, which may have cholesterol-lowering, anti-inflammatory, and antioxidant qualities, may be the reason for JTLEE's effectiveness in lowering lipid levels. In animal models of dyslipidemia, several plant-derived substances have shown comparable benefits, frequently via mechanisms involving the augmentation of reverse cholesterol transport and suppression of hepatic lipogenesis.[50,51]

On the other hand, finasteride therapy did not significantly ($p > 0.05$) reverse the TP-induced rats' HDL level decline, suggesting that finasteride alone may not have a major impact on HDL metabolism in these experimental settings. This is consistent with certain clinical findings that indicated 5α-reductase inhibitors did not affect HDL.[52] Serum HDL levels, however, were considerably ($p < 0.05$) raised by JTLEE therapy in a dose-dependent way, with the highest dose producing the most noticeable increase. According to Rohatgi,[53,] this discovery highlights the JTLEE's capacity to improve HDL-mediated reverse cholesterol transfer, a defense mechanism against atherosclerosis. It has been demonstrated that phytochemicals, including flavonoids and saponins, which are frequently present in medicinal plants, increase ApoA1 and encourage HDL synthesis.[54]

In comparison to normal controls, the subcutaneous injection of TP significantly ($p < 0.05$) reduced the amounts of catalase (CAT), glutathione (GSH), and superoxide dismutase (SOD), which are antioxidant enzymes, in the homogenate of untreated rats (Table 6). This decrease suggests increased testosterone-induced oxidative stress, which is in keeping with earlier findings that excess testosterone might weaken antioxidant defenses and encourage oxidative tissue damage.[55,56] Finasteride and JTLEE administration, however, significantly ($p < 0.05$) raised GSH levels, with the JTLEE demonstrating a dose-dependent impact. These results align with earlier research showing that finasteride and phytochemicals possess antioxidative potential in enhancing systemic antioxidant status in models of BPH.[57,58] Further evidence of the presence of active antioxidant components that can scavenge free radicals and strengthen endogenous defense mechanisms comes from the JTLEE dose-dependent rise in GSH.[59]

The high-dose JTLEE administration resulted in a significant ($p < 0.05$) increase in SOD activity, whereas finasteride and low-dose extract treatment did not significantly ($p > 0.05$) change relative to the group that was not treated. This could suggest that to increase the expression or activity of SOD, a crucial enzyme that dismutates superoxide radicals, greater amounts of bioactive substances may be required.[60] The notion that proper dosing is necessary for antioxidant enzyme regulation was further supported by similar findings regarding CAT activity, which showed that only finasteride and high-dose extract significantly ($p < 0.05$) increased CAT levels.[61] Conversely, the injection of TP led to a significant ($p < 0.05$) rise in malondialdehyde (MDA), a biomarker of lipid peroxidation, indicating increased oxidative damage in animals that were not treated. This finding is consistent with multiple studies that show cellular damage and elevated lipid peroxidation in BPH models are caused by androgen-induced oxidative stress.[62,63] The antioxidative and protective functions of finasteride and the plant extract against lipid membrane damage were highlighted by the significant ($p < 0.05$) reduction of MDA levels that both showed at the high dose.

Hepatic stress or moderate liver stress is indicated by the significant ($p < 0.05$) rise of serum alkaline phosphatase (ALP), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) activity after subcutaneous injection of TP in untreated rats (Table 7). The rise of these enzymes indicates hepatic injury or increased membrane permeability, which are well-established indicators of liver function and cellular integrity.[64,65] The rise in these enzymes brought on by testosterone is consistent with other research that found androgen excess to be a contributing factor to oxidative damage and liver dysfunction.[66,67] A hepatoprotective effect was observed because the administration of finasteride and the JTLEE significantly ($p < 0.05$) reduced the elevations in serum ALP, AST, and ALT activity in treated rats. The potential of bioactive constituents in restoring liver enzyme levels and lowering hepatic oxidative stress is highlighted by the fact that this reduction is dose-dependent in the JTLEE-treated groups. Phytochemicals have been shown to have comparable protective effects against changes in hepatic enzymes induced by testosterone.[68,69] Increased hepatic stability and enzyme regulation may be shown by the attenuation of AST activity to levels below even the typical control levels at the high JTLEE dose.

Remarkably, at the high dose, AST activity was significantly ($p < 0.05$) decreased compared to normal control levels, but ALP and ALT activities did not deviate significantly ($p > 0.05$) from the normal control, indicating that these enzymes were selectively modulated or had varying sensitivity to the JTLEE bioactive components. This finding is in line with studies suggesting ALT and ALP may represent distinct facets of hepatic or biliary function, whereas AST is more susceptible to oxidative stress and hepatocellular alterations.[70,71]

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Table 5: Effect of *Jatropha tanjorensis* leaves ethanol extract on lipid profile in testosterone-induced benign prostatic hyperplasia

Group	TAG (mmol/L)	TC (mmol/L)	LDL (mmol/L)	HDL (mmol/L)
1	2.18 ± 0.29^{a}	3.90 ± 0.32^{a}	1.64 ± 0.19^{a}	4.67 ± 0.46^{d}
2	3.65 ± 0.31^{d}	7.15 ± 0.73^{c}	3.72 ± 0.31^{c}	2.07 ± 0.23^{a}
3	3.05 ± 0.133^{c}	4.88 ± 0.38^{b}	2.70 ± 0.29^{b}	2.38 ± 0.25^{a}
4	2.59 ± 0.27^{b}	3.89 ± 0.24^{a}	2.66 ± 0.23^{b}	3.20 ± 0.16^{b}
5	2.00 ± 0.19^{a}	3.43 ± 0.43^{a}	1.83 ± 0.31^{a}	3.93 ± 0.16^{c}

Notes: Values are expressed as mean ± SD, (n = 5). Values in the same column having different superscripts differ significantly ($p < .05$). TAG = Triacylglyceride; TC = Total Cholesterol; LDL = low density lipoprotein; HDL = high density lipoprotein.

Table 6: Effect of *Jatropha tanjorensis* leaves ethanol extract on antioxidant activities in testosterone-induced benign prostatic hyperplasia

Group	GSH (mg/dl)	SOD (U/mg)	CAT (U/mg)	MDA (mg/dl)
1	3.09 ± 0.18^{c}	14.63 ± 2.10^{b}	5.44 ± 0.32^{d}	1.71 ± 0.13^{a}
2	2.10 ± 0.14^{a}	11.03 ± 1.79^{a}	2.99 ± 0.14^{a}	2.86 ± 0.27^{c}
3	2.70 ± 0.12^{b}	13.43 ± 2.69^{ab}	3.38 ± 0.19^{b}	2.30 ± 0.12^{b}
4	2.59 ± 0.12^{b}	13.02 ± 2.00^{ab}	3.13 ± 10^{ab}	2.16 ± 0.11^{b}
5	3.04 ± 0.12^{c}	14.48 ± 2.79^{b}	3.94 ± 0.19^{c}	1.70 ± 0.11^{a}

Notes: Values are expressed as mean ± SD, (n = 5). Values in the same column having different superscripts differ significantly ($p < .05$). GSH = Glutathione; SOD = Superoxide dismutase; CAT = Catalase; MDA = Malondialdehyde.

Prostate tissue histopathological analysis is still a vital method for determining the gland's structural and functional integrity in both healthy and diseased circumstances. The photomicrographs in this investigation showed clear morphological variations between the control, untreated, standard, and JTLEE-treated groups, indicating different levels of prostatic injury or protection (Figure 1). Plate A, the control prostate tissue, showed a well-preserved architecture with fibromuscular stroma, basal nuclear polarity, and convoluted glands bordered with a typical double-layered epithelium. According to traditional histological descriptions of rodent prostates, these characteristics are suggestive of a healthy prostate gland.[72]

Table 7: Effect of *Jatropha tanjorensis* leaves ethanol extract on liver function enzyme activities in testosterone-induced benign prostatic hyperplasia

Group	ALP (U/L)	AST (U/L)	ALT (U/L)
1	10.69 ± 0.91^{a}	22.55 ± 2.26^{b}	13.91 ± 1.23^{a}
2	15.40 ± 1.25^{c}	38.81 ± 1.50^{d}	30.28 ±2.63^{c}
3	12.31 ± 0.96^{b}	28.55 ± 2.17^{c}	21.65 ± 2.42^{b}
4	10.53 ± 1.29^{a}	21.54 ± 2.08^{b}	19.52 ± 1.34^{b}
5	9.37 ± 1.02^{a}	16.91 ± 1.59^{a}	14.30 ± 0.84^{a}

Notes: Values are expressed as mean ± SD, (n = 5). Values in the same column having different superscripts differ significantly ($p < .05$). ALP = Alkaline phosphatase; AST = Aspartate aminotransferase; ALT = Alanine aminotransferase.

On the other hand, the untreated group (Plate B) showed notable pathological changes, such as enlarged acini swollen with secretory materials, architectural disruption, and severe blood congestion within the fibromuscular stroma. In line with the consequences of hormonal imbalance or chemical stimulation of prostatic damage, our findings point to an inflammatory or hyperplastic response that may resemble a condition similar to BPH or an induced prostate trauma.[73] With better intra-acinar papillary infoldings and a restoration to normal acinar structure, the finasteride-treated group (Plate C) showed a partial recovery of prostatic architecture. Back-to-back micro acini show glandular epithelial reconfiguration and may be a sign of the standard agent's anti-inflammatory or regenerative properties.

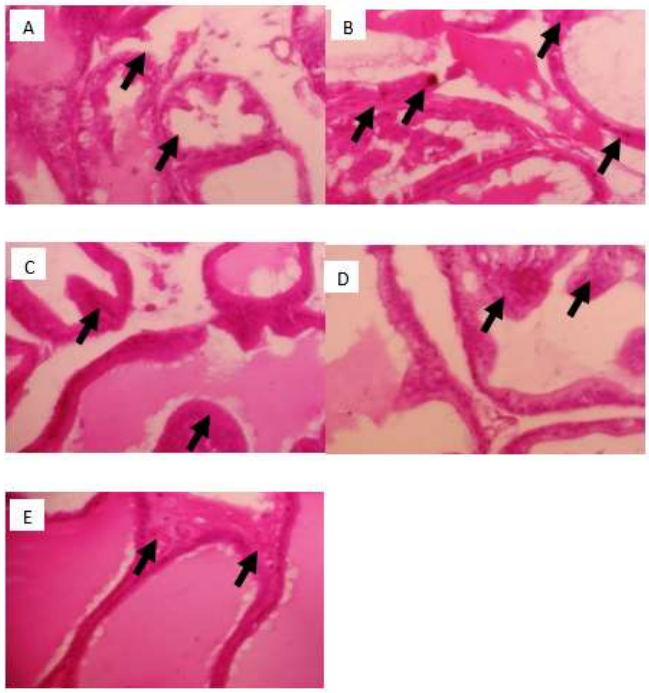

Figure 1: Prostate histology of experimental rats.
A) Normal Control: Photomicrograph of prostate tissue shows normal histological appearances composed of normal convolutions, usually lined by two layers of epithelium with clear basal polarity of the nuclei and fibro-muscular stroma; **B) Untreated Control:** The untreated prostate tissue shows severe multiple blood congestions in the fibromuscular stroma. Further degeneration such as enlarged acini, mostly distended with secretory materials was observed; **C) Standard Control:** Prostate tissue exhibited back–to–back micro acini with improved intra acini papillary convolution; **D) Low Dose:** Prostate tissue shows ectasia mainly characterized by acini, also distended with secretory materials, compression of surrounding unaffected acini with

dilation appearing cystic; **E) High Dose:** Prostate tissue shows extensive enlarged acini with secretory materials in the lumen. Infoldings with fused cuboidal epithelium lining were also observed; H&E stain (4X & 10X).

Histological improvements were dose-dependent when JTLEE was administered at 100 mg/kg (Plate D) and 200 mg/kg (Plate E) body weight. The tissue continued to show signs of acinar ectasia at 100 mg/kg, including distention, infoldings, and fused cuboidal epithelium, which may indicate a partial reversal of inflammation or hyperplasia. The continued presence of squeezed neighboring acini and cystic dilatation, however, indicates that this dosage was insufficient for complete histological healing. A more noticeable glandular restoration was indicated by the prostate tissue's widely expanded acini with secretory components in the lumen at 200 mg/kg. The preserved fibromuscular support and glandular dilatation without appreciable epithelial damage point to a protective or anti-hyperplastic action of JTLEE at this higher dosage. These findings are in line with research showing that Justicia species and related medicinal plants have anti-inflammatory, antioxidant, and androgen-modulating qualities.[74,75] Changes in secretory activity and prostatic aging or dysfunction may also be indicated by the presence of corpora amylacea in control tissues and their absence or disruption in untreated and treated groups.[76] The idea that JTLEE may stabilize glandular function is supported by the restored secretion and epithelial polarity in treated groups. The combined histological results indicate that JTLEE may have an anti-inflammatory or anti-proliferative effect on prostatic histoarchitecture in a dose-dependent manner.

Conclusion

This study provides strong evidence supporting the possible health advantages of *J. tanjorensis* extract in managing testosterone-induced BPH in rats. The findings of this research provide credence to the JTLEE potential as a treatment for BPH, as it significantly reduces prostate weight, improves antioxidant biomarkers, HDL levels, modified androgen metabolism, and subsequent indicators like PSA, testosterone, and DHT concentrations in testosterone-induced BPH rats. The pharmacological significance of JTLEE is supported by the observed dose-dependent effects on DHT levels, which call for additional research to identify the active ingredients and clarify the exact mechanisms of action.

Conflict of Interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the works presented in this article are original and that any liability for claims relating to the content of this article will be borne by them.

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