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RESEARCH ARTICLE

ELUSINE CORACANA LEAF EXTRACT MITIGATES DYSLIPIDEMIA AND MODULATES INFLAMMATION BIOMARKERS IN TESTOSTERONE PROPIONATE - INDUCED BENIGN PROSTATIC HYPERPLASIA IN RATS

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Abstract

Background: Benign Prostatic Hyperplasia (BPH) is an age-related condition characterized by prostate enlargement, lower urinary tract symptoms (LUTS), and persistent inflammation. Although conventional treatments such as α1-adrenergic blockers and 5α-reductase inhibitors (e.g., finasteride) can relieve symptoms, their use is frequently associated with undesirable side effects. This study evaluated the effect of ethanol extract of *Eleusine coracana* leaves on serum lipid profile and inflammatory cytokines in a testosterone propionate (TP)-induced BPH rat model.
Methods: Thirty male Wistar rats were randomly assigned to six groups: normal control, TP-induced BPH control, finasteride-treated group, and three groups treated with *E. coracana* extract at doses of 100, 200, and 400 mg/kg. Serum lipid parameters (triglycerides, total cholesterol, HDL, and LDL) and inflammatory cytokines (IL-6 and TNF-α) were assessed, together with prostatic levels of IL-6 and TNF-α.
Results: The results revealed that TP-induced BPH caused significant dyslipidemia (increased TG, TC, LDL, and decreased HDL) and elevated systemic and prostatic cytokines compared to controls. Treatment with *E. coracana* extract significantly improved lipid profiles and reduced pro-inflammatory cytokine levels in a dose-dependent manner, with effects comparable to finasteride. Notably, the 400 mg/kg extract normalized HDL and prostatic IL-6 levels, indicating strong anti-inflammatory and lipid-modulating activity.
Conclusion: These findings suggest that *E. coracana* leaf extract may serve as a promising adjunct or alternative therapy for BPH, particularly in patients with concurrent metabolic risk factors.
Keywords: Benign prostatic hyperplasia, *Eleusine coracana*, inflammatory cytokines, lipid profile, phytotherapeutics.

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a prevalent urological disorder among men aged 50 years and older, characterized by non-malignant enlargement of the prostate gland[1]. This enlargement commonly results in lower urinary tract symptoms (LUTS), including urinary frequency, retention, and nocturia, which substantially impair quality of life[2]. According to the Global Burden of Disease report, the number of BPH cases increased by 105.70% between 1990 and 2019, with the highest incidence occurring in individuals aged 65–69 years[3]. Epidemiological evidence further indicates that approximately 18.7% of men older than 60 years are affected, with prevalence rising by about 5.44% per decade of age[4].

Emerging evidence links metabolic syndrome with an increased risk of BPH, particularly among patients with hypertension, diabetes mellitus, and dyslipidemia, although the underlying mechanisms remain incompletely understood[5,6]. Chronic inflammation and hormonal alterations are recognized contributors to prostatic enlargement[7]. Persistent inflammation promotes lymphocyte infiltration into prostatic tissue, leading to tissue damage and playing a critical role in the onset and progression of BPH[8]. Several investigations have demonstrated a strong association between prostatic inflammation and gland enlargement[9,10]. In particular, cytokines and chemokines are implicated in prostatic tissue proliferation, with increased expression of interferon-γ, IL-17, and IL-8 reported in epithelial cells[9,10]. Chronic prostatic

inflammation is therefore widely recognized as a key driver in the pathogenesis of lower urinary tract diseases and an exacerbating factor in BPH[11,12].

Dyslipidemia, inflammation, and BPH are closely interconnected. Dyslipidemia may induce a persistent systemic inflammatory state that facilitates immune cell infiltration into the prostate, tissue remodeling, hyperplasia, and benign prostatic enlargement, thereby worsening LUTS and promoting clinical BPH. Expansion of adipose tissue can lead to hypoxia and cellular necrosis, which attract macrophages and other immune cells and increase cytokine production and reactive oxygen species (ROS) generation[13,14]. This sustained inflammatory milieu may further enhance immune cell infiltration into the prostate. Additionally, pro-inflammatory cytokines released within the prostatic stroma can stimulate stromal cell proliferation, contributing to prostate enlargement and increased LUTS severity[14].

Current pharmacological treatments for BPH, including α-blockers and 5-α-reductase inhibitors, are effective but are frequently associated with adverse effects such as dizziness, hypotension, and sexual dysfunction[15,16]. Moreover, these therapies may not adequately address associated dyslipidemia and chronic inflammation, highlighting the need for alternative therapeutic approaches, particularly from natural sources that may offer efficacy with fewer side effects. *E. coracana* (finger millet) is a staple cereal cultivated widely in tropical and subtropical regions, including Africa and South Asia. Beyond its nutritional value, it possesses notable medicinal potential due to its rich phytochemical profile, including phenolic acids, flavornoids, and alkaloids[17-19]. These bioactive compounds exhibit antioxidant, anti-inflammatory, and antimicrobial properties, suggesting potential benefits in managing chronic disorders such as BPH and related comorbidities[20-23]. However, limited studies have investigated the role of *E. coracana* in alleviating dyslipidemia and inflammation associated with BPH. Therefore, this study aimed to evaluate the effects of ethanol extract of *E. coracana* leaves on dyslipidemia and inflammatory markers in BPH-induced rats.

MATERIALS AND METHODS

Plant collection and identification

Fresh leaves of *E. coracana* were harvested in May 2023 from a local farm in Umuika Autonomous Community, Isiala Ngwa South L.G.A., Abia State, Nigeria. The plant material was authenticated by Dr. G. Omosun, Department of Plant Science and Biotechnology, Michael Okpara University of Agriculture, Umudike (MOUAU). A voucher specimen (DCAEC0282023) was deposited in the departmental herbarium and assigned accession number MOUAU/PSB/HERB/2023/028.

Preparation of ethanol extract

The collected leaves were thoroughly washed with tap water and air-dried under shade at room temperature for seven days. The dried material was pulverized using a mechanical grinder. Five hundred grams (500 g) of the powdered leaves were extracted by maceration in 2.5 L of 70% ethanol for 72 hours with intermittent agitation. The extract was filtered through Whatman No. 1 filter paper and concentrated under reduced pressure at 45°C using a rotary vacuum evaporator (BUCHI Rotavapor R-200, Switzerland) to obtain the crude ethanol extract (7.20% yield).

Animals and experimental design

Thirty-six adult male albino rats (150–250 g) were obtained from the Animal Farm, Department of Veterinary Medicine, MOUAU. Animals were housed in the Department of Biochemistry animal facility under controlled conditions (25–28°C; 12 hour light/dark cycle) with free access to feed and water. After a 7 day acclimatization period, all procedures were conducted in accordance with the National Research Council (2011) guidelines and approved by the MOUAU Animal Use Ethical Committee (Approval No.: MOUAU/EC/VM/8/2023).

Benign prostatic hyperplasia was induced by subcutaneous administration of testosterone propionate (TP, 5 mg/kg) dissolved in olive oil for 28 days. Rats were randomly assigned into six groups (n = 6):

- **Group I:** Normal control (olive oil only)
- **Group II:** BPH control (TP only)
- **Group III:** BPH + Finasteride (1 mg/kg, p.o.)
- **Group IV:** BPH + *E. coracana* extract (100 mg/kg, p.o.)
- **Group V:** BPH + *E. coracana* extract (200 mg/kg, p.o.)
- **Group VI:** BPH + *E. coracana* extract (400 mg/kg, p.o.)

Oral treatments commenced 24 hours after the first TP injection and continued for 28 days. At the end of the treatment period, animals were fasted overnight, anesthetized, and blood samples were collected via cardiac puncture. Serum was separated by centrifugation (6817×g, 10 min) and stored at −80°C until analysis.

Determination of serum lipid profile

Serum high-density lipoprotein (HDL), total cholesterol (TC), and triacylglycerol (TAG) were measured spectrophotometrically. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation as previously described[24,25]:

$$\mathrm{LDL-C} = TC - HDL - \frac{TAG}{5}$$

Tissue homogenate preparation

Prostate tissues were homogenized under ice-cold conditions in a tenfold volume of 100 mM potassium phosphate buffer containing 1.2 mM EDTA (pH 6.4). The homogenate was centrifuged at 1500×g for 25 minutes at 4°C, and the supernatant was used for cytokine analysis.

Determination of inflammatory cytokines

Serum and prostatic levels of TNF-α (E-EL-R2856) and IL-6 (E-EL-R0015) were quantified using sandwich ELISA kits. The serum or tissue samples (100 µL) were incubated with biotin-conjugated detection antibodies, followed by streptavidin–horseradish peroxidase. Absorbance was read at 450 nm, and cytokine concentrations were calculated using standard calibration curves.

Statistical analysis

Data were expressed as mean±standard error of the mean (SEM) and analyzed using GraphPad Prism version 8.0 (GraphPad Software LLC, Boston, MA, USA). Group differences were assessed by one-way ANOVA followed by Tukey's post hoc test, with $p<0.05$ considered statistically significant.

RESULTS

Effect of *E. coracana* on Serum lipid profile in BPH rats

The effect of *E. coracana* on serum lipid profile in BPH induced rats is shown in Figure 1 to Figure 4. The TG levels showed significant variations among the groups ($p<0.05$). Group 2 (BPH control) had the highest TG level, which was significantly higher than all other groups, indicating that testosterone propionate-induced BPH elevated TG levels. Group 1 (normal control) had the lowest TG level, which differed significantly from the BPH control group. Treatment with *E. coracana* extracts at 100, 200, and 400 mg/kg (Groups 4–6) significantly reduced TG levels compared to the BPH control. The finasteride-treated group (Group 3) also reduced TG levels comparable to the 200 and 400 mg/kg *E. coracana* extract groups (Figure 1).

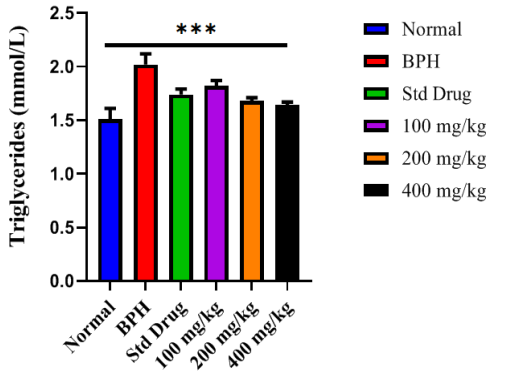

Figure 1: Effect of ethanol extract of *E. coracana* leaves on serum triglycerides level (n=6; $p<0.05$).

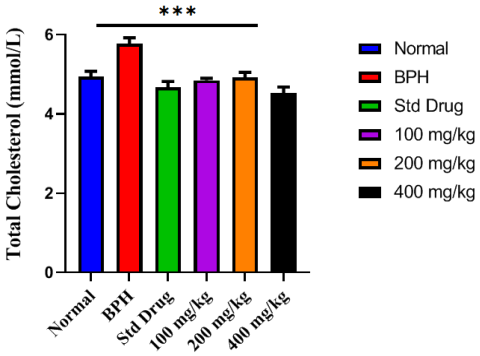

Figure 2: Effect of ethanol extract of *E. coracana* leaves on serum total cholesterol level (n=6; $p<0.05$).

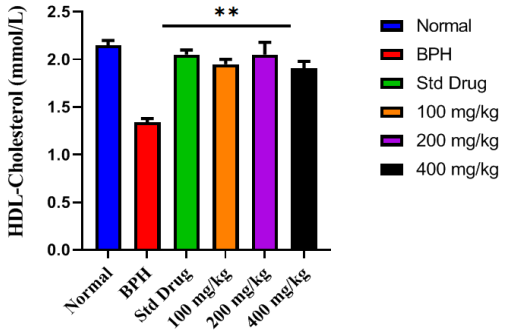

Figure 3: Effect of ethanol extract of *E. coracana* leaves on serum HDL-cholesterol level (n=6; $p<0.05$).

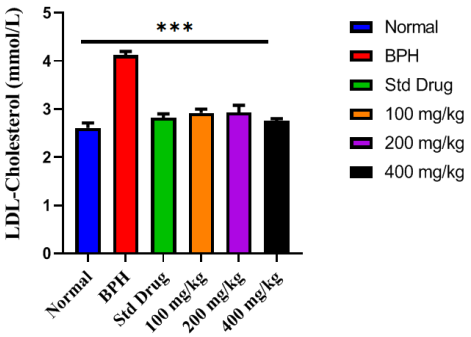

Figure 4: Effect of ethanol extract of *E. coracana* leaves on serum LDL-cholesterol level (n=6; $p<0.05$).

There was a significant increase in TC levels in Group 2 compared to the normal control confirming that BPH is associated with hypercholesterolemia. Finasteride and all *E. coracana* treated groups significantly reduced TC levels compared to the BPH control. Notably, the 400 mg/kg extract group exhibited the greatest reduction, which was not significantly different from the finasteride group, suggesting a comparable cholesterol-lowering effect (Figure 2). HDL levels were markedly reduced in Group 2, showing significantly lower values than all other groups ($p<0.05$). Administration of *E. coracana* extract produced a dose-dependent rise in HDL, with the 200 and 400 mg/kg groups exhibiting significant improvement relative to the BPH control. The highest HDL concentrations were observed in the finasteride-treated group and the 200 mg/kg extract group (Figure 3). LDL levels were significantly increased in the BPH control compared with the normal control. Treatment with finasteride and *E. coracana* extract at all tested doses significantly lowered LDL relative to Group 2, with the 400 mg/kg extract group showing LDL values closest to those of the normal control (Figure 4).

Effect of *E. coracana* on serum inflammatory cytokines (IL-6 and TNF-α)

The IL-6 levels were significantly elevated in the BPH control group compared with the normal control, confirming the presence of BPH-associated inflammation. Treatment with finasteride and *E. coracana* extract significantly reduced IL-6 concentrations, with the 400 mg/kg dose producing the most pronounced anti-inflammatory effect, comparable to finasteride. Similarly, TNF-α levels were significantly increased in the BPH control relative to the normal control. Administration of finasteride and *E. coracana* extracts significantly lowered TNF-α levels compared with the

BPH control, with no significant differences observed among the treated groups, indicating the anti-inflammatory potential of *E. coracana*.

Effect of *E. coracana* on prostatic cytokines

The BPH control group showed elevated prostatic IL-6 levels compared to the normal control. Treatment with *E. coracana* extract normalized P_IL-6 levels, which were statistically similar to the normal control and finasteride, indicating potent suppression of prostatic inflammation. Also, the P_TNF level of the treatment groups (finasteride and *E. coracana* extract at all doses) significantly reduced P_TNF levels.

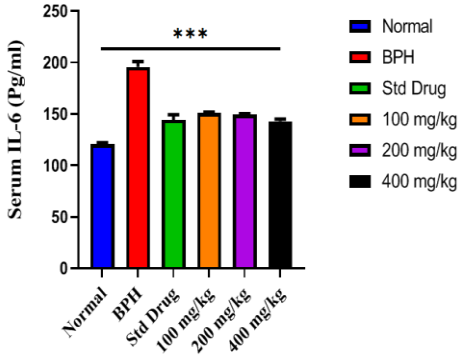

Figure 5: Effect of ethanol extract of *E. coracana* leaves on serum IL-6 levels (n=6; $p<0.05$).

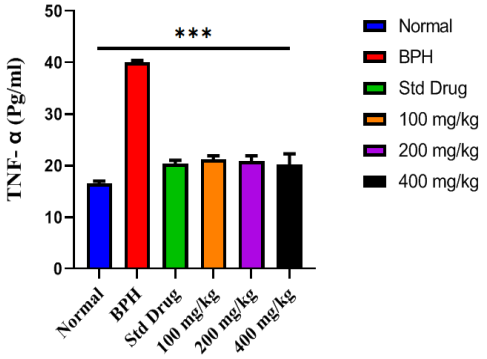

Figure 6: Effect of ethanol extract of *E. coracana* leaves on serum TNF-α levels (n=6; $p<0.05$).

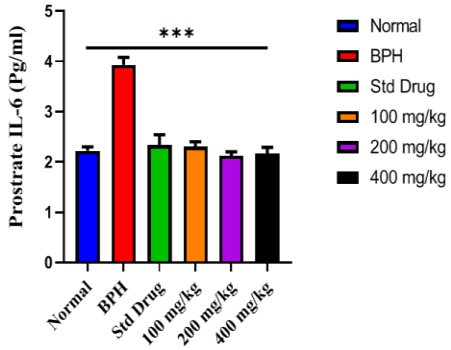

Figure 7: Effect of ethanol extract of *E. coracana* leaves on prostate tissue IL-6 levels (n=6; $p<0.05$).

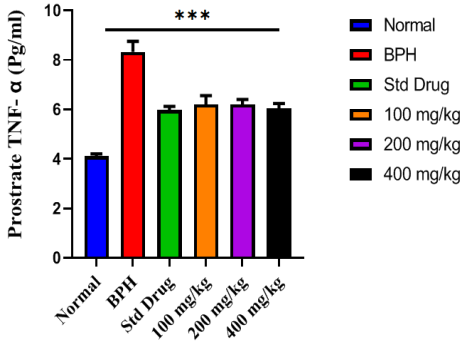

Figure 8: Effect of ethanol extract of *E. coracana* leaves on prostate tissue TNF-α levels (n=6; $p<0.05$).

DISCUSSION

Benign prostatic hyperplasia (BPH) is a highly prevalent, age-related disorder defined histologically by the non-malignant proliferation of prostatic stromal and epithelial cells and clinically by prostate enlargement that may result in bladder outlet obstruction and lower urinary tract symptoms (LUTS). Large epidemiological and autopsy studies indicate that histological BPH occurs in approximately 50–60% of men in their 60s and increases to about 80% or more in men aged 70–80 years and above, highlighting its near universality in aging males[26]. The global burden of BPH including incidence, prevalence, and disability-adjusted life years continues to rise as populations' age[20]. Current management strategies range from watchful waiting and pharmacotherapy (α1-adrenergic blockers, 5α-reductase inhibitors such as finasteride and dutasteride, and phosphodiesterase-5 inhibitors) to minimally invasive procedures and surgery. However, drug-related sexual and metabolic adverse effects, along with procedural risks, have stimulated growing interest in safer, multi-targeted phytotherapeutic interventions[27-29].

Testosterone propionate is widely used to induce experimental BPH, and its administration has been shown to promote prostatic inflammation. Increasing evidence links BPH progression with systemic metabolic disturbances, including dyslipidemia, insulin resistance, obesity, and metabolic syndrome as well as low-grade chronic inflammation within the prostate[30-32]. Because medicinal plants are rich in polyphenols that modulate lipid metabolism, oxidative stress, and inflammatory signaling, they are promising candidates for integrative BPH management. Finger millet (*E. coracana*), widely consumed across Africa and Asia, is notable for its high dietary fiber content (approximately 15-20%), abundant phenolic acids, flavonoids, and tannins, and documented hypolipi-demic, and anti-inflammatory properties[33-37]. Building on these attributes, the present study evaluated the effects of ethanol leaf extract of *E. coracana* on lipid profile and inflammatory biomarkers in a testosterone propionate-induced BPH rat model.

Lipids play a critical role in prostatic cellular proliferation, contractility, and gland enlargement, thereby constituting a potential risk factor for both BPH and prostate cancer[38,39]. As the prostate is a cholesterol-rich tissue, elevated serum cholesterol may enhance cholesterol accumulation within cell membranes, promoting the formation of enlarged lipid rafts associated with pro-carcinogenic signaling pathways

that contribute to prostate enlargement[40,41]. In this study, testosterone propionate–induced BPH was associated with marked dyslipidemia, evidenced by significant increases in triglycerides, total cholesterol, and LDL-cholesterol, alongside reduced HDL-cholesterol, in the BPH control group, consistent with previous reports[32,42]. Oral administration of *E. coracana* extract at doses of 100, 200, and 400 mg/kg significantly improved lipid parameters. Notably, the 400 mg/kg dose reduced total cholesterol and LDL-cholesterol to levels approaching those of finasteride and closest to the normal control, suggesting that bioactive constituents of the extract can counter androgen-associated dyslipidemia.

These findings align with earlier studies on finger millet and related cereals. *E. coracana* leaf extract has demonstrated antihyperlipidemic activity in alloxan-induced hyperglycemic rats by reducing total cholesterol, triglycerides, and LDL while increasing HDL[34]. Whole-grain finger millet is rich in soluble and insoluble fiber capable of binding bile acids and modulating cholesterol absorption, while its poly-phenol rich seed coat fractions have shown hypo-cholesterolemic effects in animal feeding studies[33,36]. Polyphenols may further regulate hepatic lipid metabolism through activation of AMPK, suppression of HMG-CoA reductase activity, and enhancement of antioxidant defenses[35]. Serum and prostatic levels of IL-6 and TNF-α were markedly elevated in testo-sterone propionate–induced BPH rats. Numerous histological and translational studies have reported increased expression of pro-inflammatory cytokines including IL-6, TNF-α, IL-8, and IL-17 family members in BPH tissues and circulation, linking inflammatory burden with prostate enlargement, symptom severity, and disease progression[43]. Given that 5α-reductase inhibitors primarily reduce intra-prostatic dihydrotestosterone rather than directly targe-ting cytokine activity[28], the comparable anti-inflamm-atory effects suggest that the extract may engage additional immunomodulatory pathways. These may include antioxidant mediated suppression of NF-κB signaling, inhibition of pro-inflammatory enzymes such as 5-lipoxygenase and xanthine oxidase, or modulation of gut-derived metabolites influencing systemic inflammation[35]. The observed reductions in IL-6 and TNF-α, therefore, support the anti-inflammatory potential of the extract.

Limitations of the study

The study focused mainly on biochemical and inflammatory biomarkers without a detailed investigation of the underlying molecular pathways, such as gene and protein expression related to lipid metabolism and inflammation in BPH. Therefore, further studies are required in this area.

CONCLUSION

The present study assessed the lipid profile and selected inflammatory biomarkers, and the findings showed that *E. coracana* leaf extract significantly reduced the levels of TC, triacylglycerol, LDL, and VLDL, as well as the IL-6 and TNF-α in treated rats. Since lipids are known to promote contractility, cellular proliferation, and overall enlargement of the prostate gland, it is reasonable to suggest that lowering both inflammatory mediators and lipid levels could contribute significantly to the management of benign prostatic hyperplasia.

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AUTHOR'S CONTRIBUTION

Duru CA: conceptualization, investigation, review. **Ajah O:** writing original draft, methodology, formal analysis, data curation, conceptualization, editing. **Nnaoma IE:** review and editing, methodology. **Joseph RC:** formal analysis, data curation, writing. All authors read and approved the final manuscript.

DATA AVAILABILITY

The associated author can provide the empirical data used to support the study's conclusions upon request.

CONFLICT OF INTEREST

There are no conflicts of interest in regard to this project.

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