Content available at: https://www.ipinnovative.com/open-access-journals

International Journal of Clinical Biochemistry and Research

Journal homepage: www.ijcbr.in

Original Research Article

Therapeutic roles of Aframomum melegueta on testosterone propionate induced benign prostrate hyperplasia in wistar rats

Esther Udo Umoh[1]*, Emmauel Olorufemi Ibukun[1], Olaide Bose Buraimoh[1], Adewale Ayodele Olowogbade[1], Godspower Oyubu[1]

[1]Federal University of Technology, Akure, Nigeria.

Abstract

Introduction: Herbs play a vital role in our lives, particularly in the realm of medicine. Traditional medicine, in fact, relies heavily on medicinal plants, with approximately 3.3 billion people in developing countries using them regularly (Davidson, 2010).
Materials and Methods: Collection of Plant material, In-vivo Experimental Design/Animal Grouping of Testosterone Propionate Induction and Treatment using Aframomum meleguata
Results: The damage to the prostate region were reversed on treatment with EAM. However, posterior karyolysis was observed in the hippocampus of rats treated with (2900 mg/kg) EAM alone.
Conclusion: The result obtained in the present study revealed that Aframomum meluegata leaf extract ameliorated testosterone-propionate–induced benign prostatic hyperplasia through its antioxidative, anti-inflammatory and anti-apoptotic effect.

Keywords: Benign prostatic hyperplasia (BPH), Center for Research and Development (CERAD), Aframomum meluegata

Received: 13-12-2024; **Accepted:** 13-02-2025; **Available Online**:22-03-2025

1. Introduction

Herbs play a vital role in our lives, particularly in the realm of medicine. Traditional medicine, in fact, relies heavily on medicinal plants, with approximately 3.3 billion people in developing countries using them regularly (Davidson, 2010). The use of herbs in pharmacological treatments dates back centuries, with folk healing practices worldwide incorporating herbs into their traditions.[17] Aframomum melegueta, commonly known as Grains of Paradise, plant is also known as Ewe Atare (in Yoruba), chitta (Hausa), or Guinea pepper leaf, is a prime example of an herb with remarkable healing properties. Native to West Africa, particularly in Nigeria's swampy coastal regions, this herbaceous perennial plant belongs to the ginger family.[8] Its distinctive trumpet-shaped flowers and reddish-brown seeds have been used for centuries to treat various ailments. Aframomum melegueta is widely found in countries such as Ghana, Liberia, and Nigeria, growing up to 1.5 meters in height (Inegbenebor, 2009).[8] Its unique characteristics and medicinal properties make it an invaluable resource in traditional medicine.

In Nigeria, West Africa, Aframomum melegueta, commonly known as Grains of Paradise, has been utilized for centuries to combat infectious diseases, including urinary tract infections caused by various pathogens, such as Escherichia coli and Staphylococcus saprophyticus (Moret, 2013). Additionally, it has been employed as a spice for meats, sauces, and soups. Traditionally, Aframomum melegueta is combined with other herbs to treat common ailments, including body pains, diarrhea, and sore throat, in West Africa, particularly in Nigeria (Ajaiyeoba and Ekundayo, 1999). This perennial herbal plant is cultivated for its valuable medicinal and pharmacological effects, including antimicrobial, hepato-protective, anti-cancer, and anti-diabetic properties[6,12]

*Corresponding author: Esther Udo Umoh
Email: estyluv100@gmail.com

DOI: https://10.18231/j.ijcbr.2025.004

Benign prostatic hyperplasia (BPH) is a prevalent condition affecting older men, characterized by the uncontrolled growth of prostatic glandular epithelial and stromal elements[1], 2014). As the prostate enlarges, it constricts the urethra, leading to symptoms such as weak urinary stream, incomplete bladder emptying, and bladder outlet obstruction.[18] The prostate gland's development and growth are dependent on androgen stimulation, particularly dihydrotestosterone (DHT) (Shin et al., 2012; Obeagu et al., 2017). The human prostate comprises two primary cell types: glandular cells and dense stromal cells (Patel and Parsons, 2014). Notably, serum concentrations of testosterone and DHT decrease with age in older men.[2] However, DHT production is significantly elevated in men with BPH.[3] Conventional treatments for BPH, such as 5α-reductase inhibitors, can have adverse effects, including loss of libido and erectile disorders (Traish et al., 2011). Consequently, there is growing interest in exploring phytotherapy as a treatment option for BPH, as plant-derived compounds may offer fewer side effects.

Testosterone propionate is a synthetic androstane steroid derivative of testosterone, characterized by a short half-life and slow release. As a 17β propionate ester of testosterone, it plays a crucial role in inducing protein production related to male sexual development. Clinical trials have demonstrated a decrease in plasma LH following administration of testosterone propionate. Upon intramuscular administration, testosterone propionate is absorbed and activates androgen receptors, either directly or through conversion to dihydrotestosterone (DHT). Additionally, it can be converted to estradiol, activating certain estrogen receptors. The binding of free testosterone to androgen receptors in target tissue cells influences transcriptional activity, producing androgen effects. Due to the presence of a less polar ester group, testosterone propionate exhibits slow absorption from the intramuscular site of administration. This necessitates frequent injections, particularly when compared to testosterone enanthate or testosterone cypionate. This study aims to investigate the effects of ethanol leaf extract of Aframomum melegueta on testosterone propionate-induced benign prostatic hyperplasia in Wistar rats.

2. Materials and Methods

2.1. Collection of plant material

The leaves of *Aframomum meleguata,* were collected from Ijare in Ifedore Local Government of Ondo State, identified and authenticated at Center for Research and Development (CERAD), Federal University of Technology, Akure.

2.2. Preparation of Aframomum meleguata

The leaf identification and authentication was carried out in the Department of Crop, Soil and Pest Management, at the University (FUTA) Nigeria, and voucher specimen was deposited at the Herbarium. The leaves were air dried at room temperature and pulverized into powder. A thousand grams of the powdered leaves was macerated in 3000 ml of ethanol (solvent of extraction) for 48 hrs at 50^{o}C. The filtrate was collected initially through a fine linen cloth and finally through Whatman filter paper, thereafter freeze-dried. The freeze dried extract was stored in an air-tight container in a 4^{o}C refrigiretor for further analysis.

3. *In-vivo* Experimental Design/Animal Grouping of Testosterone Propionate Induction and Treatment using *Aframomum meleguata*

A total of thirty (30) Wistar rats were purchased from Temilade rats farm, Ogbomoso, Oyo State. All the animals were housed in well ventilated cages made of wood and wire gauze. Wood shaving were used as beddings to keep each compartment dry. Here, normal standard ambient conditions of temperature between 28-31^{0}c, relative humidity between 50%-55% and a photoperiodicity of 12h natural light and 12h dark were maintained They were fed on standard rat chow (Vital Feeds LTD, Nigeria) and water ad libitum. The animals weighed between 150-250 g and were divided into five 5 (5) groups of n=6 animals each.

Testosterone propionate was induced to the animals by i.v for 14 days. Thereafter, EAM was dissolved in saline (vehicle) and administered to animals by oral gauge for twenty-eight days.

The animals were divided as follows:

1. **Group 1** (Control): Administered with vehicle (0.9% normal saline) only
2. **Group 2** (Testosterone-propionate**):** Administered with testosterone-propionate to serve as the positive control
3. **Group 3** (Testosterone-propionate): Administered with testosterone-propionate + EAM (1600 mg/kg)
4. **Group 4** (Testosterone-propionate + EAM): Administered with testosterone-propionate + EAM (2900 mg/kg)
5. **Group 5** (Fina): Administered with testosterone-propionate + Finasteride (0.5 mg/kg).

3.8. Necropsy, blood collection and preparation

3.8.1. Preparation of tissue homogenates

The prostate, brain and testes were quickly rinsed in ice-cold 1.15% potassium chloride solution, blotted and weighed. The tissues were subsequently homogenized in cold 0.1 M potassium phosphate buffer pH 7.4. The homogenates were cold-centrifuged for 10min at 3000 rpm and supernatants were collected and stored at 4°C prior to biochemical assays.

3.9. Statistical analysis

All data derived from the animal experiment analyses are expressed as group means ± standard deviation. The experimental results were analyzed using appropriate analysis of variance (ANOVA) followed by Duncan's New Multiple Range Test for the post hoc. In all the tests, $p<0.05$

was taken as a criterion for statistical significance. The statistical software used to analyze the data was Statistical Package for Social Science (SPSS 17.0) for windows.

4. Results

4.1. Prostatic indices of EAM

Testosterone Propionate induction significantly decreased ($p<0.05$) the concentration of DHT (**Figure 1**) when compared to the un-intoxicated groups (negative control group). Also, treatment with EAM boosted the DHT concentration in the treated groups (groups 3 and 4) and Finasteride group (Group 5), thus restoring the depleted defense against free radicals. Free Testosterone concentration (**Figure 2**) was depressed in the testes due to testosterone propionate induction, but treatment with dose-dependent of EAM significantly ($p<0.05$) elevated the concentration of testosterone in both the EAM treated group and the standard drug group. The concentration of testes (**Figure 3**) was significantly ($p<0.05$) inhibited by testosterone propionate-inhibition when compared to the negative control group ($p<0.05$), administration of dose-dependent of EAM and standard drug to their respective groups significantly ($p<0.05$) elevated the concentration of serum testosterone. Prostate specific antigen (**Figure 4**) activity increased after treatment with dose-dependent EAM and standard drug but the assaulted group (group 2) were observed to have decreased in their activity. Prostrate weight **Figure 5**: the heavy weight of the prostrate was seen in group 2, this could be due the toxicant induction, but group 3, 4 and 5 was observed to have reduction /lighter weight. This means that SPM extract has the potential in reduction of the effect cause by the toxicant that result to the reduction in weight observed in group 3 and 4 respectively.

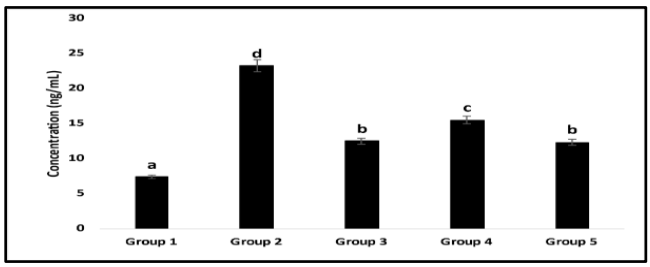


Figure 1: Effects of EAM on the concentration of DHT in benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p\leq0.05$).

Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

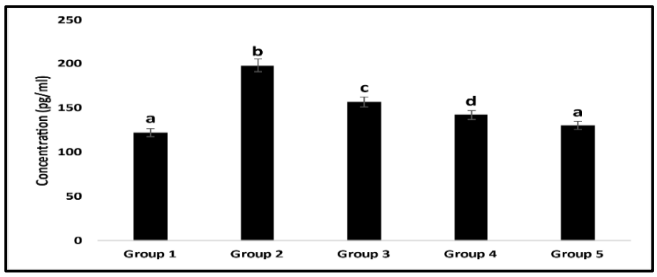


Figure 2: Effects of EAM on the concentration of free testosterone in benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p\leq0.05$).

4.3. Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

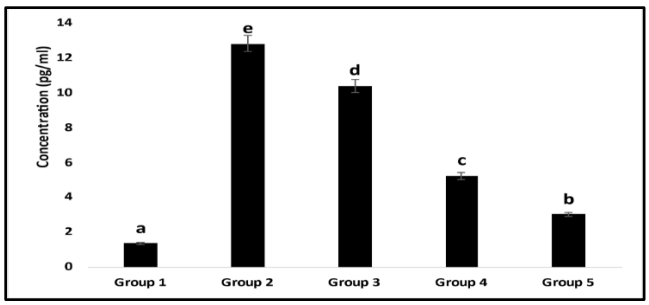


Figure 3: Effects of EAM on the concentration of prostate specific antigen in benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p\leq0.05$).

Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

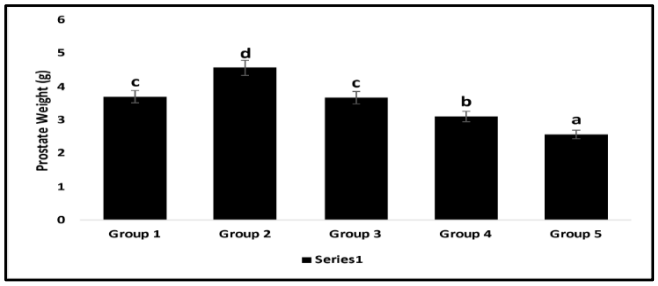


Figure 4: Effects of EAM on the concentration of prostrate weight in benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p \leq 0.05$).

Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

4.2 Neuroprotective potential of EAM.

Brain dopamine level (**Figure 7**) was significantly decreased ($p<0.05$) in testosterone propionate induced group (positive group) as compared to the EAM groups and the negative control (induced but not treated). Treatment with EAM significantly elevated dopamine level in a dose-dependent manner as compared with positive control group ($p<0.05$), and in Finasteride treated group (the reference group) which had the highest concentration of dopamine.

The activity of aceylcholinesterase, was revealed in **Figure 8**, there was a significant increase ($p<0.05$) in the activity of aceylcholinesterase in the brain in rotenone induced rat. EAM treated groups significantly increased the cholinergic transmission impaired by the testosterone propionate ($p<0.05$). **Figure 4**.[9] revealed the activity of tyrosine hydroxylase, it was shown that, there was a significant decrease ($p<0.05$) in the activity of tyrosine hydroxylase in the brain of testosterone propionate-induced rats group (positive control group), compared to the experimental groups. Treatment with EAM significantly elevated the activity of tyrosine hydroxylase in a dose-dependent manner ($p<0.05$). Effects of testosterone propionate-intoxication and EAM dose-dependent treatment on the activity of complex-I enzyme was revealed in Figure 4.10. The result showed that there was a significant decrease ($p<0.05$) in the activity of NADH-Ubiquinone dehydrogenase in the brain of testosterone propionate-induced neurotoxic rats (positive control group), treatment with EAM significantly ($p<0.05$) elevate NADH-Ubiquinone dehydrogenase activity in a dose-dependent manner compared to the testosterone propionate-induced neurotoxic rats.

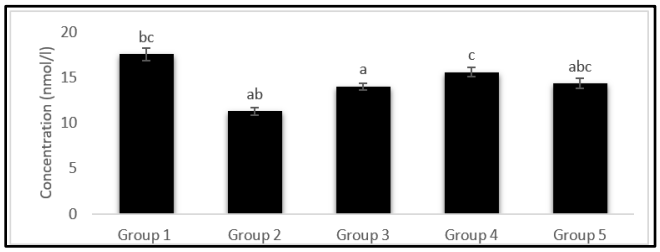


Figure 5: Effects of EAM on the dopamine concentration of in neurological dysfunction connecting to benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p \leq 0.05$).

Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract;
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

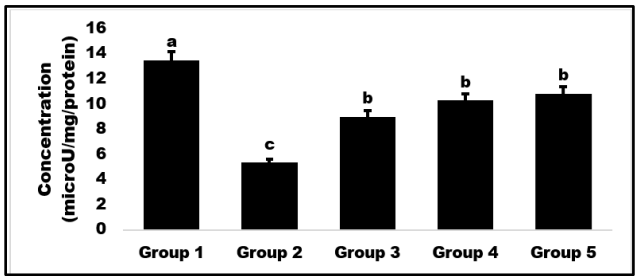


Figure 6: Effects of EAM on the acetylcholineterase activity of in neurological dysfunction in connection to benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p \leq 0.05$).

Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

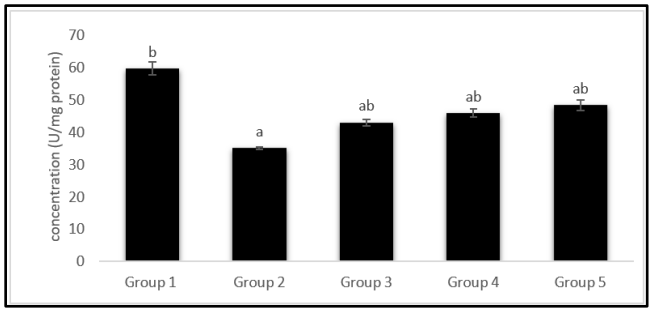


Figure 7: Effects of EAM on the tyrosine hydroxylase activity of in neurological dysfunction in connection to benign prostatic hyperplasia in wistar rats. Results were presented as mean ± standard deviation where n=6. Values with different superscripts are significantly different ($p \leq 0.05$).

Treatments

Group 1: Negative Control;
Group 2: testosterone propionate assault without treatment (Positive Control);
Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

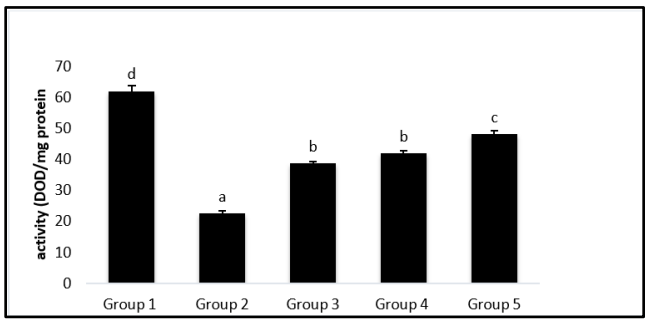


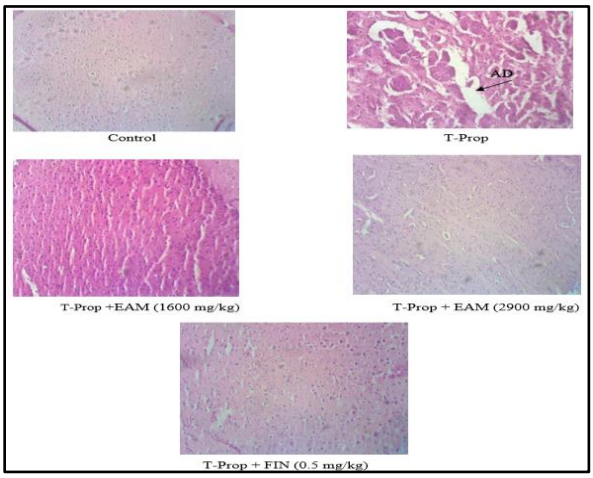
Figure 8: Effects of EAM on the NADH-Ubiquinone dehydrogenase activity of in neurological dysfunction in connection to benign prostatic hyperplasia in wistar rats. Results were presented as mean±standard deviation where n=6. Values with different superscripts are significantly different ($p \leq 0.05$).

Treatments

1. Group 1: Negative Control;
2. Group 2: testosterone propionate assault without treatment (Positive Control);
3. Group 3: testosterone propionate assault and 1600mg/kg BWT of EAM extract;
4. Group 4: testosterone propionate assault and 2900mg/kg BWT of EAM extract; and
5. Group 5: testosterone propionate assault with Finasteride (0.5 mg/kg).

5. Effect of EAM on Histo-architecture of Testosterone Propionate -Toxified Rats

Represent hematoxylin and eosin stained sections of the prostate region of rats subjected to testosterone-propionate-induced Benign Prostatic hyperplasia and treated with EAM. Atrophic degeneration of cell was observed in the cortical brain region of testosterone-propionate administered rats (positive control group) while dissolution of nucleus (Karyolysis) was observed in the hippocampal brain region of the testosterone-propionate -induced rats. These histopathological effects were not present in the control group. The damage to the prostate region were reversed on treatment with EAM. However, posterior karyolysis was observed in the hippocampus of rats treated with (2900 mg/kg) EAM alone.



6. Discussion

Benign prostatic hyperplasia (BPH), also known as prostate gland enlargement, is a common condition that affects the prostate gland in men leading to significant morbidity and mortality.[2] Animal models play a critical role in studying prostatic hyperplasia and evaluating potential treatments.[7] The testosterone propionate model is widely used and has been shown to replicate key clinical and pathophysiological features of benign prostatic hyperplasia.[9] Most of the drugs currently being used have unpleasant side effects and unpredictable pharmacological actions.[15] The application of herbal medicines in the treatment of various diseases is growing at a fast rate globally, the low toxicity and easy accessibility being major contributors to its global acceptance. The leaf extract *Aframomum meluegata*e exhibited antioxidative, antiinflamtory and antiapoptotic effects in animal models of benign prostatic enlargement. Hence this study assessed the ameliorating effect of selected polyherbal mixture on testosterone-proprionate-induced BPH.

6.1 Induction with testosterone propionate and it effect

In this study, testosterone propionate administration on neuroprotective potentials led to an increase in the

hippocampal and cortical dopamine level, increased activities of tyrosine hydroxylase and monoamine oxidase, increased level of dopamine is in agreement with studies.[4,16] There is a resultant increase in the level of dopamine, tyrosine hydroxylase, acetylcholinesterase and NADH-Ubiquinone dehydrogenase on the treated groups, and this changes in dopamine levels and/or its specific receptors can alter the neuromodulatory action of dopamine on brain circuits especially in the limbic system,[16] which is as a result of the positive impact observed from the used EAM for the treatment of BHP.

Result from Prostatic indices (DHT, PSA, Free testosterone concentration, Serum testosterone concentration and Prostrate weight), it was observed that the induced group showed a significant decreased in their concentration in all the prostrate indices except in the Serum testosterone concentration which was observed that the induced but not treated group had an increase in it concentration, but pre-treatment with the EAM was able to alter the negative effect cause by testosterone propionate during induction. Other assays such as (DHT, PSA, Testosterone concentration and prostrate weight), was observed to showed decrease in their concentration during the cause of induction and pre-treated with EAM leads to an increased showed as seen in **Figure 4** and **Figure 7**.[10]

The observed ameliorative effects of EAM in testosterone propionate-induced BHP from biochemical evaluations were further corroborated by the histopathological observations of the cortical and hippocampal sections of the experimental animals. The histological sections showed that testosterone propionate-induced rats had hypostatic congestion, distorted nerves, atrophic degeneration and karyolysis compared to the control group and treated groups. Atrophic degeneration which led to forced apoptosis and neuronal loss in the hippocampus and cortex has been reported in patients seen in the induced but treated group as well as high brain glutamate causes excitability toxicity and leads to nerve damage.[2,10] This may be the underlying cause of the degenerative changes in cells of the hippocampus and cortex in this study, groups treated with EAM showed considerable recovery patterns in the histoarchitecture of these brain regions indicating neuroprotective action of the extract.

7. Conclusion

The result obtained in the present study revealed that *Aframomum meluegata* leaf extract ameliorated testosterone-propionate–induced benign prostatic hyperplasia through its antioxidative, anti-inflammatory and anti-apoptotic effect. This finding is in agreement with the ethnomedicinal uses of the plant. Hence, *Aframomum meluegata* could be a better pharmacological approach in the management of human benign prostatic hyperplasia and other related LUTS.

8. Source of Funding

None.

9. Conflict of Interest

None.

References

1. Afriyie D.K. Pharmacological and Toxicological Evaluation of Stem Extracts of Croton Membranaceus in Benign Prostatic Hyperplasia and Prostate Cancer Models (Doctoral dissertation, University of Cape Coast). 2010; P–307.
2. Cai Y, Jia Z., Zhang Y, Kang B, Chen C, Liu W, Zhang W. Cell-free fat extract restores hair loss: a novel therapeutic strategy for androgenetic alopecia. *Stem Cell Rese Ther*. *2023;*14(1):219.
3. Carson III, C., & Rittmaster, R. The role of dihydrotestosterone in benign prostatic hyperplasia. *Urology*, 2013;61:(4), 2–7.
4. Clinckers R, Gheuens S, Smolders I, Meurs A, Ebinger G, Michotte Y. In vivo modulatory action of extracellular glutamate on the anticonvulsant effects of hippocampal dopamine and serotonin. *Epilepsia*. 2015;46(6):828–36.
5. Davidson S, Ralston S, Walker BR, Colledge NR, (2010) Davidson's principles and practice of medicine. p–1274
6. El-Halawany AM, Dine RSE, Sayed EL, Hattori M. Protective effect of Aframomum melegueta phenolics against CCl4-induced rat hepatocytes damage; role of apoptosis and pro-inflammatory cytokines inhibition. *Sci Rep*. 2014;4(1):5880.
7. Hieble J. Animal Models for Benign Prostatic Hyperplasia. Handbook of experimental pharmacology. 2021;(202):69–79.
8. Inegbenebor U. Effect of aqueous extract of alligator pepper (Zingiberaceae aframomum melegueta) on gestational weight gain. *Niger J Physiol Sci*. 2009;24(2):165–9.
9. Jarvis TR, Chughtai B, Kaplan SA. Testosterone and benign prostatic hyperplasia. *Asian J Androl* 2015;17(2):212–6.
10. Kang M., Xin H., Yuan J., Ali S.T., Liang Z., Zhang J., Wu P. (2022). Transmission dynamics and epidemiological characteristics of SARS-CoV-2 Delta variant infections in Guangdong, China, May to June 2021. *Eurosurveillance*, *27*(10):2100815.
11. Luo J., Zha S., Gage W.R., Dunn T.A., Hicks J.L., Bennett C.J., De Marzo A.M. (2002). α-Methylacyl-CoA racemase: a new molecular marker for prostate cancer. *Cancer Res*. 62:(8):2220–6.
12. Mohammed H.S., Abdel-Aziz M.M., Abu-Baker M.S., Saad A.M., Mohamed M.A., Ghareeb M. A.. Antibacterial and potential antidiabetic activities of flavone C-glycosides isolated from Beta vulgaris subspecies cicla L. var. flavescens (Amaranthaceae) cultivated in Egypt. *Curr Pharma Biotech*. 2019;*20*(7):595–604.
13. Erica S. Trans-Atlantic Diaspora Ethnobotany: Legacies of West African and Iberian Mediterranean Migration in Central Cuba. Afric Ethnobotany in the Am. Springer. 2013; pp. 217–45.
14. Obeagu E.I., Amilo G.I., Obeagu G.U., Ugwuja S.E.,Agbo E.A. Evaluation of impact of level of prostate specific antigen on haematological parameters of men in Owerri, Nigeria. *J Biomed Sci Appl*.2017;(1):1–3.
15. Rabiei Z, Naderi S, Rafieian-Kopaei M. Study of antidepressant effects of grape seed oil in male mice using tail suspension and forced swim tests. *Bangladesh J Pharmacol*. 2017;12:(4)397–402.
16. Rezaei Z., Marandi, S. M., Alaei, H., & Esfarjani, F. The effect of preventive exercise on the neuroprotection in 6-hydroxydopamine-lesioned rat brain. *Appl. Physiol. Nutr. Metab*. 2019;44(12):1267–75.
17. Schulz V., Hnawl, R. and Tyler, V. E. Rational phytothertapy. A physician;s guide to herbal medicinem.202. 4th ed. p-402, berlin, springer-verlag.
18. Trumbeckas D., Milonas D., Jievaltas M., Matjosaitis A.J., Kincius M., Grybas A., Kopustinskas, V. Importance of prostate volume and urinary flow rate in prediction of bladder outlet obstruction in men with symptomatic benign prostatic hyperplasia. *Cent Eur J Urol*.2011; 64(2):75.

19. Zhang J., Zhang M., Tang J., Yin G., Long Z., He L., Zhou C. Animal models of benign prostatic hyperplasia. *Prost Cancer Prost Dis*. 2021;24(1):49-57.

Cite this article: Umoh EU, Ibukun EO, Buraimoh OB, Olowogbade AA. Therapeutic roles of Aframomum melegueta on testosterone propionate induced benign prostrate hyperplasia in wistar rats. *Oyubu GInt J Clin Biochem Res*. 2025;12(1):24-30