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Original Research Article

Effect of virgin coconut oil, lauric acid and myristic acid on serum and prostatic markers of benign prostatic hyperplasia

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Abstract

Purpose: *To investigate the effect of virgin coconut oil, lauric acid and myristic acid on serum and prostatic androgens in testosterone-induced prostatic hyperplasia.*
Methods: *Benign prostatic hyperplasia (BPH) was induced in the animals by repeated subcutaneous injection of testosterone propionate (5 mg/kg) at the inguinal region once a day for 28 days. Thereafter, BPH was treated for 56 days by oral administration of virgin coconut oil, lauric acid and myristic acid. Following the treatment period, the rats were sacrificed and blood samples were collected through cardiac puncture for biochemical analysis.*
Results: *Virgin coconut oil, lauric acid and myristic acid led to a significant reduction ($p < 0.05$) in serum prostatic acid phosphatase (PAcP), prostate specific antigen (PSA) and dihydrotestosterone (DHT) levels, and also in prostatic DHT level.*
Conclusion: *This study provides evidence that virgin coconut oil, lauric acid and myristic acid may be useful in the management of BPH because they exerted some anti-proliferative effects in the development and progression of BPH. Therefore, coconut may be a potential functional food for the management of BPH patients because it is rich in both lauric and myristic acid. However, further investigations, including clinical trials are required to buttress this.*

Keywords: *Benign prostatic hyperplasia, Virgin coconut oil, Prostatic acid phosphatase, Prostate specific antigen, Dihydrotestosterone*

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INTRODUCTION

Benign prostatic hyperplasia (BPH) refers to the non-cancerous growth of the prostate seen very commonly in aging men. This leads to an appreciable growth in the glandular-epithelial and stromal/muscle tissue in the prostate, resulting in the formation of large, fairly discrete nodules [1]. When sufficiently large, these nodules encroach on the urethra and increase resistance to flow of urine from the bladder.

Serum prostatic acid phosphatase and prostate specific antigen (PSA) are prostatic secretions

used as markers of prostatic diseases and indicators of treatment progress [2,3]. They increase in concentration when there is an enlarged prostate. Benign prostatic hyperplasia appears to play a central role in BPH pathogenesis, in addition to being necessary for prostate development. Increased risk of BPH with higher serum testosterone levels has never been reported in any of the studies [4–6]. This implies that higher serum testosterone concentrations do not promote BPH. In contrast, several studies have reported an increased risk of BPH with increased serum concentrations of DHT and its metabolites [5,6]. 5α-reductase inhibitors (finasteride and dutasteride) decrease serum concentrations of DHT and prevent clinical progression of BPH and lower urinary tract symptoms (LUTS). These observations are consistent with the role of DHT in hyperplastic prostate growth and show that testosterone, per se, is not critically involved directly.

Pharmacologic therapy for relieving LUTS in BPH patients is alpha-1-adrenergic receptor antagonists (e.g., alfuzosin, doxazosin, tamsulosin, terazosin) used to reduce smooth muscle tone in bladder neck and prostrate; and 5α-reductase inhibitors (finasteride and dutasteride) used to reduce prostrate size. These drugs have side effects such as changes in ejaculation, headaches, nasal congestion, interaction with anti-hypertensives, gynecomastia and weakness [7-9]. Benign prostatic hyperplasia patients have low compliance to standard pharmaceutical medications because of concerns about their unpleasant side effect. Phytotherapies are less expensive and well tolerated and adverse events are generally mild and infrequent.

Virgin coconut oil is reported to have anti-cholesterogenic, antimicrobial, antifungal, antioxidant, antitumor, immune-booster activities [10,11]. High lauric and myristic acid concentration in virgin coconut oil is believed to be primarily responsible for its special and beneficial effects. Noa *et al* [12] revealed that coconut oil reduced prostate weight and relative prostate weight in testosterone-induced prostatic hyperplasia. However, there is no evidence of studies on the effect of virgin coconut-oil on serum PAP, PSA, DHT and prostatic DHT in the management of BPH in a rat model. The major features of human BPH, including functional and histological changes can be reproduced in prostate enlargement induced by testosterone. Therefore, prostate enlargement induced by testosterone was used to assess the effects of potential treatments for BPH.

METHODS

Animals and grouping

A total of thirty-five (35) adult male albino rats weighing 165 ± 12.9 g were used in this study. The animals were kept in metal cages with 12 h dark and 12 h light period throughout the experimental period. After acclimatization for one (1) week, the rats were randomly divided into 7 groups (n = 5). Commercial feed and clean water were made available to the rats *ad libitum*. Ethical approval was gotten from the animal research and ethics committee of University of Nigeria, Enugu campus approval no. NHREC/21/03/2020B-FWA00002748-IRB00002123). The experiments followed international guidelines for animal studies.

Extraction of virgin coconut oil

Extraction of virgin coconut oil was done using wet cold-pressed extraction method according to Agarwal and Bosco [13]. Coconut milk was extracted from freshly harvested coconuts, and fermented for 24 - 36 h. During this period, the oil phase got separated from aqueous phase. The separated oil phase was harvested and slightly heated for a short time to remove the moisture and finally filtered into a container.

BPH induction and drug administration

Benign prostatic hyperplasia was induced in the experimental animals. This was done by repeated subcutaneous injection of testosterone propionate (5 mg/kg) at the inguinal region once a day for 28 days according to Sayed *et al* [14]. Afterwards, BPH was treated for 56 days. Group 1 (normal control group) did not receive any treatment. Group 2 was BPH untreated group (negative control). Group 3 was BPH treated with standard drug (positive group). Groups 4 received 800 mg/kg of coconut oil. Group 5 received 360 mg/kg of lauric acid and 140 mg/kg of myristic acid. Group 6 received 360 mg/kg of lauric acid while group 7 received 140 mg/kg of myristic acid. Lauric acid and myristic acid were suspended in distilled water using Tween 80 and administered orally. All rats were treated once a day for eight weeks.

Blood and tissue sample collection

After the treatment period of 56 days (8 weeks), the rats were sacrificed under light anaesthesia (using diethyl ether) and blood samples collected through cardiac puncture. Blood samples of the animals were collected into plain tubes. The blood samples were allowed to clot, centrifuged

to get the serum and stored at -80°C until used. The experimental animals were dissected to obtain prostate specimens, which were used for the determination of intra-prostatic DHT concentration.

Measurement of serum PSA

The serum levels of Prostate Specific Antigen (PSA) were determined with a PSA ELISA kit according to the manufacturer's instructions (Rapid Labs. Ltd, Colchester, Essex, UK). The absorbance was measured at 450 nm using a microplate ELISA reader. The values were expressed as ng/mL.

Measurement of serum PAcP

Following manufacturer's instruction, the levels of PAP in the serum were determined using Biosystem kit (S. A. Costa Brava 30, Barcelona, Spain).

Measurement of levels of DHT and testosterone in serum and prostrate

Testosterone levels in the serum and prostrate were measured with ELISA kits (Bio Check, Inc. 323 Vintage Park Drive, Foster City, CA), while DHT ELISA kit (Iceberg Technology Shanghai China) was used to measure DHT in serum and in prostrate at the absorbance of 450 nm using a microplate ELISA reader and values were expressed in ng/mL.

Statistical analysis

IBM SPSS 20.0 for Windows was adopted for statistical analysis. Data are expressed as mean ± SEM. One-way ANOVA was used for group comparisons. This was followed by independent sample t-test, and $p < 0.05$ were considered statistically significant.

RESULTS

Serum PAcP levels

Significant elevations ($p < 0.05$) in serum prostatic acid phosphatase were observed in the negative control (4.69 ± 1.33 ng/mL) when compared to all the other groups. Serum prostatic acid phosphatase level was lowest (1.26 ± 0.44 ng/mL) in the myristic acid group, followed by positive control (1.29 ± 0.50 ng/mL). Compared with the positive control, coconut oil group increased (2.15 ± 0.86 ng/mL) though not significantly ($p < 0.05$) in its level of serum acid phosphatase activity. Similar moderate increases were also observed in the serum activity level of acid phosphatase in lauric and myristic acid group (2.20 ± 0.46 ng/mL) and in Lauric acid (2.27 ± 1.31 ng/mL) group when compared to the positive control (Table 1).

Table 1: Effect of different treatments on serum marker of BPH (PAcP, ng/mL). ***Note:*** BPH: benign prostatic hyperplasia; PAcP: prostatic acid phosphatase

Group	PAcP (ng/mL)
Group 1: Normal control (un-induced)	1.84±0.44[a]
Group 2: Negative control (BPH untreated)	4.69±1.33[b]
Group 3: Positive control (BPH + standard drug)	1.29±0.50[a]
Group 4: BPH + coconut oil	2.15±0.86[a]
Group 5: BPH + lauric and myristic acid	2.20±0.46[a]
Group 6: BPH + lauric acid	2.27±1.31[a]
Group 7: BPH + myristic acid	1.26±0.44[a]

Different superscripts on the same column implies that significant difference exists between their mean values. Mean values with same alphabet superscript on the same column show no significant difference at $p < 0.05$

Serum PSA levels

From Table 2, PSA level in the serum was highest in the negative control (0.75 ± 0.10 ng/mL) when compared to all the other groups. The PSA with lowest value was found in myristic acid (0.49 ± 0.20 ng/mL), followed by the finasteride-treated group (0.65 ± 0.08 ng/mL).

Table 2: Effect of different treatments on serum marker of BPH (PSA) (ng/mL)

Group	PSA (ng/mL)
Group 1: Normal control (un-	0.62±0.03[a]
Group 2: Negative control (BPH	0.75±0.10[a]
Group 3: Positive control (BPH +	0.65±0.08[a]
Group 4: BPH + coconut oil	0.65±0.07[a]
Group 5: BPH + lauric and myristic	0.72±0.14[a]
Group 6: BPH + lauric acid	0.69±0.03[a]
Group 7: BPH + myristic acid	0.49±0.20
P-value	0.066

BPH: Benign prostatic hyperplasia; PSA: Prostate specific antigen. Different superscripts on the same column implies that significant difference exists between their mean values. Mean values with same alphabet superscript on the same column shows no significant difference at $p < 0.05$

Interestingly, PSA level in the coconut oil group (0.65 ± 0.07 ng/mL) and positive control group (0.65 ± 0.08 ng/mL) were almost of same value. Non-significant ($p < 0.05$) increases were also observed in the PSA level found in lauric and myristic acid group (0.72 ± 0.14 ng/mL) and lauric acid group (0.69 ± 0.03 ng/mL) respectively, when compared with the positive control.

Serum testosterone and DHT levels

Serum testosterone level was lowest (0.95 ± 0.12 ng/mL) in the normal control group when compared to all the other groups. Of all the treated groups, serum testosterone level was highest in the finasteride-treated group (standard) (1.33 ± 0.13 ng/mL). In other words, serum testosterone level decreased in all the experimental groups when compared with the standard (Table 3). As expected, DHT level in serum was significantly ($p < 0.05$) highest in the negative control (5.97 ± 2.13 ng/mL), when compared to all the other groups. Serums DHT in all the other groups were decreased. However, the least value of serum DHT was observed in the myristic acid group (1.69 ± 0.17 ng/mL).

Testosterone and DHT levels in prostate

In the prostate, testosterone level was lower than DHT level in all the groups (Table 4); similar to observation made in the serum levels of testosterone and DHT. Prostatic testosterone was significantly low ($p < 0.05$) in the negative control (0.67 ± 0.06 ng/mL), when compared with the normal (0.77 ± 0.09 ng/mL). Coconut oil group and lauric acid group had a significantly higher ($p < 0.05$) concentration of testosterone (0.77 ± 0.09 ng/mL) when compared to the standard (finasteride group) (0.63 ± 0.03 ng/mL). As expected, prostatic DHT level in the negative control had the highest value (1.77 ± 0.15 ng/mL), when compared to all the other groups. On the other hand, coconut oil group recorded the lowest prostatic DHT (1.08 ± 0.47 ng/mL) among the groups. All the other treatment groups (standard, lauric acid, myristic acid, lauric and myristic group) exhibited decreases in prostatic DHT, when compared to negative control.

Table 3: Effect of different treatments on serum markers of BPH (testosterone and DHT) (ng/mL)

Group	Testosterone (ng/mL)	DHT (ng/mL)
Group 1: Normal control (un-induced)	0.95±0.12^{a}	1.59±0.10^{a}
Group 2: Negative control (BPH untreated)	1.27±0.20^{a}	5.97±2.13^{b}
Group 3: Positive control (BPH + finasteride)	1.33±0.13^{a}	1.94±0.10^{a}
Group 4: BPH+ coconut oil	1.29±0.24^{a}	2.53±0.64^{a}
Group 5: BPH + lauric and myristic acid	1.19±0.43^{a}	1.97±0.34^{a}
Group 6: BPH + lauric acid	1.07±0.11^{a}	2.36±0.47^{a}
Group 7: BPH + myristic acid	1.29±0.13^{a}	1.69±0.17^{a}
P-value	0.150	0.001

BPH: Benign prostatic hyperplasia; DHT: dihydrotestosterone. Different superscripts on the same column implies that significant difference exists between their mean values. Mean values with same alphabet superscript on the same column showed no significant difference ($p < 0.05$)

Table 4: Effect of different treatments on prostate markers of BPH (testosterone and DHT)

Group	Prostatic testosterone	Prostatic DHT
Group 1: Normal control (un-induced)	0.77±0.09^{b}	1.56±0.11^{b}
Group 2: Negative control (BPH untreated)	0.67±0.06^{a}	1.77±0.15^{b}
Group 3: Positive control (BPH + finasteride)	0.63±0.03^{a}	1.63±0.14^{b}
Group 4: BPH+ coconut oil	0.77±0.09^{b}	1.08±0.47^{a}
Group 5: BPH + lauric and myristic acid	0.60±0.03^{a}	1.73±0.09^{b}
Group 6: BPH + lauric acid	0.83±0.02^{b}	1.72±0.20^{b}
Group 7: BPH + myristic acid	0.61±0.02^{a}	1.65±0.14^{b}

Key: BPH: Benign prostatic hyperplasia; DHT: dihydrotestosterone. Different superscripts on the same column implies that significant difference exists between their mean values. Mean values with same alphabet superscript on the same column shows no significant difference at $p < 0.05$

DISCUSSION

The present study determined the effect of coconut oil, lauric acid and myristic acid on the treatment of testosterone- induced prostatic hyperplasia. These effects were compared with the effect of the standard drug (finasteride) which is currently being used to treat BPH. After 56 days of BPH treatment, it was observed that virgin coconut oil, lauric acid and myristic acid inhibited the development of testosterone-induced BPH. This was evident in the reduction in serum level of BPH markers (PAcP, PSA and DHT), as well as in the prostate BPH marker (DHT).

Prostatic acid phosphatase is one of the prostatic secretions used as a marker of prostatic diseases and as an indicator of treatment progress. Elevated PAcP level is seen in benign conditions such as BPH and also in malignant conditions such as prostate cancer with and without metastasis [2]. As a result, high values of PAcP were obtained in the negative group that were left untreated which is due to increase in secretory activity of PAcP following hyperplasia. However, the treatment groups revealed a reduction in serum PAcP. Reduction in the secretory activity of PAcP implies that there is a reduction in hyperplasia. In other words, the various treatments may have prevented the growth of prostatic cells (hyperplasia) in the test groups leading to decreased secretion of PAcP in the prostatic cells.

Prostate specific antigen is used as a marker for BPH and prostate cancer due to its tissue specificity and high sensitivity. Together with acid phophatase, PSA gives useful information about prostatic disease especially BPH and prostate cancer. Serum PSA level was highest in the negative control (untreated group), when compared to all the other groups, which confirms that PSA value can be used to predict enlargement of the prostate. High PSA value correlates with increase in prostate mass [3]. The low values of PSA seen in the treatment groups show that the treatment groups may have some anti-proliferative effect in the growth of prostatic cells. From the study, testosterone propionate injection increased the level of serum testosterone in all the BPH-induced groups when compared to the normal control. However, serum testosterone level observed was lower than the serum DHT level in all the BPH-induced rats. The reason for the elevation in DHT is not clear. According to Anawalt [15], all exogenous testosterone formulations increase serum DHT concentrations above physiologically normal serum concentrations. The significantly high serum testosterone concentration observed in finasteride-treated group (standard), when compared to all the other groups confirms finasteride as an 5α-reductase inhibitor. This is because the inhibition of the conversion of testosterone to DHT leads to an increase in the concentration of testosterone.

Several studies have reported an increased risk of BPH with increased serum concentrations of DHT and its metabolites [5,6]. Therefore, the decreased serum DHT level in the finasteride-treated group (standard) further substantiates the fact that finasteride is a good 5α-reductase inhibitor. In a similar manner, virgin coconut oil, lauric acid, myristic acid, and a combination of lauric and myristic acid treatments were also able to partially reduce the serum DHT level. Animal studies carried out by Patil *et al* [16] have also shown that a combination of lauric and myristic acid reduces serum DHT. Coconut oil contains a high percentage of lauric and myristic acid [17]. This implies that coconut oil, lauric acid and myristic acid may inhibit the conversion of testosterone to dihydrotestosterone, thereby reducing an enlarged prostate.

Increased levels of circulating testosterone led to increased expression of 5α-reductases, particularly the type 1 isoenzyme which is expressed in the prostate [18,19]. Increased 5α-reductases in turn, lead to increased conversion of testosterone to DHT, which also leads to stimulation of cell division and prostate development. This could explain the reason why intra-prostatic testosterone level was lower than DHT level in all the groups. It could also be the reason why prostatic testosterone was significantly lower in the negative control, than in the normal.

There was an increased conversion of testosterone to DHT in the absence of an inhibitor (treatment), which is in tandem with the increased prostatic DHT level observed in the negative control (untreated) which was significantly higher when compared to other groups. The low intra-prostatic DHT level observed in finasteride treated group confirms that is good 5α- reductase inhibitor. However, the significantly lower intra-prostatic DHT concentration observed in virgin coconut oil group, when compared to other groups strongly suggests that virgin coconut oil may be effective in blocking the conversion of testosterone to dihydrotestosterone, thereby discouraging further growth of the prostate. According to Nickel *et al* [20], the inhibition of testosterone conversion to

DHT reduces the levels of intra-prostatic DHT and prevents the enlargement of the prostatic epithelium, resulting in a 20 – 30 % decrease in prostate volume after 12 months of treatment. All the other treated groups (lauric acid, myrisitic acid, and a combination of lauric and myristic, respectively) showed moderate reductions in intra-prostatic DHT which still suggests that lauric and myristic acid may be helpful in the management of BPH. According to previous study carried out by Patil *et al* [18], a combination of lauric and myristic acid decrease prostatic DHT.

CONCLUSION

This study provides evidence that virgin coconut oil, lauric acid and myristic acid may be used in the management of BPH. This is because they exerted some anti-proliferative effects on of BPH. These effects could be seen in the reduction in markers of BPH. Reductions were observed in serum PAcP, serum PSA, serum DHT, and prostatic DHT. Therefore, coconut is a potential functional food for the management of BPH patients because it is rich in both lauric and myristic acid. However, further *in vivo* research and clinical trials should be carried out to ascertain this.

DECLARATIONS

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Conflict of interest

No conflict of interest is associated with this work.

Contribution of authors

We declare that this work was done by the authors named in this article, and all liabilities pertaining to claims relating to the content of this article will be borne by the authors. Okorie Claribel Chikwue, Ezeagu Edwin Ikechukwu, Onyekwelu Kenechukwu Chibuike and Ikekpeazu Joy Ebele were involved in the study design, interpretation of data and manuscript preparation. Okorie Chukwuemeka Ogueri, Okorie Claribel Chikwue and Onyekwelu Kenechukwu Chibuike performed laboratory analysis. Ezeagu Edwin Ikechukwu and Ikekpeazu Joy Ebele made substantial contributions to the conception of this study and project administration. All authors read and approved the final manuscript for publication.

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