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Research Article

Body Mass Index, Nutrient Intakes and Serum Anti-oxidant Status of Elderly Men with and Without Benign Prostatic Hyperplasia in Ibadan, Nigeria

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Abstract

Background and Objective: Worldwide, approximately 30 million men have symptoms related to benign prostatic hyperplasia (BPH). There is a high prevalence of the disease, therefore, the associated costs of medical care are also high. Little is documented on the nutritional status of men with BPH in sub-Saharan Africa. This study therefore aimed to determine the body mass index (BMI), nutrient intakes and serum antioxidant status of male adults with and without BPH (benign prostatic hyperplasia) in Ibadan. **Materials and Methods:** This case-controlled, analytical study recruited 28 male adults with BPH from the Urology Section of the Surgical Out-patients Department, University College Hospital, Ibadan and the controls were 17 male adults without BPH, age-matched, living in the same city. BMI was computed and mean daily nutrient intakes were assessed using repeated 24 h dietary recalls and analyzed using the software "Total Diet Assessment". Beta-carotene, retinol and lycopene levels in the serum of the respondents were also determined using high performance liquid chromatography. Statistical analysis was done using SPSS. **Results:** There were no significant differences in the BMI, carbohydrate, fat, protein, energy, iron, sodium and zinc intakes between both groups ($p > 0.05$). However, potassium intakes (807.3 ± 352.2 mg vs 567.2 ± 278.2 mg) and calcium intakes (188.3 ± 163.6 mg vs 103.8 ± 62.3 mg) of the BPH and controls differed significantly ($p = 0.021$ and 0.048 , respectively). Lycopene (2443.1 ± 265.4 μ g) and beta-carotene (8.6 ± 0.3 mg) intakes of the BPH were significantly lower than those of the controls (3862.3 ± 316.2 μ g and 10.1 ± 8.3 mg) respectively, $p < 0.05$. The mean serum lycopene, beta-carotene and retinol (24.2 ± 10.2 , 47.7 ± 28.8 and 356.9 ± 150.7 ng mL⁻¹) of the BPH were significantly lower than the values 70.8 ± 49.8 , 57.6 ± 47.7 and 395.4 ± 275.6 ng mL⁻¹ of the controls respectively ($p < 0.05$). A significant inverse correlation was observed between serum lycopene and BPH ($r = -0.552$, $p = 0.000$). **Conclusion:** Elderly men had low nutrient intakes and the mean calcium and potassium intakes of men with BPH were significantly higher than that of men without BPH, while the mean lycopene and beta-carotene intakes of men without BPH were significantly higher than those with BPH. Furthermore, the serum antioxidant status of men with BPH was significantly lower than that of men without BPH.

Key words: Nutrient intakes, serum antioxidants, body mass index, male adults, benign prostatic hyperplasia

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Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a common disease of elderly men and a risk factor for developing prostate cancer later in life¹. Worldwide, approximately 30 million men have symptoms related to benign prostatic hyperplasia². Little is documented on the prevalence of BPH in Sub-Saharan Africa³. In Nigeria however, the prevalence of BPH has been shown to be as high as 25-35%⁴. There is a high prevalence of the disease, therefore the associated costs of medical care are also high (approximately 4 billion dollars/year in the United States)⁵.

Data from epidemiologic studies provide evidence of a possible significant relationship between dietary habits and the incidence of prostate disorders, with dietary factors including different issues such as the total caloric intake, macronutrients (carbohydrates, proteins and fat) and micronutrients (vitamins and minerals)⁶.

According to Ejike and Ezeanyika³ the eating of processed foods and a sedentary lifestyle apparently are status symbols among the middle and upper classes in Sub-Saharan Africa, resulting in a surge in the disease burden of Sub-Saharan Africa. Clinically apparent BPH represents a considerable health problem for elderly men due to the negative effects it has on quality of life⁷. Wang *et al.*⁸ have indicated an association between body mass index and increased risk of BPH. Benign prostatic hyperplasia may also be caused or exacerbated by chronic inflammation and subsequent oxidative damage⁹ and thus dietary factors such as n-3 fatty acids, polyunsaturated fats and antioxidants may also affect risk.

Antioxidant nutrients that might influence cell growth and differentiation may beneficially influence disorders underlying BPH¹⁰. Retinol and its natural metabolites and synthetic derivatives play important roles in regulating cell proliferation, differentiation, cell arrest at G1 phase and embryonic development¹¹. A re-analysis of physician's health study data provides insight which lends support to the idea that beta-carotene may be protective against prostate enlargement at the doses available from dietary intake¹².

The abundance of lycopene in prostatic tissue is indirectly implicated in the chemoprevention of pathologies likely to affect the prostate gland in the ageing male, such as slowing the progression of BPH¹³. The common pathologic hyper-proliferation of prostate cells in adult men developing BPH may be positively affected by lycopene¹⁴. Schwarz *et al.*¹⁵ also observed that there was no progression of BPH in patients supplemented with lycopene as compared with a placebo group. The novel finding that lycopene reduces

local androgen signaling in the prostate also suggests efficacy in prevention of benign prostate hyperplasia.

In Nigeria, there is a dearth of information on the nutrient and antioxidant intakes of male adults, since they do not represent a vulnerable segment of the population. Furthermore, there is sparse documentation of the relationship between nutritional status and BPH. This study therefore aimed at determining the nutritional status (body mass index, nutrient intakes and serum levels of antioxidants) of male adults with and without BPH and exploring the relationship between their nutritional status and BPH.

MATERIALS AND METHODS

This case-control study of males with and without BPH was carried out in accordance with the ethical standards of the University of Ibadan/University College Hospital Ibadan Ethical Review Committee. Twenty-eight subjects (cases) were randomly selected from the Urology Division of the Outpatient Department of Surgery of the University College Hospital, Ibadan, Oyo state, Nigeria. Criteria for inclusion were histologically confirmed, symptomatic (BPH), high serum PSA or high Prostate Symptom Scores¹⁶, age above 40 years, absence of acute illness and informed consent of willingness to participate in the study. The presence of chronic diseases of the liver and kidneys, histologically confirmed malignancies and inflammatory diseases of the urogenital tract were the exclusion criteria. The controls were seventeen¹⁷ men age-matched and living in the same city, with low serum PSA and who did not report any of the symptoms of prostate enlargement (the controls have been described previously¹⁸, as this study was part of a comprehensive study).

A semi-structured questionnaire was used to collect socio-demographic data (Table 1). Their weights and heights were measured and body mass index (BMI) was computed from their weight and height measurements (Fig. 1). Researchers assessed and analyzed the respondents' daily nutrient intakes using repeated 24 h dietary recalls and an adapted version of the food database 'total diet assessment 3.0'.

Serum lycopene, beta-carotene and retinol of the respondents were extracted from a single 5 mL blood sample drawn from each subject by venipuncture by a phlebotomist using a method similar to that described by Tzeng *et al.*¹⁷ and determined using high performance liquid chromatography (a C18 column, a Rheodyne model 7161 injector, an Agilent model 1100 pump, an Agilent model 1100 UV-VIS detector (wavelength of 272 nm), a binary solvent system of

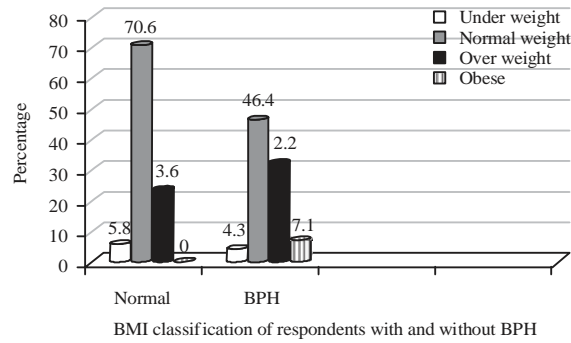


Fig. 1: Body mass index of the respondents

Table 1: Socio-economic characteristics of the respondents

	Controls		BPH		p-value
	n	(%)	n	(%)	
Mean age years (\pm SD)	59.5	12.5	63.8	8.7	0.154
Marital status (N, %)					0.323
Single	1	5.9	0	0	
Widowed	0	0	0	0	
Divorced/separated	0	0	1	3.8	
Married	16	94.1	27	96.4	
Total	17	100.0	28	100.0	
Education					0.570
None	1	5.8	2	7.2	
Non-formal	1	5.8	4	14.2	
Primary	10	58.8	9	32.2	
Secondary	4	23.5	9	32.2	
Tertiary	1	5.8	4	14.2	
Total	17	100.0	28	100.0	
Occupation					0.281
Not employed	0	0	1	3.6	
Farming	3	17.6	4	14.3	
Trading and business	3	17.6	3	10.7	
Artisan	3	17.6	5	17.9	
Civil servant	2	12.0	7	25.0	
Self-employed	3	17.6	0	0.0	
Pensioner	3	17.6	8	28.5	
Total	17	100.0	28	100.0	
Income					0.268
Cannot estimate	0	0.0	3	10.7	
< 5,000	7	41.1	4	14.3	
5,000-10,000	4	23.5	7	25.0	
10,001-15,000	1	5.9	1	3.6	
15,001-20,000	2	11.8	2	7.2	
20,001-25,000	0	0	2	7.2	
25,001-30,000	1	5.9	3	10.7	
>30,000	2	11.8	6	21.3	
Total	17	100.0	28	100.0	

No significant differences ($p > 0.05$) were observed in the mean ages of the BPH and the control (59.5 ± 12.5 years versus 63.8 ± 8.7 years). Other socio-demographic characteristics such as income, occupation and educational status also did not differ significantly between the groups ($p > 0.05$), BPH = Benign prostatic hyperplasia, SD = Standard deviation

HPLC-grade methanol/deionised water (95:5, v/v), flow rate of 1 mL min⁻¹) (Detailed procedures have been described elsewhere)¹⁸.

Statistical analysis: The data were analyzed by computing frequencies, percentages, means and standard deviations of the various variables. Chi-square tests were done

to ascertain if there were any significant differences in the socio-demographic characteristics of the respondents. Student's t-test was used to test for differences in mean BMI, nutrient intakes and serum antioxidant levels. The mean nutrient intakes were also compared with the daily required intake (DRI) for men^{19,20}. Spearman's rank correlation was used to assess the relationship between their serum and dietary antioxidant status and BPH. All analysis were done at the 0.05 level of significance using Statistical Package for the Social Sciences (SPSS) software (version 16.0 for Windows; SPSS Inc., Chicago, IL).

RESULTS

The body mass index of the respondents did not differ significantly between the BPH and controls ($t=0.742$, $p=0.462$).

The mean daily nutrient intakes of the respondents is shown in Table 2. The mean carbohydrate, energy, protein, fat, magnesium and zinc intakes of the respondents were not significantly different ($p>0.05$). However the mean calcium and potassium intakes of the BPH group were significantly higher than those of the controls ($p=0.048$ and 0.021 , respectively). Conversely, the mean lycopene and beta-carotene intakes ($3862.2\pm316.4\ \mu\text{g}$ and $10.1\pm8.3\ \text{mg}$) of the controls were significantly higher than that of the BPH group ($2443.1\pm265.4\ \mu\text{g}$ and $8.6\pm5.3\ \text{mg}$) ($p=0.043$ and 0.039 ,

respectively). There was no significant difference between the retinol intake ($536.4\pm170.7\ \mu\text{g RAE}$) (retinol activity equivalent) of the BPH group and ($545.6\pm870.5\ \mu\text{g RAE}$) of the controls ($p=0.993$).

In Table 3, the serum antioxidant status of the respondents is presented. The mean serum lycopene, beta-carotene and retinol of the BPH were significantly lower than the mean serum lycopene, beta-carotene and retinol of the controls ($p<0.05$).

There were no significant associations between the respondents' dietary intakes of energy, protein, fat, calcium, magnesium, iron, sodium, zinc, lycopene, beta-carotene and retinol ($p>0.05$) and BPH as shown in Table 4. Similarly, no significant associations were observed between the respondents' serum beta-carotene and retinol ($p>0.05$) and BPH. However, a significant positive correlation ($r=0.318$, $p=0.033$) was observed for potassium intake and BPH, while an inverse correlation was observed between the serum lycopene of respondents and BPH ($r=-0.552$, $p=0.000$).

DISCUSSION

Previous studies have indicated that increased adiposity is positively associated with obesity-related metabolic diseases, such as type 2 diabetes, hypertension and dyslipidemia and prostatic hyperplasia^{21,22}. However, this study did not indicate any association between BMI and BPH. The

Table 2: Mean daily nutrient intakes of the respondents

Nutrients	Control intake (SD)	BPH intake (SD)	DRI ^{19,20}	p-value
Carbohydrate (g)	287.80±83.5	243.10±88.0	130	0.100
Energy (kcal)	1678.7±44.5	1628.7±371.8	1800	0.194
Protein (g)	56.7±22.8	57.2±24.4	56	0.941
Total fat (g)	29.5±14.7	29.3±15.8		0.973
Calcium(mg)	103.8±62.3	188.3±163.6	1200	0.048*
Iron (mg)	14.5±5.0	14.5±5.9	8	0.308
Magnesium(mg)	108.40±52.1	142.39±104.3	420	0.219
Potassium (mg)	567.2±278.2	807.3±352.2	4700	0.021*
Sodium (mg)	215.9±146.5	275.9±249.3	1200	0.373
zinc (mg)	8.29±3.39	8.38±4.41	11	0.943
Lycopene (µg)	3862.3±316.2	2443.1±265.4 ^a		0.043*
Beta-carotene (mg)	10.1±8.3	8.6±5.3 ^a		0.039*
Retinol (µg RAE)	545.6±870.5	536.4±170.7	900	0.993

*Significant difference detected, ^aThere are no established DRIs for lycopene and beta-carotene, values are Mean ± SD, BPH = Benign prostatic hyperplasia, DRI = Daily recommended intake, SD = Standard deviation

Table 3: Mean serum lycopene, beta-carotene and retinol status of the respondents

	Control	BPH	p-value
Lycopene (ng mL ⁻¹)	70.8±49.8	24.2±10.2	0.000*
Beta-carotene (ng mL ⁻¹)	57.6±47.7	47.7±28.8	0.041*
Retinol (ng mL ⁻¹)	395.4±275.6	356.9±150.7	0.043*

*Significant difference detected, values are Mean ± SD, BPH = Benign prostatic hyperplasia

Table 4: Correlations between nutrient intakes and BPH

Nutrient	BPH	
	r-value	p-value
Energy	-0.212	0.163
Protein	-0.011	0.945
Fat	-0.071	0.645
Calcium	0.236	0.118
Iron	-0.215	0.156
Magnesium	0.099	0.518
Potassium	0.318	0.033*
Sodium	0.071	0.645
Zinc	-0.019	0.899
Dietary intake of lycopene	-0.234	0.122
Dietary intake of beta-carotene	-0.030	0.845
Dietary intake of retinol	-0.399	0.537
Serum lycopene	-0.552	0.000
Serum beta-carotene	-0.032	0.836
Serum retinol	-0.035	0.818

*Significant correlation, BPH = Benign prostatic hyperplasia

proportion of overweight respondents in this study was higher than the proportions indicated by Oldewage-Theron *et al.*²³ and Lee *et al.*²⁴. In contrast however, the prevalence of obesity in the current study was much lower than those indicated in these two studies.

The carbohydrate, energy, protein, fat, calcium, retinol, potassium and sodium intakes observed in this study were much lower, while the iron and beta-carotene intakes were higher than those observed among BPH patients in a much older study in Japan²⁵. However, the mean energy intake in this study population was similar to that observed in a healthy population of elderly men in South Africa in a more recent study²³. On the contrary, the mean iron and zinc intakes observed in this study were lower than those observed in the study in South Africa²⁴. In the current study, dietary energy intake was not associated with BPH. This is consistent with the findings of Schenk *et al.*²⁶ and Meigs *et al.*²⁷, but contrary to the findings of Suzuki *et al.*²⁸ and Parsons²⁹, who observed direct associations between BPH and energy intakes. Protein intakes were not significantly associated with BPH in this study, contrary to findings by Suzuki *et al.*²⁸ and Parsons²⁹.

In this study, beta-carotene intakes were observed to be higher than the mean beta-carotene intake observed among BPH patients in Italy³⁰. Similarly, the mean intakes of beta-carotene in this study were also found to be higher than the suggestion of an intake of 3–6 mg/day of β -carotene from food sources to maintain plasma β -carotene concentrations in the range associated with a lower risk of various chronic disease outcomes¹⁹. The beta-carotene intakes of the controls in the current study were significantly higher than the BPH group while mean serum beta-carotene levels also differed significantly between the groups. However, a non-significant association was observed between serum beta-carotene

concentrations and BPH, in agreement with the findings that a null association exists between serum beta-carotene levels and the risk of prostate enlargement^{31,32}.

The retinol intakes of the respondents in this study were much lower than the DRI for vitamin A. This may be due to low consumption of foods rich in vitamin A. Furthermore, the non-significant association between BPH and retinol intake is comparable to the indication in recent studies that dietary retinol is not associated with BPH^{30,33,34}.

Highly proliferative prostate epithelium as found in BPH is a risk factor for prostate cancer³⁵. Lycopene is a promising component for the prevention of prostate pathologies³⁶. Although there is an abundance of literature on serum lycopene status of prostate cancer patients in various populations, there is little documentation on the serum lycopene status of BPH patients in many populations. The mean serum lycopene concentrations of the respondents in both groups in this study were much lower than those indicated in a study in the United Kingdom³⁷. Furthermore, the mean lycopene intake and mean serum lycopene of the control group were much higher than those of the BPH group. Similarly, we observed a significant negative association between serum lycopene and BPH. These findings are consistent with the indication that evidence for inverse associations of serum lycopene with prostate enlargement exists⁵.

Further studies need to be carried out to elucidate the relationship between potassium intakes and BPH as there is sparse literature on this topic.

CONCLUSION AND RECOMMENDATIONS

It was concluded that:

- Elderly men with BPH had low nutrient intakes. The mean calcium and potassium intakes of men with BPH were significantly higher than that of men without BPH, while the mean lycopene and beta-carotene intakes of men without BPH were significantly higher than those with BPH
- The serum lycopene, beta-carotene and retinol status of men with BPH was significantly lower than that of men without BPH
- Potassium intake was positively associated, while serum lycopene was negatively associated with BPH

Further epidemiologic studies and clinical trials are necessary to explore the relationship between these antioxidants and prostate enlargement, their role in the

reduction of the risk of prostate enlargement and to elucidate the mechanisms by which they modulate the risk of BPH. Stakeholders in nutrition and public health should embark upon extensive nutrition education in Nigeria in order to enlighten the public on the importance of healthy food choices to eliminate antioxidant nutrient deficiencies which can compromise the health of individuals and the well-being of communities. Preventive strategies should be adopted to reduce the risk of prostate enlargement, such as dietary diversification, the consumption of fruits and vegetables, (especially those that are rich in lycopene such as tomatoes) particularly for men who are at a high risk of BPH.

REFERENCES

1. McVary, K.T., 2006. BPH: Epidemiology and comorbidities. *Am. J. Managed Care*, 12: S122-S128.
2. Emberton, M., E.B. Cornel, P.F. Bassi, R.O. Fourcade, J.M.F. Gomez and R. Castro, 2008. Benign prostatic hyperplasia as a progressive disease: A guide to the risk factors and options for medical management. *Int. J. Clin. Pract.*, 62: 1076-1086.
3. Ejike, C.E.C.C. and L.U.S. Ezeanyika, 2008. Metabolic syndrome in sub-Saharan Africa: Smaller twin of a region's prostatic diseases? *Int. Urol. Nephrol.*, 40: 909-920.
4. Ezeanyika, L.U.S., C.E.C.C. Ejike, O. Obidoa and S.O. Elom, 2006. Prostate disorders in an apparently normal Nigerian population 1: Prevalence. *Biokemistri*, 18: 127-132.
5. Kristal, A.R., K.B. Arnold, J.M. Schenk, M.L. Neuhaus, P. Goodman, D.F. Penson and I.M. Thompson, 2008. Dietary patterns, supplement use and the risk of symptomatic benign prostatic hyperplasia: Results from the prostate cancer prevention trial. *Am. J. Epidemiol.*, 167: 925-934.
6. Tubaro, A., 2006. Micronutrients and BPH. *Eur. Urol.*, 50: 413-415.
7. Donovan, J.L., H.E. Kay, T.J. Peters, P. Abrams and J. Coas *et al.*, 1997. Using the *ICSQoL* to measure the impact of lower urinary tract symptoms on quality of life: Evidence from the ICS-'BPH' study. *BJU Int.*, 80: 712-721.
8. Wang, S., Q. Mao, Y. Lin, J. Wu, X. Wang, X. Zheng and L. Xie, 2012. Body mass index and risk of BPH: A meta-analysis. *Prostate Cancer Prostatic Dis.*, 15: 265-272.
9. Kramer, G., G.E. Steiner, A. Handisurya, U. Stix and A. Haitel *et al.*, 2002. Increased expression of lymphocyte-derived cytokines in benign hyperplastic prostate tissue, identification of the producing cell types and effect of differentially expressed cytokines on stromal cell proliferation. *Prostate*, 52: 43-58.
10. Rohrmann, S., E. Giovannucci, W.C. Willett and E.A. Platz, 2007. Fruit and vegetable consumption, intake of micronutrients and benign prostatic hyperplasia in US men. *Am. J. Clin. Nutr.*, 85: 523-529.
11. Means, A.L. and L.J. Gudas, 1995. The roles of retinoids in vertebrate development. *Annu. Rev. Biochem.*, 64: 201-233.
12. Cook, N.R., M.J. Stampfer, J. Ma, J.E. Manson, F.M. Sacks, J.E. Buring and C.H. Hennekens, 1999. β carotene supplementation for patients with low baseline levels and decreased risks of total and prostate carcinoma. *Cancer*, 86: 1783-1792.
13. Goyal, A., G.H. Delves, M. Chopra, B.A. Lwaleed and A.J. Cooper, 2006. Prostate cells exposed to lycopene *in vitro* liberate lycopene enriched exosomes. *BJU Int.*, 98: 907-911.
14. Obermuller-Jevic, U.C., E. Olano-Martin, A.M. Corbacho, J.P. Eiserich and A. van der Vliet *et al.*, 2003. Lycopene inhibits the growth of normal human prostate epithelial cells *in vitro*. *J. Nutr.*, 133: 3356-3360.
15. Schwarz, S., U.C. Obermuller-Jevic, E. Hellmis, W. Koch, G. Jacobi and H.K. Biesalski, 2008. Lycopene inhibits disease progression in patients with benign prostate hyperplasia. *J. Nutr.*, 138: 49-53.
16. Wertz, K., U. Siler and R. Goralczyk, 2004. Lycopene: Modes of action to promote prostate health. *Arch. Biochem. Biophys.*, 430: 127-134.
17. Tzeng, M.S., F.L. Yang, G.S. Wang-Hsu and B.H. Chen, 2004. Determination of major carotenoids in human serum by liquid chromatography. *J. Food Drug Anal.*, 12: 79-83.
18. Sosanya, M.E., G.T. Fadupin, T. Atinmo and O.B. Shittu, 2014. Anti-oxidant status of male adults with and without prostate cancer in Ibadan, Nigeria. *Food Nutr. Sci.*, 5: 516-524.
19. Food and Nutrition Board and Institute of Medicine, 2000. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids. The National Academies Press, USA., pp: 325-382.
20. USDA., 2010. Nutrient database for standard reference 2010. US. Department of Agriculture (USDA), Release 23, Agricultural Research Service.
21. Patel, N.D. and J.K. Parsons, 2014. Epidemiology and etiology of benign prostatic hyperplasia and bladder outlet obstruction. *Ind. J. Urol.*, 30: 170-176.
22. Hammarsten, J. and B. Hogstedt, 2001. Hyperinsulinaemia as a risk factor for developing benign prostatic hyperplasia. *Eur. Urol.*, 39: 151-158.
23. Oldewage-Theron, W.H., F. Samuel, C. Grobler and A.A. Egal, 2008. Anaemia prevalence and dietary intake of elderly persons living in a peri-urban settlement in South Africa. *Tydskrif vir Gesinsekologie en Verbruikerswetenskappe*, Vol. 36. 10.4314/jfec.v36i1.47111.
24. Lee, S., H.G. Min, S.H. Choi, Y.J. Kim and S.W. Oh *et al.*, 2006. Central obesity as a risk factor for prostatic hyperplasia. *Obesity*, 14: 172-179.
25. Ohno, Y., O. Yoshida, K. Oishi, K. Okada, H. Yamabe and F.H. Schroeder, 1988. Dietary β -carotene and cancer of the prostate: A case-control study in Kyoto, Japan. *Cancer Res.*, 48: 1331-1336.

26. Schenk, J.M., G.S. Calip, C.M. Tangen, P. Goodman, J.K. Parsons, I.M. Thompson and A.R. Kristal, 2012. Indications for and use of nonsteroidal antiinflammatory drugs and the risk of incident, symptomatic benign prostatic hyperplasia: Results from the prostate cancer prevention trial. *Am. J. Epidemiol.*, 176: 156-163.
27. Meigs, J.B., B. Mohr, M.J. Barry, M.M. Collins and J.B. McKinlay, 2001. Risk factors for clinical benign prostatic hyperplasia in a community-based population of healthy aging men. *J. Clin. Epidemiol.*, 54: 935-944.
28. Suzuki, S., E.A. Platz, I. Kawachi, W.C. Willett and E. Giovannucci, 2002. Intakes of energy and macronutrients and the risk of benign prostatic hyperplasia. *Am. J. Clin. Nutr.*, 75: 689-697.
29. Parsons, J.K., 2007. Modifiable risk factors for benign prostatic hyperplasia and lower urinary tract symptoms: New approaches to old problems. *J. Urol.*, 178: 395-401.
30. Tavani, A., E. Longoni, C. Bosetti, L. dal Maso and J. Polesel *et al.*, 2006. Intake of selected micronutrients and the risk of surgically treated benign prostatic hyperplasia: A case-control study from Italy. *Eur. Urol.*, 50: 549-554.
31. Key, T.J., P.N. Appleby, N.E. Allen, R.C. Travis and A.W. Roddam *et al.*, 2007. Plasma carotenoids, retinol and tocopherols and the risk of prostate cancer in the European prospective investigation into cancer and nutrition study. *Am. J. Clin. Nutr.*, 86: 672-681.
32. Watters, J.L., M.H. Gail, S.J. Weinstein, J. Virtamo and D. Albanes, 2009. Associations between α -tocopherol, β -carotene and retinol and prostate cancer survival. *Cancer Res.*, 69: 3833-3841.
33. Beilby, J., J.L. Ambrosini, E. Rossi, N.H. de Klerk and A.W. Musk, 2010. Serum levels of folate, lycopene, β -carotene, retinol and vitamin E and prostate cancer risk. *Eur. J. Clin. Nutr.*, 64: 1235-1238.
34. Bosetti, C., R. Talamini, M. Montella, E. Negri, E. Conti, S. Franceschi and C. La Vecchia, 2004. Retinol, carotenoids and the risk of prostate cancer: A case control study from Italy. *Int. J. Cancer*, 112: 689-692.
35. De Marzo, A.M., M.J. Putzi and W.G. Nelson, 2001. New concepts in the pathology of prostatic epithelial carcinogenesis. *Urology*, 57: 103-114.
36. Herzog, A., U. Siler, V. Spitzer, N. Seifert and A. Denelavas *et al.*, 2005. Lycopene reduced gene expression of steroid targets and inflammatory markers in normal rat prostate. *FASEB J.*, 19: 272-274.
37. Almushatat, A.S.K., D. Talwar, P.A. McArdle, C. Williamson and N. Sattar *et al.*, 2006. Vitamin antioxidants, lipid peroxidation and the systemic inflammatory response in patients with prostate cancer. *Int. J. Cancer*, 118: 1051-1053.