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RESEARCH PAPER

# Assessment of Lipid Peroxidation and Antioxidant Status in a Sample of Prostate Cancer Patients in Bangladesh

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## Abstract

***Background:*** Prostate cancer (PCa) is the second leading cause of cancer-related mortality in men. Oxidative stress has long been implicated in cancer development and progression.

***Objective:*** To evaluate the lipid peroxidation and antioxidant status in a sample of prostate cancer patients in Bangladesh.

***Methods:*** This case-control study included 207 histopathologically confirmed cases of prostate cancer and 200 age-matched healthy controls. After taking informed written consent, preset questioners were filled up, and about 5 ml of venous blood were collected with all aseptic precaution from each study subject for estimation of serum PSA, MDA, GST, SOD and erythrocyte reduced glutathione (GSH). All data were plotted in SPSS version 23, and different statistical analyses were done.

***Results:*** In this study, the mean age of cases was 67.27±8.28 years, and among control, it was 62.17±6.77 years. Oxidative stress marker malondialdehyde was found significantly increased in prostate cancer patients than control. On the other hand, antioxidant erythrocyte reduced glutathione (GSH), and superoxide dismutase (SOD) were significantly reduced, and glutathione S transferase (GST) activity was significantly increased in prostate cancer patients, compared to control group.

***Conclusion:*** This study revealed that overall oxidative stress was increased, and antioxidant levels were impaired in prostate cancer patients, which might play an important role in carcinogenesis. So, screening of oxidative stress and antioxidant status in the elderly male in a regular interval is recommended for early detection and proper management to prevent the aetiopathogenesis of prostate cancer.

**Keywords:** Prostate cancer, Lipid peroxidation, Antioxidant, Malondialdehyde

## Introduction

Prostate cancer (PCa) is the most common noncutaneous malignancy among men and is the second leading cause of cancer-related mortality.[1-3] Based on GLOBOCAN 2018 estimates, 1,276,106 new cases of prostate cancer were registered worldwide in 2018, representing 7.1% of all cancers in men.[2] Prostate cancer incidence rates are highly variable worldwide. African-American men have the highest incidence of prostate cancer and more likely to develop the disease earlier in life when compared to other racial and ethnic groups.[4] In Bangladesh, the prevalence of prostate cancer is low compared to the developed world, it has shown an increasing trend.[5] Despite the much higher incidence rate in more developed countries compared to less developed countries (69.5 vs 14.5), the differences in mortality data were cooperatively modest (10.0 vs 6.6).[6] Considering that medical care and assistance are not widely accessible in developing countries, this may cause high mortality despite the lower incidence.[7] The aetiology of prostate cancer is not well understood; however, studies examining genetics, diet, lifestyle, and certain chemicals exposure are increasingly attracting attention.[8] Epidemiological, experimental, and clinical studies suggested that oxidative stress (OS) plays a significant role in explaining prostate cancer development and progression.[9-11]

Oxidative stress, defined as an imbalance between reactive oxygen species (ROS) production and antioxidant defenses.[12-13] Men diagnosed with prostate cancer have been shown to have higher

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oxidative stress, lower antioxidant enzyme activity.[14] The antioxidant network comprises the enzymes superoxide dismutase (SOD), catalase, glutathione peroxidase, glutathione reductase and glutathione-s-transferase (GST) that play an important role in prostate cancer prevention, protecting cells from genomic damage mediated by carcinogens and ROS generated during inflammation. The expression of several enzymes involved in oxidative stress and detoxification is repressed in prostate cancer, in particular, glutathione S-transferase.[15] Furthermore; men with prostate cancer may be subject to greater oxidative stress and exhibit the lower activity of erythrocyte glutathione peroxidase and superoxide dismutase, compared with controls.[14]

Lipid peroxidation is a free-radical-mediated chain of reactions that, once initiated, results in an oxidative deterioration of polyunsaturated lipids, of which most common targets are components of the biological membrane.[16] The most frequently used biomarkers indicating the overall lipid peroxidation level is the plasma concentration of malondialdehyde (MDA).[17] Highly reactive aldehydes (MDA), products of lipid peroxidation, are capable of modifying both DNA and proteins, resulting in the mutagenic, genotoxic, and cytotoxic events.[18] Therefore, many of the literature reported that MDA levels were significantly high in patients with prostate cancer.[16]

Though several worldwide studies were done regarding the influence of oxidative stress on prostate cancer, limited data were found in Bangladesh. Therefore, the present study was aimed to investigate lipid peroxidation and antioxidant status in a sample of prostate cancer patients in Bangladesh.

## Materials and Methods

This cross-sectional study was conducted during. According to inclusion criteria, after taking informed written consent, 207 histopathologically diagnosed cases of prostate cancer, and 200 healthy subjects as control was taken from inpatient and outpatient department of urology, BIRDEM General Hospital, BSMMU, and Dhaka Medical College hospital. Detailed data regarding age, occupation, education, religion, family history of cancer, previous history of any cancer or chronic disease, smoking history was recorded in the preset questionnaire. About 05 ml of whole venous blood was collected with all aseptic precautions. 02 ml of whole blood was immediately collected in EDTA tube, mixed thoroughly for estimation of erythrocyte reduced glutathione (GSH) and SOD, and remaining 3 mL blood was collected to clot activator tube for serum separation and estimation of serum PSA, MDA, and GST. All the samples were stored at -20ºC till the biochemical analyses were done. Serum PSA was measured by automated immunoassay analyzer (ADVIA CENTAUR). Serum MDA was measured by the methods described by Rice and Anthony. MDA in the catabolite of lipid peroxide can react with thiobarbituric acid (TBA) and produce a red compound, which has a maximum absorption peak at 532 nm. The Glutathione S-Transferase (GST) Assay Kit utilizes 1-Chloro-2,4-dinitrobenzene (CDNB), which is suitable for the broadest range of GST isozymes. Upon conjugation of the thiol group of glutathione to the CDNB substrate, there is an increase in the absorbance at 340 nm. The increase in absorbance is directly proportional to the GST activity. The linearity of the reaction determined by plotting the absorbance values against time. The level of erythrocyte reduced glutathione was assayed by the method described by Beutler et al. The colorimetric substrate that reacts with the free thiol group of GSH to produce the highly colored product. The GSH concentration was then calculated as micromole per gram of hemoglobin by estimating hemoglobin concentration of whole blood. Measurements of SOD were performed by manual procedure of Bio Vision Assay Kit (Catalog#335-100 assays) and the absorbance was measured at a wave length of 560 nm with measurement unit U/mL. Statistical analyses were performed using the statistical package, SPSS version 23.0. Descriptive statistics were presented as Mean±SD. Differences in baseline variables between patients and control subjects were tested using Student's *t* test. $p<0.05$ was considered statistically significant. Pearsons correlation was used to find out the correlations between different parameters. Prior to the commencement of the study, the research protocol was approved by the National Ethics Review Committee of BMRC, Dhaka.

## Results

A total of 407 study subjects (207 cases and 200 controls) were enrolled in this study, according to the inclusion criteria. The age of the control was 62.17±6.77 years, and it was 67.27±8.28 years in

cases (table I). The study subjects were categorised into four different age group categories (<50, 50-60, 61-70, and >70 years). The majority (51.6%) of the cancer patients belong to 61-70 years age group (figure 1). BMI among control and cases was 24.07±1.95 and 23.06±2.17, respectively. Statistically, no significant differences were found between the cases and control regarding age and BMI. No statistically significant differences were also observed between the cases and control for the educational status, monthly income, and residential status. There were 52.0% and 60.8% smokers among the control and case group respectively but it was not statistically significant ($p$>0.05). Whereas, the family history of cancer among the study subjects revealed, only 4.5% of the control group had family history of cancer and, on the other hand, 18.4% of cases had positive family history of cancer and it was statistically significant.

Serum PSA, malondialdehyde (MDA), Glutathione S transferase (GST), erythrocyte reduced glutathione (GSH), and superoxide dismutase (SOD) were estimated in all the study subjects. Mean±SD of PSA (ng/mL), MDA (nmol/mL), erythrocyte reduced glutathione (GSH) (ìmol/gm of Hb), GST (nmol/mL/min) and SOD (U/mL) among the control were 3.06±0.49 mg/mL, 6.67±1.37 (nmol/mL), 2.51±0.77 (µmol/gm of Hb), 98.62±10.23 (nmol/mL/min) and 159.79±19.44 respectively, on the other hand, among the prostate cancer patients, those were 44.54±35.50 (ng/mL), 13.23±2.20 (nmol/mL), 1.74±0.89 (µmol/gm of Hb), 101.03±10.90 (nmol/mL/min) and 111.84±12.60 respectively (table II). Significant differences of biochemical parameters (including oxidative stress parameters) were found between the control and cases.

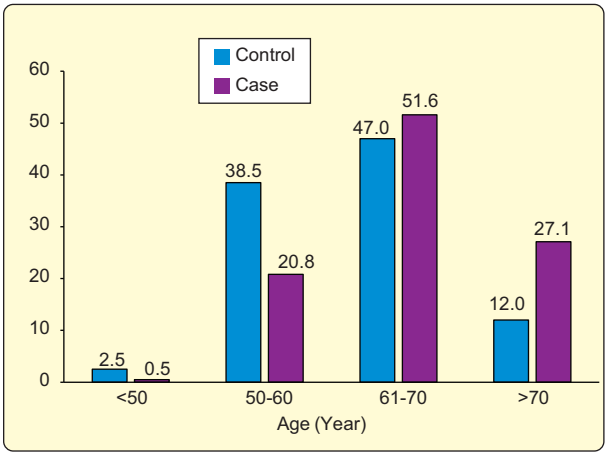


**Figure 1:** Frequency distribution of study subject according to age group.

PSA was significantly positively correlated with MDA & significantly negatively correlated with erythrocyte reduced Glutathione (GSH) and SOD. Significant negative correlation was also observed for MDA with GSH and SOD. No significant correlation was observed for other studied biochemical parameters (table III).

**Table I:** Baseline characteristics of study subjects

| Variables | Study subjects (n=407) | |
|---|---|---|
| | Control (n=200) | Case (n=207) |
| Age (years)† | 62.17 ± 6.77 | 67.27 ± 8.28 |
| BMI (kg/m$^2$)† | 24.07 ± 1.95 | 23.06 ± 2.17 |
| Occupation, n (%) | | |
| Dye factory | 06 (03) | 12 (5.8) |
| Farmer | 84 (42) | 91 (44.0) |
| Others | 110 (55) | 104 (50.2) |
| Educational status, n (%) | | |
| Illiterate | 13 (6.5) | 14 (6.8) |
| Primary | 84 (42) | 94 (45.4) |
| Secondary | 81 (40.5) | 82 (39.6) |
| Graduation/ above | 22 (11) | 17 (8.2) |
| Residence, n (%) | | |
| Rural | 103 (51.5) | 111 (53.6) |
| Urban | 97 (48.5) | 96 (46.4) |
| Smoking status | | |
| Nonsmoker | | |
| Smoker | 96 (48.0) | 81 (39.1) |
| | 104 (52.0) | 126 (60.9) |
| Family history of cancer | | |
| No | 191 (95.5) | 169 (81.6) |
| Yes | 09 (4.5) | 38 (18.4)* |

Results expressed as number (percentage); †Values are Mean±SD; BMI: Body mass index, *$p$<0.05 was taken as the level of significance.

**Table II:** Biochemical and oxidative stress parameters of study subjects (n=407)

| Variables | Control (n=200) | Case (n=207) |
|---|---|---|
| PSA (ng/ml) | 3.06 ± 0.49 | 44.54 ± 35.50** |
| MDA (nmol/ml) | 6.67 ± 1.37 | 13.23 ± 2.20** |
| Reduced Glutathione (GSH) (µmol/gm of Hb) | 2.51± 0.77 | 1.74 ± 0.89* |
| GST (nmol/ml/min) | 98.62±10.23 | 101.03± 10.90 |
| SOD (U/mL) | 159.79±19.44 | 111.84±12.60 |

Values are presented as Mean ±SD, p<0.05 was taken as the level of significance, PSA: Prostate specific antigen, MDA: Malondialdehyde, GSH: Erythrocyte reduced glutathione, GST: Glutathione S transferase, SOD: Superoxide dismutase.

**Table III:** Correlation among PSA, MDA, GSH, GST, and SOD

| Variable | PSA | MDA | GSH | GST activity | SOD |
|---|---|---|---|---|---|
| PSA | - | 0.539* | -0.233* | 0.035 | -0.529* |
| MDA | - | - | -0.400* | 0.094 | -0.697* |
| GSH | - | - | - | -0.003 | 0.349* |
| GST | - | - | - | - | -0.077 |
| SOD | - | - | - | - | - |

*Correlation is significant at the 0.01 level (2-tailed). PSA: Prostate specific antigen, MDA: Malondialdehyde, GSH: Erythrocyte reduced glutathione, GST: Glutathione S transferase, SOD: superoxide dismutase.

## Discussion

The mean age of prostate cancer cases was 67.27±8.28 year, and among control, it was 62.17±6.77 year (table I), which is similar to that of the age group reported by Iguchi et al. and Kosova et al.[23, 24] Most of the prostate cancer patients in this study, were of 61-70 years of age (figure-1), which is consistent with the previous study of Plaskon et al.[25] Age plays an important role in the development of prostate cancer, and its incidence increases with increased age.[26] This study also found a significant association of family history of cancer with the risk of prostate cancer. Similarly, Jr Rovito et al reported that the family history of prostate cancer in the first-degree blood relatives was a significant risk factor for prostate cancer.[27]

In accordance the present study, it was to be found that, men with prostate cancer had significantly high serum PSA compared to healthy men.[28,29] A study done described that PSA elevations during disease processes are believed to be a product of the disruption of the normal cellular architecture of the prostate gland.[30] Study of oxidative stress-related parameters in the study subjects revealed significant increased level of MDA and decreased level of erythrocyte reduced Glutathione (GSH), and SOD in the prostate cancer patients in comparison to controls. It was also found significant rise of MDA in prostate cancer patients due to increase generation of ROS.[11,25] In a study suggested that the lower GSH levels in prostate cancer may be due to the increased turnover of GSH for preventing oxidative damage.[31] Significant decreased level of SOD in prostate cancer patients were also reported.[32] In the present study, similar to GST activity was found significantly higher in prostate cancer patients than controls.[33] Rise in the levels of GST activity may attributed to its induction to counter the effect of increased oxidative stress.

The co-relational study of oxidative stress parameters and serum PSA found a significant positive correlation of serum PSA with MDA and significant negative correlation with GSH and SOD. Similar findings also reported by the other researchers.[19,34] In agreement with the findings of Srivastava and Mittal, significant negative correlation of MDA with GSH was also found in this study (Table III), which indicates the generation of more free radicals that may result in the destruction of protein structure or formation of DNA adduct.[33]

There are several limitations to this study like, the study had a relatively small sample size and consisted of only the Bangladeshi male race. The exact roles of oxidative DNA damage in the pathogenesis of disease were not completely defined in this study. Further studies on a large scale, more rigorous study design, especially stratified for gene-environment interaction, may eventually lead to a better comparative understanding of their possible role in prostate cancer.

## Conclusion

This study revealed that overall oxidative stress was increased in prostate cancer patients which may play an important role in the carcinogenesis. So, screening of oxidative stress and antioxidant status in the elderly male in a regular interval is recommended for early detection and proper management of prostate cancer.

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## Reference


1. Jernal A, Siegel R, Ward E, Hao Y, Xu J, Thun MJ. Cancer statistics, 2002. CA cancer J Clin. 2002;52:23-47. DOI: 10.3322/ca.2007.0010
2. Dianat SS, Margreiter M, Eckersberger E, Finkelstein J, Kuehas F, Herwig R, Ayati M, Lepor H, Djavan B. Gene polymorphisms and prostate cancer: the evidence. BJU International. 2009;104:1560-72. DOI: 10.1111/j.1464-410X.2009.08973.x
3. Bray F, Ferlay J, Soerjomataram I, Siegel RL, Torre LA, Jemal A. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: A Cancer Journal for Clinicians. 2018;68:394-424. DOI: 10.3322/caac.21492
4. Kheirandish P, Chinegwundoh F. Ethnic differences in prostate cancer. British Journal of Cancer. 2011;105: 481-5. Available From: www.nature.com/articles/bjc2011273
5. Salam MA. Early detection of Prostate Cancer Bangladesh Perspective. Journal of Bangladesh College of Physicians and Surgeons. 2014;32:89-93. Available From: www.banglajol.info/index.php/JBCPS/article/download/26037/17436
6. Wong MC, Goggins WB, Wang HH, Fung FD, Leung C, Wong SY, Ng CF, Sung JJ. Global incidence and mortality for prostate cancer: analysis of temporal patterns and trends in 36 countries. European Urology. 2016;70:862-74. DOI: 10.1016/j.eururo.2016.05.043
7. Rawla P. Epidemiology of prostate cancer. World Journal of Oncology. 2019;10:63. Available From: www.ncbi.nlm.nih.gov/pmc/articles/PMC6497009/
8. Hein DW, Grant DM, Sim E. Update on consensus arylamine N-acetyltransferase gene nomenclature. Pharmacogenetics and Genomics. 2000;10:291-2. Available From: journals.lww.com/jpharmacogenetics/toc/2000/06000
9. Lim HW, Hong S, Jin W, Lim S, Kim SJ, Kang HJ, Park EH, Ahn K, Lim CJ. Up-regulation of defense enzymes is responsible for low reactive oxygen species in malignant prostate cancer cells. Experimental & Molecular Medicine. 2005;37:497-506. Available From: www.nature.com/articles/emm200562
10. Venkataraman S, Jiang X, Weydert C, Zhang Y, Zhang HJ, Goswami PC, Ritchie JM, Oberley LW, Buettner GR. Manganese superoxide dismutase overexpression inhibits the growth of androgen-independent prostate cancer cells. Oncogene. 2005;24:77-89. Available From: www.nature.com/articles/1208145
11. Aydin A, Arsova-Sarafinovska Z, Sayal A, Eken A, Erdem O, Erten K, Özgök Y, Dimovski A. Oxidative stress and antioxidant status in non-metastatic prostate cancer and benign prostatic hyperplasia. Clinical Biochemistry. 2006;39:176-9. DOI: 10.1016/j.clinbiochem.2005.11.018
12. Sies HE. Hydroperoxides and thiol oxidants in the study of oxidative stress in intact cells and organs. Oxidative Stress. 1985;1:73-90 Available From: www.elsevier.com/books/oxidative-stress/sies/978-0-12-642760-8
13. Sies H. Oxidative stress: oxidants and antioxidants. Experimental Physiology: Translation and Integration. 1997;82:291-5. DOI: 10.1113/expphysiol.1997.sp004024.
14. Arsova-Sarafinovska Z, Eken A, Matevska N, Erdem O, Sayal A, Savaser A, Banev S, Petrovski D, Dzikova S, Georgiev V, Sikole A. Increased oxidative/nitrosative stress and decreased antioxidant enzyme activities in prostate cancer. Clinical Biochemistry. 2009;42:1228-35. DOI: 10.1016/j.clinbiochem.2009.05.009
15. Singal R, Van Wert J, Bashambu M. Cytosine methylation represses glutathione S-transferase P1 (GSTP1) gene expression in human prostate cancer cells. Cancer Research. 2001;61:4820-6. Available From: cancerres.aacrjournals.org/content/canres/61/12/4820.
16. Grotto D, Maria LS, Valentini J, Paniz C, Schmitt G, Garcia SC, Pomblum VJ, Rocha JB, Farina M. Importance of the lipid peroxidation biomarkers and methodological aspects for malondialdehyde quantification. Quimica Nova. 2009;32:169-74. DOI: 10.1590/S0100-40422009000100032
17. Nielsen F, Mikkelsen BB, Nielsen JB, Andersen HR, Grandjean P. Plasma malondialdehyde as biomarker for oxidative stress: reference interval and effects of life-style factors. Clinical Chemistry. 1997;43:1209-14. DOI: 10.1093/clinchem/43.7.1209
18. Merendino RA, Salvo F, Saija A, Di Pasquale G, Tomaino A, Minciullo PL, Fraccica G, Gangemi S. Malondialdehyde in benign prostate hypertrophy: a useful marker?. Mediators of Inflammation. 2003;12:127-8. DOI: 10.1080/0962935031000097745
19. Rice-Evans CA, Diplock AT, Symons MR. Techniques in free radical research. Laboratory Techniques in Biochemistry and Molecular Biology. 1991;22:1-278 DOI: 10.1016/0014-5793(92)81064-S.
20. Habig WH, Pabst MJ, Jakoby WB. Glutathione S-transferases. The first enzymatic step in mercapturic acid formation. J Biol Chem. 1974;249:7130-9. PMID: 4436300
21. Wilce MC, Parker MW. Structure and function of glutathione S-transferases. Biochim Biophys Acta. 1994 ;1205:1-18. DOI: 10.1016/0167-4838(94)90086-8. PMID: 8142473.
22. Beutler E, Duron O, Kelly BM. Improved method for the determination of blood glutathione. J Lab Clin Med. 1963;61:882-8. https://pubmed.ncbi.nlm.nih.gov/13967893/
23. Iguchi K, Hamatake M, Ishida R, Usami Y, Adachi T, Yamamoto H, Koshida K, Uchibayashi T, Hirano K. Induction of necrosis by zinc in prostate carcinoma cells and identification of

proteins increased in association with this induction. European journal of biochemistry. 1998 ;253:766-70. DOI: 10.1046/j.1432-1327.1998.2530766.x

24. Kosova B, Çetintaþ VB, Çal AÇ, Tetik A, Özel R, Aktan Ç, Gündüz C, Topçuoðlu N, CÜREKLÝBATIR ÝK, Eroðlu FZ. N-acetyltransferase 2 gene polymorphisms and susceptibility to prostate cancer: a pilot study in the Turkish population. Turkish Journal of Medical Sciences. 2010;40:629-936. Available From: dergipark.org.tr/en/download/article-file/128788
25. Plaskon LA, Penson DF, Vaughan TL, Stanford JL. Cigarette smoking and risk of prostate cancer in middle-aged men. Cancer Epidemiology and Prevention Biomarkers. 2003;12:604-9. https://cebp.aacrjournals.org/content/12/7/604.full
26. Malik SS, Masood N, Yasmin A. Prostate cancer and glutathione S-transferase deletions. EXCLI Journal. 2015;14:1049. DOI: 10.17179%2Fexcli2015-192
27. Rovito PM, Morse PD, Spinek K, Newman N, Jones RF, Wang CY, Haas GP. Heterocyclic amines and genotype of N-acetyltransferases as risk factors for prostate cancer. Prostate Cancer and Prostatic Diseases. 2005;8:69-74 https://www.nature.com/articles/4500780
28. Sivoòová M, Waczulíková I, Dobrota D, Matáková T, Hatok J, Raèay P, Kliment J. Polymorphisms of glutathione-S-transferase M1, T1, P1 and the risk of prostate cancer: a case-control study. Journal of Experimental & Clinical Cancer Research. 2009;28:1-8. https://link.springer.com/article/10.1186/1756-9966-28-32
29. Kaya E, Ozgok Y, Zor M, Eken A, Bedir S, Erdem O, Ebiloglu T, Ergin G. Oxidative stress parameters in patients with prostate cancer, benign prostatic hyperplasia and asymptomatic inflammatory prostatitis: A prospective controlled study. Advances in Clinical and Experimental Medicine. 2017;26:1095-9. DOI: 10.17219/acem/66837
30. Gretzer MB, Partin AW. PSA levels and the probability of prostate cancer on biopsy. European Urology Supplements. 2002;1:21-7. DOI: 10.1016/S1569-9056(02)00053-2
31. Surapaneni KM, Venkata GR. Lipid peroxidation and antioxidant status in patients with carcinoma of prostate. Indian Journal of Physiology and Pharmacology. 2006;50:350-4. Available From: europepmc.org/article/med/17402264
32. Kotrikadze N, Alibegashvili M, Zibzibadze M, Abashidze N, Chigogidze T, Managadze L, Artsivadze K. Activity and content of antioxidant enzymes in prostate tumors. Experimental Oncology. 2008. http://dspace.nbuv.gov.ua/handle/123456789/139908
33. Srivastava DS, Mittal RD. Free radical injury and antioxidant status in patients with benign prostate hyperplasia and prostate cancer. Indian Journal of Clinical Biochemistry. 2005;20:162-5. Available From: link.springer.com/article/10.1007/BF02867419
34. Duru R, Njoku O, Maduka I. Oxidative stress indicators in patients with prostate disorders in Enugu, South-East Nigeria. BioMed Research International. 2014;2014. DOI: 10.1155/2014/313015