Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(2), 1962-1972 | ISSN:2251-6727

Redox Dysregulation in Tobacco Consumers: Impact on Antioxidant Capacity, Liver Function, And Lipid Profile

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(Received: 16 January 2026 Revised: 25 February 2026 Accepted: 17 March 2026)

KEYWORDS

Tobacco consumption
Oxidative stress;
Total antioxidant status;
Lipid profile;
Liver enzymes;
FRAP assay

ABSTRACT:

Background: Tobacco consumption is a major source of oxidative stress due to increased production of free radicals, which may adversely affect antioxidant status, lipid metabolism, liver enzymes, and anthropometric indices.

Objective: To evaluate total antioxidant capacity and its association with anthropometric measures, and liver enzymes in tobacco users compared with healthy controls.

Methods: A cross-sectional study was conducted among 40 Indian tobacco consumers and 40 healthy volunteers. Waist and hip circumferences were measured, and Body Mass Index (BMI) was calculated. Biochemical parameters including lipid profile, liver enzymes, and total antioxidant capacity were assessed.

Results: Tobacco users and healthy volunteers showed a significant association with anthropometric indices, particularly waist and hip circumferences, whereas BMI was not significantly associated. Lipid profile parameters such as total cholesterol, triacylglycerol, LDL-C, and VLDL-C were significantly different between the two groups, while HDL-C showed no significant association. Alanine aminotransferase (ALT) and alkaline phosphatase (ALP) differed significantly using parametric analysis, whereas aspartate aminotransferase (AST) and total antioxidant capacity (TAC) assessed by the FRAP assay showed strong associations using non-parametric tests.

Conclusion: The findings indicate that tobacco usage is associated with adverse changes in antioxidant status, lipid profile, liver enzymes, and central adiposity. Antioxidant depletion in tobacco users may increase susceptibility to oxidative damage, contributing to accelerated aging and morbidity.

Introduction

Cigarette smoking is a major public health concern worldwide and one of the leading causes of early, preventable death. It negatively impacts human health in so many ways including cancer, atherosclerosis, and chronic obstructive pulmonary disease (1). Gupta et al reported nearly 23.7% of the deaths among men and 5.7% of the deaths among women aged 35–69 years are due to tobacco-attributable illnesses (2). It has been estimated that the smoking related mortality will rise to 10 million annually by 2030, with 70% of these deaths occurring in developing countries (3). Smoking generates a significant amount of reactive oxygen species, which affects cellular function and alters

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JCHR (2026) 16(2), 1962-1972 | ISSN:2251-6727

inflammatory indicators (4). Smoking is one of the main contributors of oxidative stress (5). Excessive synthesis of free radicals or a lack of antioxidant defense systems is considered to cause increased free radical activity (6). Reactive oxygen species come in a wide variety of forms, including hydroxyl radicals, superoxide anion radicals, hydrogen peroxide, etc. Cells are damaged when these react with membrane lipids, nucleic acids, proteins, enzymes, and small molecules (7). Smoking cigarettes for an extended period of time can lead to a systemic oxidant- antioxidant imbalance, as evidenced by an increase in the products of lipid peroxidation and a decrease in antioxidant levels. Total antioxidant capacity testing, reflects the plasma's antioxidative status, it might be more informative than measuring individual antioxidant levels in cells and plasma (8).

The low TAC levels can be a sign of oxidative stress or an increased vulnerability to oxidative damage (9).

Bello et al reported that decrease in serum antioxidants with a concomitant increase in Malondialdehyde (MDA) concentrations when compared to the control group. Cigarette smoking depletes many serum antioxidants required to scavenge excess free radicals, thus increasing the rate of lipid peroxidation (10). The results of the study by Jayaram et al showed that smokers had decreased levels of total antioxidant capacity. These results largely confirm the damaging effects of smoking on the body's antioxidant defense system and the development of oxidative stress due to smoking. Therefore, it is obvious that higher production of oxidants and free radicals is related to decreased antioxidant capacity in cigarette smokers (11).

Mohammed et al in their study estimated that increased total antioxidant capacity in smokers can validate the concept of the antioxidant system's compensatory mechanism, and higher levels of Thiobarbituric acid reactive substance in smokers can demonstrate the obvious risk of smoking and its chemical constituents (12). According to research by Ahmadkanika et al., both active and inactive smokers have lower antioxidant capacities than the control group (non-smokers). Smoking makes the body's antioxidant defense system less active and makes the oxidative stress system more active. These results make it evident that smoker's decreased antioxidant capacity is linked to their increased generation of oxidants and free radicals (13). The first fluid that comes in contact with cigarette smoke is saliva. The antioxidant system in the saliva is highly essential to its anti-cancer potential. The average total antioxidant capacity of saliva in smokers and non-smokers was significantly different. Based on the results, Fateme Arbabi-Kalati says et al smoking can reduce the total antioxidant capacity of saliva (14).

Growing evidence that tobacco dependency induces oxidative stress and may adversely influence body composition and metabolic health, it is important to understand how antioxidant status relates to anthropometric and biochemical characteristics in smokers compared with healthy individuals. Total antioxidant capacity reflects the cumulative effect of circulating antioxidants and may serve as an integrated marker of oxidative balance. However, data examining its relationship with body fat related indices and metabolic parameters in smokers remain limited.

Therefore, the present study was undertaken to investigate the association of total antioxidant capacity in smokers and healthy subjects, to evaluate its interrelationship with anthropometric indices in both groups, and to examine the combined associations among total antioxidant capacity, anthropometric measures and biochemical parameters in the study population.

Methods

Study participants were chosen based on the following eligibility criteria:

Inclusion Criteria:

Case Group – Tobacco users from 18 to 60 years of age attending tertiary care hospital.

Control Group – Healthy control of the age group 18 to 60 years.

Exclusion Criteria:

The subjects who have Type II Diabetes mellitus, Respiratory diseases, Cardiovascular diseases, Cancer, Renal dysfunction and other chronic illness were excluded.

Sample Collection and Processing:

3 ml of fasting venous blood sample was collected under aseptic precaution and medical supervision. The biochemical parameters assessed in both groups are Fasting Blood Glucose, Cholesterol, Triacylglycerols, HDL, LDL, VLDL, Total Protein, Albumin, Globulin, A/G Ratio, AST, ALT, ALP, Total Bilirubin, Direct Bilirubin, Indirect Bilirubin, Urea, Creatinine and Uric Acid.

Serum was used for all assays; samples containing precipitate were centrifuged, prior to performing the assays. Serum glucose was estimated by the glucose oxidase - peroxidase (GOD–POD) method, while total cholesterol was measured using the cholesterol oxidase - peroxidase technique. Triacylglycerols were analysed by an enzymatic lipase glycerol oxidation method, and HDL-cholesterol was determined following polyanion

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JCHR (2026) 16(2), 1962-1972 | ISSN:2251-6727

precipitation with enzymatic measurement of the supernatant fraction. LDL-cholesterol was calculated using Friedewald's equation [TC − HDL-C − (TAG/5)], and VLDL-cholesterol was estimated as TAG/5. Total serum protein was measured by the biuret method, albumin by the bromocresol green (BCG) dye - binding method, and globulin was derived by subtracting albumin from total protein. Aspartate aminotransferase and alanine aminotransferase activities were assessed by kinetic UV methods based on NADH oxidation, while alkaline phosphatase was estimated using p-nitrophenyl phosphate as substrate. Serum urea was measured by the urease - GLDH method, uric acid by the enzymatic uricase method, and creatinine by Jaffe's kinetic reaction. Total bilirubin was estimated by the Jendrassik and Gróf diazo method, direct bilirubin by a photometric diazo reaction using 2,4-dichloroaniline, and indirect bilirubin was calculated as the difference between total and direct bilirubin concentrations. Total antioxidant capacity of serum was assessed using the ferric reducing antioxidant power (FRAP) assay.

Anthropometric Measurements:

For anthropometric measurements, hip and waist circumference were noted. Waist Circumference (WC) was measured at the midpoint between the lower rib margin and iliac crest. Hip Circumference (HC) was measured at the widest point of the buttocks.

Results

Data were recorded using Microsoft Excel. Statistical analysis was performed using JASP (version 0.16). Categorical variables were expressed as percentages, while continuous variables were presented as mean ± standard deviation. Independent t - test was used to compare mean values between two groups, and the Mann - Whitney U test was applied for non-parametric data. Receiver operating characteristic (ROC) curve analysis was performed to determine the area under the curve (AUC) for evaluating the predictive utility of biochemical markers. An independent 't' - test was used to compare the data between two groups.

Independent t-test analysis revealed that waist circumference (WC) and hip circumference (HC) were significantly higher in the case group compared to controls (WC: 38.38 ± 3.28 vs. 35.32 ± 5.36 cm, $P = 0.003$; HC: 41.22 ± 3.38 vs. 37.68 ± 5.45 cm, $P = 0.001$). No significant difference was observed in body mass index (BMI) between groups ($P = 0.143$). Regarding lipid profile, cases exhibited markedly elevated total cholesterol (TC), triglycerides (TGL), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C) compared to controls (HDL-C) levels were comparable between groups ($P = 0.989$). These findings indicate a pattern of dyslipidemia in the case group.

Table 1 - Descriptive characteristics of anthropometric indices and biological parameters

Parameters	Mean ± SD (Control n=40)	Mean ± SD (Case n=40)	P value (< 0.05)
BMI	24.8685 ± 3.97134	26.1176 ±3.56607	0.143
WC	35.32 ± 5.361	38.38 ± 3.279	**0.003***
HC	37.68 ± 5.446	41.22 ± 3.378	**0.001***
FBS	85.75 ± 9.012	85.98 ± 10.199	0.917
TC	189.12 ± 21.585	235.82 ± 46.007	**0.000#**
TGL	100.18 ± 32.723	157.58 ± 71.387	**0.000#**
HDL	49.9 ± 11.712	49.86 ± 11.372	0.989
LDL	122.02 ± 23.262	156.1 ± 39.786	**0.000#**
VLDL	20.55 ± 6.763	31.58 ± 14.335	**0.000#**
ALT	26.6 ± 16.4	40.42 ± 24.306	**0.004***
ALP	69.07 ± 25.661	81.45 ± 20.346	**0.019***
Urea	21.6 ± 5.348	19.82 ± 6.733	**0.196**
Uric acid	4.95 ± 1.1719	4.85 ± 1.4836	**0.739**
Creatinine	1.1303 ± 0.14208	1.1452 ± 0.11934	**0.611**
TP	7.21 ± 0.3959	7.175 ± 0.4578	**0.719**
Albumin	4.425 ± 0.2933	4.328 ± 0.3566	**0.186**
Globulin	2.782 ± 0.2908	2.823 ± 0.4933	**0.66**
AGR	1.602 ± 0.2057	1.592 ± 0.3668	**0.881**

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BMI- Body mass index, WC-waist circumference, HC-Hip circumference, FBS- fasting blood glucose, TC - total cholesterol, TGL- triacylglycerol, HDL- high density lipoprotein, LDL- low density lipoprotein, VLDL- very low-density lipoprotein, ALT- alanine aminotransferase, ALP- alkaline phosphatase, TP- total protein, AGR- albumin globulin ratio.

* Indicating statistically significant p value <0.05, # indicating statistically significant p value <0.001

Independent t-test analysis revealed that waist circumference (WC) and hip circumference (HC) were significantly higher in the case group compared to controls (WC: 38.38 ± 3.28 vs. 35.32 ± 5.36 cm, $P = 0.003$; HC: 41.22 ± 3.38 vs. 37.68 ± 5.45 cm, $P = 0.001$). No significant difference was observed in body mass index (BMI) between groups ($P = 0.143$).

Regarding lipid profile, cases exhibited markedly elevated total cholesterol (TC), triglycerides (TGL), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C) compared to controls (HDL-C) levels were comparable between groups ($P = 0.989$). These findings indicate a pattern of dyslipidemia in the case group.

Liver function markers were also significantly altered, with higher alanine aminotransferase (ALT) ($p = 0.004$) and alkaline phosphatase (ALP) ($p = 0.019$) in cases, suggesting hepatic involvement. In contrast, fasting blood sugar (FBS), renal parameters (urea, uric acid, creatinine), and protein indices (total protein, albumin, globulin, albumin-to-globulin ratio) did not differ significantly between groups.

The case group demonstrated significantly greater central adiposity and dyslipidemia compared to controls, accompanied by elevated liver enzymes suggestive of hepatic stress. Notably, fasting glucose and renal function markers remained comparable between groups, highlighting a phenotype characterized by altered lipid metabolism and liver involvement rather than overt hyperglycemia or renal compromise.

Table 2: Comparison effect of Total antioxidant assay and biochemical parameters for case and control groups using Mann Whitney U test

Parameters	MEDIAN(IQR) Control n=40	MEDIAN(IQR) Case=40	P VALUE (< 0.05)
Total bilirubin	0.6(0.5,0.85)	0.6(0.5,0.85)	0.973
Direct bilirubin	0.2(0.3,0.3)	0.2(0.3,0.3)	0.62
Indirect bilirubin	0.4(0.3,0.6)	0.4(0.3,0.6)	0.741
AST	19.5(16,22.5)	24(21,33)	**0.000#**
FRAP	0.15(0.12.0.3)	0.13(0.11,0.16)	**0.036***

AST- aspartate aminotransferase, FRAP- ferric reducing antioxidant power assay

* indicating statistically significant p value <0.05, # indicating statistically significant p value <0.001

Median (IQR) values of bilirubin fractions (total, direct, and indirect) did not differ significantly between case and control groups, indicating that bilirubin metabolism remained largely unaffected. In contrast, aspartate aminotransferase (AST) levels were significantly elevated in the case group indicating hepatocellular stress or injury.

Furthermore, ferric reducing antioxidant power (FRAP) was significantly reduced in cases compared to controls reflecting diminished systemic antioxidant capacity.

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Table 3: Statistical difference in the case and control using ROC curve for predicting the biochemical parameters and ferric reducing antioxidant power assay.

Parameters	Cut off	Sensitivity	Specificity	Youden index	AUC ± SEM	95% CI	P value (<0.05)
TC	200.50	70%	70%	40%	0.808± 0.052	0.707-0.910	**0.000**#
TGL	109.50	75%	70%	45%	0.788±0.050	0.690-0.887	**0.000**#
LDL	33.50	70%	62.5%	32.5%	0.760±0.055	0.652-0.867	**0.000**#
VLDL	3.50	72.5%	70%	42.5%	0.771±0.052	0.668-0.873	**0.000**#
ALT	5.50	55%	60%	25%	0.698±0.058	0.585-0.812	**0.002***
ALP	5.50	62.5	57.5%	20%	0.658±0.061	0.538-0.777	**0.015***
AST	0.500	77.5%	55%	32.5%	0.736±0.055	0.628-0.844	**0.000**#
FRAP	.13650	60%	57.5%	17.5%	0.636±0.062	0.514-0.758	**0.036***

TC-total cholesterol, TGL- triacylglycerol, LDL- low density lipoprotein, VLDL- very low-density lipoprotein, ALT-alanine aminotransferase, ALP-alkaline phosphatase, AST-aspartate aminotransferase, FRAP- ferric reducing antioxidant power assay, AUC- area under curve, SEM-standard error of mean, CI-confidence interval.

*indicating statistically significant p value <0.05, # indicating statistically significant p value <0.001

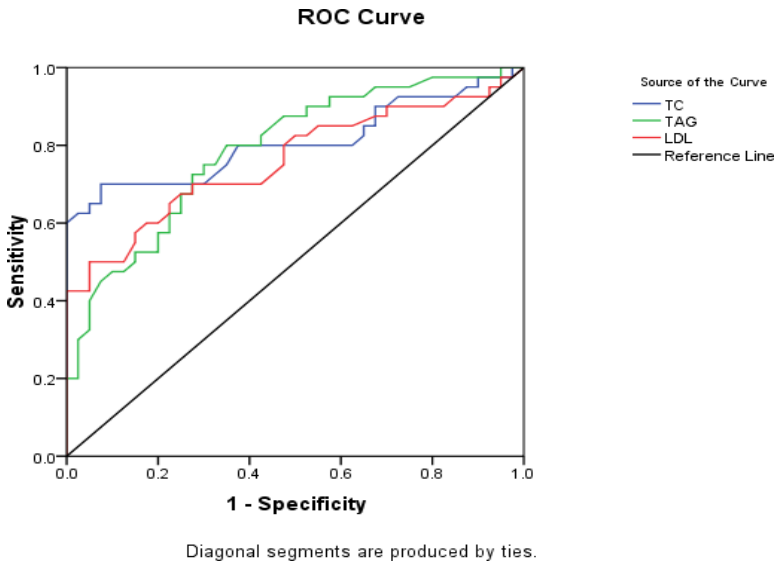

Figure.1 - ROC curve for the parameters based on cut off, sensitivity and specificity with area under curve values for TC, TGL and LDL as 0.808, 0.788, 0.760 respectively.

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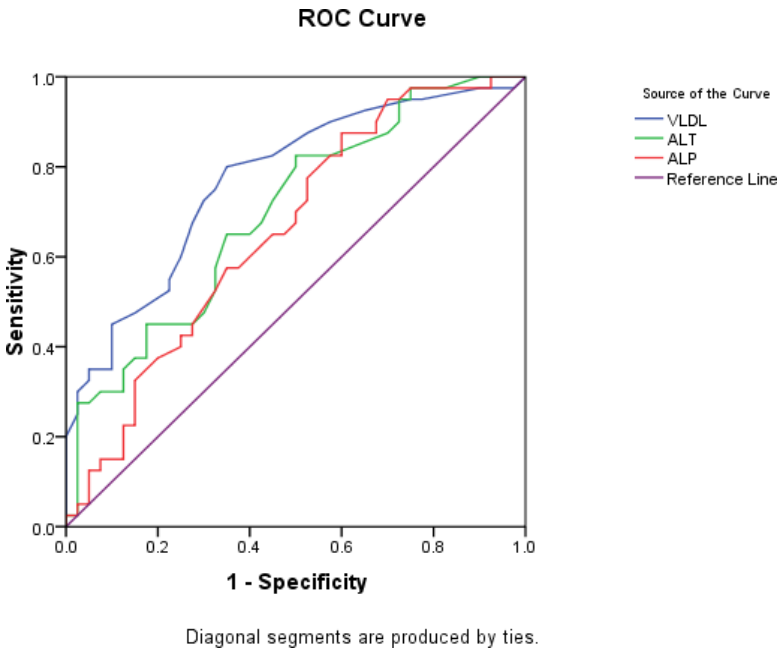

Figure. 2 - ROC curve for the parameters based on cut off, sensitivity and specificity with area under curve values for VLDL, ALT and ALP as 0.771, 0.585, 0.658 respectively.

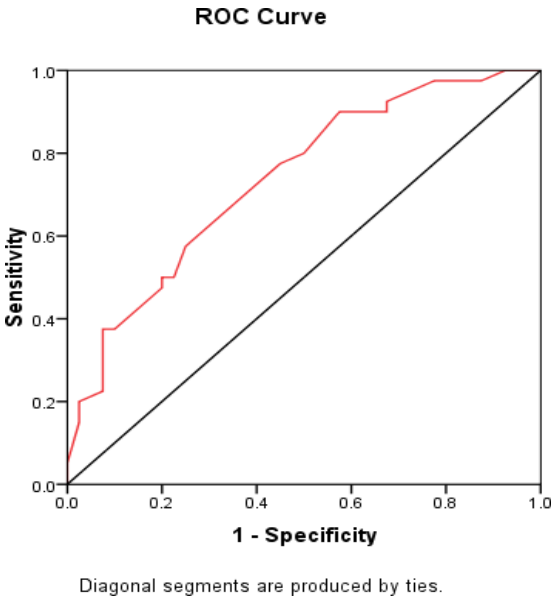

Figure.3 - ROC curve for the parameter based on cut off, sensitivity and specificity with area under curve values for AST as 0.736 respectively.

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In Fig.1, TC shows poor discrimination (AUC 0.608), while TGL and LDL show good performance (AUCs ~0.76), supporting their role in identifying lipid abnormalities.

Fig.2 shows ALT stands out with excellent discrimination (AUC 0.853), confirming its utility as a liver stress marker. VLDL also performs well (AUC 0.771), indicating its relevance in metabolic risk. ALP shows moderate accuracy (AUC 0.683), suggesting supportive but not standalone diagnostic value. Fig.3 indicates AST curve (AUC 0.736) reflects reliable but less robust liver injury detection compared to ALT. In fig.4, FRAP curve (AUC 0.636) shows limited diagnostic utility, despite biological relevance in oxidative stress.

The ROC analysis confirms that ALT, VLDL, TGL, LDL, and AST are the most effective markers for distinguishing cases from controls, with ALT showing the highest diagnostic accuracy. These findings align with earlier results showing elevated liver enzymes and dyslipidemia in the case group.

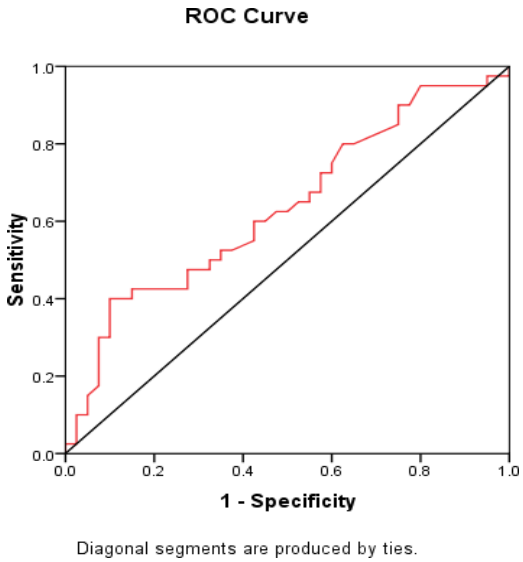

Figure.4 - ROC curve for the parameter based on cut off, sensitivity and specificity with area under curve values for FRAP as 0.636 respectively.

Discussion

In our study, Total antioxidant assay is associated with anthropometric indices in tobacco users. BMI was not associated with total antioxidant assay but waist and hip circumference are strongly accompanied. Thus, total antioxidant assay is inversely proportional in tobacco consumers when compared with healthy control in association of anthropometric indices specially waist hip circumferences.

According to the results of Reejamol et al reported that smoking may produce free radicals that increase the activity of reactive oxygen species (ROS) and have an impact on the levels of lipid peroxide, Thiobarbituric acid reactive substance (TBARS), total thiol, and catalase. As a result of an increase in free radicals, smokers may have lower levels of antioxidants such reduced glutathione and superoxide dismutase in their bodies (15). According to these findings, Mojtaba et al concluded that reduced antioxidant capacity in cigarette smokers is associated with increased production of oxidants and free radicals (16).

According to this study Bloomer et al estimated that smoker's Malondialdehyde (MDA) levels were increased and their antioxidant reducing capacity was lower than non-smokers. Smokers had somewhat lower levels of both total glutathione and Trolox equivalent antioxidant capacity (TEAC), but there was no statistically significant difference between them and non – smokers (17). Eugene et al reported that the smokers had a greater average reactive oxygen metabolites (ROM) concentration than non-smokers, but a higher total oxidant status (TOS) concentration. Average serum concentrations of uric acid, the principal antioxidant in serum and a significant contributor to the serum's antioxidant status, decreased in smokers, while average

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serum concentrations of biological antioxidant potential (BAP), Ferric reducing antioxidant power assay (FRAP), and total antioxidant status (TAS), reduced with smoking. Smoking had no effect on the redox status biomarker. The total thiol levels (TTL). These findings lead to the conclusion that when applying oxidative stress and antioxidant indicators in epidemiological investigations, the impact of smoking should be taken into consideration (18).

A study by Chauhan et al indicates that tobacco use enhances oxidative stress, as reflected by increased MDA levels and reduced antioxidant enzymes SOD and GPx, suggesting an imbalance between oxidants and antioxidants. The observed dose–response relationship with duration and frequency of use supports a cumulative harmful effect of tobacco exposure (19). Graff-Iversen et al reported that the consistent negative associations of smoking with increased hip circumference. In perspective of research concluding that a higher percentage gluteofemoral fat is linked with lower cardiovascular disease risk, our results suggest that tobacco intake could be a modifying cardio vascular disease risk factor through mechanisms that reduce the capacity of fat storage in the lower body region (20).

Herath et al reported that smoking exposure resulted in increased Total cholesterol, Low density lipoprotein, very low- density lipoprotein, Triacylglycerol and decreased High density lipoprotein levels. The risk of a rise in serum cholesterol with an increase in low density lipoprotein in smokers is very important since it is associated with coronary heart disease (21).

The descriptive characteristics of anthropometric indices and biochemical parameters between smokers and non-smokers are measured by independent t- test. The mean value of the parameters such as waist circumference (p value - 0.003), hip circumference (p value- 0.001), total cholesterol (p value 0.000), triacylglycerol (p value - 0.000), low density lipoprotein (p value 0.000), very low-density lipoprotein (p value - 0.000), alanine aminotransferase (p value 0.004) and alkaline phosphatase (p value - 0.019) were significantly high in case (p value < 0.005) compared to control, but there was no significantly difference between mean value of body mass index, fasting blood glucose, HDL, urea, creatinine ,uric acid , total protein, albumin, globulin, albumin globulin ratio between healthy control and tobacco users.

The non-parametric test for the parameters Bilirubin, AST and FRAP by Mann-Whitney U test between non-smokers and smokers as shown in table 2. The median value for parameters such as aspartate aminotransferase (p value - 0.000) and Ferric reducing antioxidant power assay (p value - 0.036) were significant in non-smokers compared to smokers (p value < 0.005) but there was no significance between total bilirubin, direct bilirubin, indirect bilirubin.

ROC curve as shown in table 3 with cut off, sensitivity, specificity, Youden index, Area under curve, standard error of mean, confidence interval values of total cholesterol l(p value - 0.000), triacylglycerol (p value - 0.000), low density lipoprotein (p value - 0.000), very low-density lipoprotein (p value - 0.000), Alanine aminotransferase (p value - 0.002), Alkaline phosphatase (pvalue - 0.015), Aspartate aminotransferase(p value - 0.000), Ferric reducing antioxidant power Assay (p value - 0.036), Area under curve, Standard error of mean, Confidence interval are highly significant with the P value (<0.005).

A study by Khaled Salem Alsalhen et al estimated that smokers have higher plasma concentrations of Malondialdehyde (MDA) compared with non-smokers. Heavy smoking was associated with low total protein, globulin and albumin levels and raised AST, ALT and ALP levels. The levels of cholesterol and triglycerides were significantly increased in all smokers as compared to non- smokers. Cigarette smoking decreases serum bilirubin is effective in detecting free radicals produced by smoking, which contribute to serious diseases. Thus, our study concludes that smokers are at higher risk of lipid peroxidation and thus production of reactive oxygen species (free radicles) that may induce micro and macrovascular complications than that of non – smokers (22).

Agarwal et al showed Tobacco use induces oxidative stress, leading to decreased SOD and CAT activity while GPx was increased as a compensatory adaptive mechanism (23). The alterations observed in lipid profile, liver enzymes, and antioxidant status among smokers in the present study are in agreement with findings reported by Preethi et al., demonstrated significant tobacco-induced changes in thyroid and hepato-biliary biomarkers. These observations further support the role of cigarette smoking in inducing systemic biochemical disturbances through oxidative stress - mediated mechanisms (24).

The reduced total antioxidant capacity observed among smokers in the present study supports the role of cigarette

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smoking in enhancing oxidative stress and lipid peroxidation. Similar protective effects of antioxidants have been reported from our previous study which demonstrated that caffeic acid significantly reduced lipid peroxidation and improved antioxidant defense in an experimental rat model. Together, these findings suggest that antioxidant depletion contributes to smoking-related biochemical alterations and that antioxidant-rich diets may help mitigate oxidative damage in smokers (25).

Several studies report that cigarette smoking is associated with increased systemic oxidative stress and reduced antioxidant defenses, reflected by lower total antioxidant status and higher oxidative stress indices (26–28). Smokers also exhibit exaggerated oxidative responses following metabolic challenges such as high-fat meals, accompanied by reductions in antioxidant capacity (29). Decreased concentrations of vitamins C and E and impairment of enzymatic antioxidant systems including superoxide dismutase, catalase, and glutathione-related pathways have been described in smokers compared with non-smokers (30). Oxidative imbalance has also been observed in saliva and other biological fluids, indicating widespread redox perturbations even among apparently healthy smokers, while smoking related diseases appear to intensify oxidative injury and suppress antioxidant capacity (31, 32). Population and biochemical studies further suggest that smoking-associated oxidative stress frequently coexists with dyslipidaemia and other cardiometabolic alterations, linking redox imbalance with anthropometric and metabolic risk (33).

CONCLUSION:

The study suggests that tobacco consumption is associated with central adiposity, hepatic enzyme elevation, and reduced antioxidant capacity, even in the absence of overt hyperglycemia or renal dysfunction. ROC analysis underscores ALT as the most reliable early biomarker of smoking-related hepatic stress, with AST and ALP contributing additional diagnostic value. The oxidative imbalance indicated by FRAP further highlights the vulnerability of smokers to oxidative damage, though its predictive utility is modest.

These findings emphasize hepatic involvement and oxidative imbalance as early consequences of tobacco usage, underscoring the importance of targeted screening and preventive strategies in this population. Individuals dependent on tobacco might benefit from consuming antioxidant supplements, or a diet rich in fruits and vegetables, which can reduce the oxidative stress.

LIMITATIONS OF THE STUDY:

- Tobacco usage was based on self-report which introduces the possibility of misclassification due to recall bias or underreporting.
- The scope of biomarkers assessed was also limited, with oxidative stress measured only by FRAP and without inclusion of other relevant markers such as malondialdehyde, glutathione, or inflammatory cytokines, thereby narrowing the mechanistic insight.
- Potential confounding factors including diet, alcohol consumption, physical activity, and co-morbid conditions were not recorded, as these could have influenced lipid profile, liver enzymes, and antioxidant levels.

FUTURE RECOMMENDATIONS:

Future studies should employ larger, longitudinal cohorts with validated measures of tobacco exposure, incorporating a broader panel of oxidative stress and inflammatory biomarkers, and adjusting for lifestyle and clinical confounders. Such approaches would provide deeper mechanistic insights and enhance the translational relevance of smoking-related biochemical alterations in diverse populations.

FUNDING:

No funding was received for the study.

ACKNOWLEDGEMENTS:

The Authors thank Prof. Seetesh Ghose, Dean, Mahatma Gandhi Medical College and Research Institute, a constituent teaching unit of Sri Balaji Vidyapeeth, Dr. K. S. Reddy, Dean Research, Sri Balaji Vidyapeeth and Dr. Nihar Ranjan Biswas, Vice – Chancellor, Sri Balaji Vidyapeeth for their support and patronage in enabling this research communication.

REFERENCES:

1. V. Andure, D.; M. Raut, A.; K. Padalkar, R.; M. Patil, S.; S. Bhagat, S. Study of Interaction of Cigarette Smoke with Thiol (-SH) Group of Sulfhydryl Proteins in Smokers. International Journal of Clinical Biochemistry and Research **2019**, 6 (1), 25–28.

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www.jchr.org

JCHR (2026) 16(2), 1962-1972 | ISSN:2251-6727

2. Gupta, P. C.; Pednekar, M. S.; Parkin, D.; Sankaranarayanan, R. Tobacco Associated Mortality in Mumbai (Bombay) India. Results of the Bombay Cohort Study. International Journal of Epidemiology **2005**, 34 (6), 1395–1402.
3. Fagerstrom, K. The Epidemiology of Smoking. Drugs **2002**, 62 (Supplement 2), 1–9.
4. Shirzaiy, M.; Aiub Rigi Ladiz, M.; Dalirsani, Z.; Dehghan Haghighi, J.; Nakhaii, A. Evaluation of Salivary Total Antioxidant Capacity in Smokers with Severe Chronic Periodontitis. International Journal of High Risk Behaviors and Addiction **2017**.
5. Kahnamoei, J.; Rahmani, F.; Maleki, M. R.; Nasirzadeh, F.; Kishizadeh, F. The Effects of Cigarette Smoking on Plasma MDA and TAC in University Students. *Bull. Environ. Pharmacol. Life Sci.* **2014**, *3*, 95–98.
6. Rao, G. M.; Sumita, P.; Roshni, M.; Ashtagimatt, M. N. Plasma Antioxidant Vitamins and Lipid Peroxidation Products in Pregnancy Induced Hypertension. Indian Journal of Clinical Biochemistry **2005**, 20 (1), 198–200.
7. Jayachandra, S.; Selvaraj, R.; Agnihotram, G. Determination of Serum Total Antioxidant Capacity in Male Smokers and Non-Smokers. National Journal of Physiology, Pharmacy and Pharmacology **2017**, 1.
8. Buico, A.; Cassino, C.; Ravera, M.; Betta, P.-G.; Osella, D. Oxidative Stress and Total Antioxidant Capacity in Human Plasma. Redox Report **2009**, 14 (3), 125–131.
9. Young, I. S. Measurement of Total Antioxidant Capacity. Journal of Clinical Pathology **2001**, 54 (5), 339.
10. Hauwa'u, A. B.; Abdullahi, D.; Gaddafi, I. D. Effect of Cigarette Smoking on Lipid Peroxidation and Serum Antioxidant Vitamins. *J. Pharm. Biol. Sci.* **2017**, *12*, 40–44.
11. Gadham, J.; Prasanna, M. L. A Comparative Study of Serum Total Antioxidant Capacity (TAC) in Male Smokers and Non Smokers. International Journal of Physiology **2017**, 5 (1), 81.
12. Moballegholeslam, M.; et al. Comparison of Thiobarbituric Acid Reacting Substances and Total Antioxidant Capacity in Saliva of Smokers and Nonsmokers. *Caspian J. Dent. Res.* **2018**, *7*, 44–48.
13. Ahmadkhaniha, R.; Yousefian, F.; Rastkari, N. Impact of Smoking on Oxidant/Antioxidant Status and Oxidative Stress Index Levels in Serum of the University Students. *J. Environ. Health Sci. Eng.* **2021**, *19* (1), 1043–1046.
14. Arbabi-Kalati, F.; Nosratzehi, T.; Salimi, S.; Sadeghi Sabzevari, R.; Arbabi-Kalati, P. Comparison of Total Antioxidant Capacity of Saliva in Smokers and Non-Smokers. J. Mashhad Dent. Sch. **2014**, 38 (2), 93–98.
15. Reejamol, M. K.; Swaminathan, M. Estimation of Lipid Peroxides and Antioxidants in Smokers and Non-Smokers with Periodontitis. King Saud University Journal of Dental Sciences **2013**, 4 (2), 53–56.
16. Eizadi, M.; Khorshidi, D.; Dooaly, H. Lower Total Antioxidant Capacity in Smokers Compared to Non-Smokers. *Biol. Forum* **2014**, *6*, 305–309.
17. Bloomer, R. J. Decreased Blood Antioxidant Capacity and Increased Lipid Peroxidation in Young Cigarette Smokers Compared to Nonsmokers: Impact of Dietary Intake. Nutrition Journal **2007**, 6 (1).
18. Jansen, E.; Beekhof, P.; Ruskovska, T. The Effect of Smoking on Biomarkers of (Anti)Oxidant Status. *J. Mol. Biomark. Diagn.* **2014**, *5*, 1000152.
19. Chauhan, P.; Reddy, S. S.; Chokkanna, V. K.; Singh, P.; Majumdar, K. Oxidant and Antioxidant Status among Tobacco Users: A Cross-Sectional Study. National Journal of Maxillofacial Surgery **2023**, 14 (3), 444–449.
20. Graff-Iversen, S.; Hewitt, S.; Forsén, L.; Grøtvedt, L.; Ariansen, I. Associations of Tobacco Smoking with Body Mass Distribution; a Population-Based Study of 65,875 Men and Women in Midlife. BMC Public Health **2019**, 19 (1).
21. Herath, P.; Wimalasekera, S.; Amarasekara, T.; Fernando, M.; Turale, S. Adverse Effects of Cigarette Smoking on Exhaled Breath Carbon Monoxide, Blood Carboxyhemoglobin, and Hematological Parameters amongst Sri Lankan Adult Tobacco Smokers: A Descriptive Study. Population Medicine **2021**, 3 (October), 1–10.
22. Alsalhen, K. S.; Abdalsalam, R. D. Effect of Cigarette Smoking on Liver Functions: A Comparative Study Conducted among Smokers and Non-Smokers Male in El-Beida City, Libya. International Current Pharmaceutical Journal **2014**, 3 (7), 291–295
23. Agarwal, P.; Bagewadi, A.; Keluskar, V.; Vinuth, D. P. Superoxide Dismutase, Glutathione Peroxidase, and Catalase Antioxidant Enzymes in Chronic Tobacco Smokers and Chewers: A Case–Control Study. *Indian J. Dent. Res.* **2019**, *30* (2), 234–240
24. Preethi, K.; Dhanavathi, T.; Rajendran, J. Tobacco-Induced Alterations in Thyroid and Hepato-Biliary

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JCHR (2026) 16(2), 1962-1972 | ISSN:2251-6727

Biomarkers: A Cross-Sectional Insight. *Zhuzao* **2025**, *28* (10), 366–377.

25. Jayanthi, R.; Subash, P. Antioxidant Effect of Caffeic Acid on Oxytetracycline Induced Lipid Peroxidation in Albino Rats. *Indian J. Clin. Biochem.* **2010**, *25* (4), 371–375.
26. Karademirci, M.; Kutlu, R.; Kilinc, I. Relationship between Smoking and Total Antioxidant Status, Total Oxidant Status, Oxidative Stress Index, Vit C, Vit E. *Clin. Respir. J.* **2018**, *12* (6), 2006–2012.
27. Lykkesfeldt, J.; Viscovich, M.; Poulsen, H. E. Plasma Malondialdehyde Is Induced by Smoking: A Study with Balanced Antioxidant Profiles. *Br. J. Nutr.* **2004**, *92* (2), 203–206.
28. Kocyigit, A.; Erel, O.; Gur, S. Effects of Tobacco Smoking on Plasma Selenium, Zinc, Copper and Iron Concentrations and Related Antioxidative Enzyme Activities. *Clin. Biochem.* **2001**, *34* (8), 629–633.
29. Bloomer, R. J.; Solis, A. D.; Fisher-Wellman, K. H.; Smith, W. A. Postprandial Oxidative Stress Is Exacerbated in Cigarette Smokers. *Br. J. Nutr.* **2008**, *99* (5), 1055–1060.
30. Nsonwu-Anyanwu, A.; Offor, S.; John, I. Cigarette Smoke and Oxidative Stress Indices in Male Active Smokers. *Reactive Oxygen Species* **2018**, *5* (15), 199–208.
31. Ahmadi-Motamayel, F.; Falsafi, P.; Abolsamadi, H.; Goodarzi, M. T.; Poorolajal, J. Evaluation of Salivary Antioxidants and Oxidative Stress Markers in Male Smokers. *Comb. Chem. High Throughput Screen* **2019**, *22* (7), 496–501.
32. Pandi, A.; Lal, V.; Chakraborty, B.; Kalappan, V. M. Evaluation of Oxidative Stress and Antioxidant Biomarkers in Chronic Cigarette Smokers: A Pilot Study. *Cureus* **2024**, *16* (5), e60629.
33. Pasupathi, P.; Saravanan, G.; Farook, J. Oxidative Stress Biomarkers and Antioxidant Status in Cigarette Smokers Compared to Nonsmokers. *J. Pharm. Sci. Res.* **2009**, *1* (3), 55–58.