Original Article

DOI: 10.7860/JCDR/2022/55196.16223

# Evaluation of Lipid Profile and Antioxidant Status in Hypertensive Smokers: A Case-control Study

Biochemistry Section

SYED SADATH ULLA[1], VIVIAN D'SOUZA[2], BHAGYAJYOTHI M BHAT[3]



## ABSTRACT

**Introduction:** Hypertension and smoking are two independent risk factors for oxidative stress and dyslipidaemia, thereby development of cardio and cerebrovascular diseases are common. The effect of smoking on oxidants, antioxidants and lipid profile in hypertensive individuals is the matter of concern.

**Aim:** To compare fasting serum lipid profile, Glutathione (GSH) and Thiobarbituric Acid Reactive Substances (TBARS) of hypertensive smokers and hypertensive non smokers.

**Materials and Methods:** This case-control study was conducted in the Department of Biochemistry, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India, between September 2021 to December 2021. The study population consisted of 58 hypertensive smokers and 58 hypertensive non smokers visiting the Department of Internal Medicine, tertiary care hospital. Height, weight, smoking intensity, family history of hypertension was recorded, fasting serum lipid profile, GSH and TBARS were estimated. Smoking intensity was expressed as pack-years. Body Mass Index (BMI) was calculated using the measured height and weight. The data were analysed using Chi-square test, independent sample t-test and Pearson's correlation.

**Results:** The mean age of cases were 40.38±14.96 years whereas among controls, it was 45.98±14.96 years. Among cases, 50 were males and eight were females, whereas among the controls, 47 were males and 11 were females. Out of total 58 subjects in each group, 43 in cases and 45 in controls had a family history of hypertension. A significant increase in the levels of serum TC, TAG, LDL-C, TBARS and a significant decrease in GSH, BMI in cases compared to the control ($p<0.001$) was observed. Total Cholesterol (TC), Triacylglycerol (TAG), Low Density Lipoprotein- Cholesterol (LDL-C) and TBARS exhibited a significant positive correlation whereas, BMI, HDL-C and GSH showed a negative correlation with pack-years.

**Conclusion:** Smoking was found to be significantly associated with dyslipidaemia and oxidative stress in hypertensive individuals. It was observed that the dyslipidaemia and oxidative damage was correlated with the pack-years in hypertensive smokers.

**Keywords:** Cholesterol, Cigarette, Glutathione, Hypertension, Malondialdehyde, Triacylglycerol

## INTRODUCTION

Hypertension is a major growing global health issue leading to mortality by being one of the causative factors for various disorders such as cardiovascular disease, renal disease, metabolic syndrome, diabetes mellitus, preeclampsia and many more [1]. Hypertension causes upregulation of the renin-angiotensin-aldosterone system, inflammatory response, activation of the immune system and increased oxidative stress which may end up in both micro and macrovascular complications [2]. Studies showed that reactive oxygen species have a considerable role to play in the pathophysiology of hypertension [3,4]. There are pieces of evidence that elevated lipid peroxidation indicated by Malondialdehyde (MDA) which is estimated as TBARS and dyslipidaemia are observed in hypertensive individuals [3,5,6].

Cigarette smoking is a commonly observed habit in individuals of both genders regardless of socio-economic status. The risk of several complications like cerebral and cardiovascular diseases increases with cigarette smoking [7]. The mechanism involved in the development of coronary heart diseases by cigarette smoke is not yet very clear [8]. Dyslipidaemia was observed in smokers and the degree of dyslipidaemia showed a dose-dependent relationship with the grades of smoking [9-11]. Smoking was found to affect the oxidant, antioxidant status of the body, where the serum antioxidant levels, such as that of vitamin A, E, C reduced GSH and superoxide dismutase were lowered whereas the extent of lipid peroxidation was increased, the product being MDA, which is measured as TBARS [12]. Earlier studies documented that smoking decreases the body weight [13-15]. Authors obtained sufficient materials on the effect of smoking and hypertension separately on general population, to the best of the knowledge, the effect of smoking on lipid profile and antioxidant status in hypertensive individuals has not been documented. Knowing the ill-effects of smoking, the effect of the same on hypertension and its complications is a matter of concern. Thus, the present study was aimed to compare the levels of serum MDA, GSH and lipid profile of hypertensive smokers and hypertensive non smokers.

## MATERIALS AND METHODS

This case-control study was conducted in the Department of Biochemistry, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India, between September 2021 to December 2021. Ethical clearance was obtained from the Institutional Ethical Committee (IEC) before starting the project work (IEC/31-08-2021).

**Inclusion criteria:** The study included hypertensive individuals visiting the Outpatient Department (OPD) having a Systolic Blood Pressure (SBP) >140 mmHg or Diastolic Blood Pressure (DBP) >90 mmHg [16], of either sex, aged between 18-65 years. Cases included hypertensive smokers who have been smoking atleast from past one year. The controls were age and sex-matched hypertensive non smokers.

**Exclusion criteria:** Hypertensive individuals who quit smoking and female subjects diagnosed with gestational hypertension were excluded.

**Sample size:** Sample size for the present study was calculated using the formula: $n=\{2(Z_{1-\alpha}+Z_{1-B})^2\sigma^2\}/d^2$, Where, $Z_{1-\alpha}=1.96$ for 95% confidence interval, $Z_{1-B}=0.84$ for 80% statistical power, $\sigma=3.92$ is anticipated standard deviation [3], d=1.22 is acceptable margin of

error. The minimum sample size was estimated to be 58 subjects each in case and control group. Non probability convenience sampling technique was used.

The study subjects were explained about the protocol, informed consent was obtained from the willing individuals. Personal information such as the age, blood pressure, smoking habit, frequency of smoking, past history of illness was collected by interacting with the individuals. A general physical examination including measurement of height and weight of the individual was conducted and the findings were noted. The serum lipid profile estimation included TC, TAG, LDL-C, High Density Lipoprotein Cholesterol (HDL-C) and Very Low Density Lipoprotein Cholesterol (VLDL-C). Serum TBARS and GSH were also estimated.

Serum TC, TAG, LDL-C, HDL-C were estimated using appropriate kits from ortho-clinical diagnostics in dry-chemistry analyser, Vitros 5600, as per the standard procedures mentioned in the kits. VLDL-C was calculated using the formula, VLDL-C=1/5×TAG [17].

TBARS was estimated as a measure of MDA [18] and GSH was estimated by its reaction with 5,5'-dithio nitrobenzoic acid [19] using UV/Vis analyser Labotech from BD instrumentation using standard procedures. The biological reference intervals of the lipid profile parameters in the central clinical laboratory are given in [Table/Fig-1].

| Parameter | Test | Normal range |
|---|---|---|
| TC | Cholesterol oxidase peroxidase method | <200 mg/dL |
| TAG | Lipase-glycerol kinase method | <150 mg/dL |
| LDL-C | Precipitation method | <100 mg/dL |
| HDL-C | Precipitation method | 40-59 mg/dL |
| VLDL-C | Calculated by the formula | 2-30 mg/dL |

**[Table/Fig-1]:** Reference intervals of the biochemical parameters.
TC: Total cholesterol; TAG: Triacylglycerol; LDL-C: Low density lipoprotein-cholesterol; HDL-C: High density lipoprotein-cholesterol; VLDL-C: Very low density lipoprotein-cholesterol

Using the measured values of height and weight, BMI was calculated using the formula, BMI=W/H$^2$, where W=weight in kg, H=height in meters [20]. BMI was categorised according to the World Health Organisation (WHO) Asia-pacific classification, where BMI of <18.5 kg/m$^2$ was considered as underweight, 18.5-22.9 kg/m$^2$ as normal BMI, 23-24.9 kg/m$^2$ as overweight and obese when BMI was >25 kg/m$^2$ [21]. The smoking burden was expressed as pack-years, which is the product of the average number of cigarettes smoked in a day and the duration of smoking in years [22].

## STATISTICAL ANALYSIS

Statistical Package for the Social Sciences (SPSS) version 20.0 was used for the analysis of data. Qualitative data was represented as frequency and percentage whereas quantitative data were expressed as mean±Standard Deviation (SD) for both case and control groups. Independent t-test was used to compare the values of serum TBARS, GSH, lipid profile and BMI between case and control groups. Correlation between serum TBARS, GSH, lipid profile parameters and BMI in cases was done by using Pearson's correlation test. The p<0.05 was considered to be statistically significant.

## RESULTS

In the present study, the mean age of cases were 40.38±14.96 years whereas among controls the mean age was 45.98±14.96 years (p=0.216). No significant difference was observed between cases and control in terms of gender (p=0.451) and family history of hypertension (p=0.664) [Table/Fig-2]. There was a significant association of BMI with cases and controls (p=0.001) [Table/Fig-3]. The intensity of smoking, expressed as pack-years in cases, was on average 7.89 with a standard deviation of 4.5.

| Parameters | | Case | Control | p-value |
|---|---|---|---|---|
| Age (years) | | 40.38±14.96 | 45.98±14.96 | 0.216 |
| Gender n (%) | Male | 50 (86.2) | 47 (81.1) | 0.451 |
| | Female | 8 (13.8) | 11 (18.9) | |
| Family history of hypertension n (%) | Present | 43 (74.1) | 45 (77.6) | 0.664 |
| | Absent | 15 (25.9) | 13 (22.4) | |

**[Table/Fig-2]:** Age, gender wise distribution and family history of hypertension in study participants (N=58 in each group).
Statistical test used for age: Student-t test, and for gender and family history: Chi-square test

| BMI | Cases n (%) | Control n (%) | p-value |
|---|---|---|---|
| Underweight (<18.5 kg/m$^2$) | 2 (3.45) | 1 (1.72) | 0.001 |
| Normal (18.5-22.9 kg/m$^2$) | 45 (77.59) | 23 (39.66) | |
| Overweight (23.0-24.9 kg/m$^2$) | 8 (13.79) | 18 (31.03) | |
| Obese (>25 kg/m$^2$) | 3 (5.17) | 16 (27.59) | |

**[Table/Fig-3]:** Association between BMI among cases and controls (N=58 in each group).
Statistical test used: Chi-square test; p-value <0.05 considered significant

[Table/Fig-4] demonstrates that mean TC level, TAG, LDL-C, TBARS among cases were comparatively higher than controls and the difference was found to be statistically significant (p-value=0.001). A significant decrease in serum GSH was observed in cases compared to controls (p-value=0.001). There was no statistically significant difference in HDL-C and VLDL-C levels between the cases and controls (p=0.774 and 0.968, respectively). A significant positive correlation of parameters was observed in TC, TAG, LDL-C, VLDL-C and TBARS with the pack-years of smoking among the cases, whereas GSH and HDL-C showed a significant negative correlation with the pack-years (p=0.001). However, BMI did not show significant correlation with the pack-years [Table/Fig-5].

| Biochemical parameters | Cases (n=58) | Control (n=58) | p-value |
|---|---|---|---|
| TC (mg/dL) | 256.41±21.738 | 203.93±14.99 | 0.001 |
| TAG (mg/dL) | 198.47±25.330 | 177.33±26.683 | 0.001 |
| HDL-C (mg/dL) | 49.86±5.443 | 50.24±6.934 | 0.774 |
| LDL-C (mg/dL) | 137.65±15.63 | 91.05±12.09 | 0.001 |
| VLDL-C (mg/dL) | 38.55±11.407 | 38.40±27.034 | 0.968 |
| GSH (mg/dL) | 85.50±9.705 | 156.16±10.801 | 0.001 |
| TBARS (mM/L) | 5.55±1.26074 | 2.57±0.637 | 0.001 |

**[Table/Fig-4]:** Comparison (Mean±SD) of serum biochemical parameters between cases and controls.
GSH: Reduced glutathione; TBARS: Thiobarbituric acid reactive substances; The values provided for cases and controls- mean±SD; Statistical test used- Independent sample t-test; p-value <0.05 considered significant

| Clinical parameters | Smoking (Pack-years) | |
|---|---|---|
| | r value | p-value |
| BMI (kg\m$^2$) | -0.059 | 0.662 |
| TC (mg/dL) | 0.803 | 0.001 |
| TAG (mg/dL) | 0.496 | 0.001 |
| HDL-C (mg/dL) | -0.493 | 0.001 |
| LDL-C (mg/dL) | 0.652 | 0.001 |
| VLDL-C (mg/dL) | 0.56 | 0.001 |
| GSH (mg/dL) | -0.540 | 0.001 |
| TBARS (mM/L) | 0.822 | 0.001 |

**[Table/Fig-5]:** Correlation between clinical parameters and smoking (pack-years).
Statistical analysis used- Pearson's correlation test; p-value <0.05 considered significant

## DISCUSSION

The present study showed elevated levels of serum TC, TAG, LDL-C and MDA and lowered levels of BMI, HDL-C and GSH in hypertensive smokers compared to hypertensive non smokers. Hypertension and dyslipidaemia are the risk factors for the development

of cardiovascular diseases [23]. The observed higher BMI in hypertensive controls indicated that the majority of this population was obese. Obesity was found to be one of the causative factors for hypertension and cardiovascular disease by the activation of the rennin angiotensin-aldosterone system, increased sympathetic activity, increased insulin resistance, elevated leptin resistance, intense procoagulatory activity and endothelial dysfunction [24].

Nicotine present in tobacco may increase energy utilisation, may reduce appetite leading to less food intake and more energy expenditure resulting in low body weight [13]. This may be the probable reason in present study finding of low BMI in cases when compared to the controls.

An earlier study on the effect of smoking on Blood Pressure (BP) in normotensive individuals has shown that smoking causes an increase in BP, where, the non smokers had a SBP of 119±3 mmHg and DBP of 80±2 mmHg, whereas smokers who smoke an average number of 20±5 cigarettes daily for a period of 12±5 years had a SBP of 137±5 and DBP of 90±4 mmHg [12]. A slightly different observation was obtained in a retrospective cohort study by Andriani H et al., [25]. In this study, the effect of smoking for seven years was studied, where they have found that male and female smokers had different presentations of BP. The male smokers had SBP and DBP of 130.72±18.43 and 80.44±10.95 mmHg against male non smokers, who presented SBP and DBP of 136.47±20.57 and 82.55±mm Hg, respectively. In female population, smokers had SBP and DBP of 140.46±24.86 and 82.51±10.33 mmHg against non smokers, who presented SBP and DBP of 133.71±23.66 and 81.77±11.70 mm Hg, respectively [25]. The explanation for this finding was that the male smokers experienced relief from stress. However, protracted smoking further exacerbates hypertension.

The present observation of dyslipidaemia in hypertensive smokers was in agreement with a few other studies where normotensive smokers showed dyslipidaemia compared to non smokers [26-28]. Smokers exhibited dyslipidaemia with low HDL (p=0.001) along with high systolic and diastolic BP compared to non smokers [29]. Smoking induces the release of catecholamines, which may cause increased circulatory free fatty acids, resulting in increased VLDL-C and LDL-C levels and lower HDL-C [30]. Insulin resistance induced by smoking causes hyperinsulinaemia which may reduce the lipoprotein lipase activity resulting in dyslipidaemia [31]. Contradictory to the present observation, one of the earlier studies explained dyslipidaemia associated with smoking only with respect to changes in TC (mmol/L), where smokers and non smokers had the values of 4.05±0.81 and 4.21±0.87 (p=0.017) [32]. Hypertension and smoking are two individual risk factors of dyslipidaemia. As dyslipidaemia is a risk factor of coronary artery disease, hypertensive smokers have a further increased risk of suffering from cardiovascular diseases. As observed in the present study, hypertensive smokers showed further disturbed lipid profile. Findings of a study conducted by Moradinazar M et al., was that the heavy smokers who smoked on an average more than 20 cigarettes per day had a normalised level of lipid profile upon quitting smoking compared to the population who smoke fewer cigarettes with an average of less than 10 cigarettes per day [10].

Lipid peroxidation results in the generation of MDA as the product. Estimation of MDA is useful to find the extent of lipid peroxidation by free radicals [33]. Cigarette smoke contains reactive oxygen species like $H_2O_2$, which is taken up and reduced by GSH with the help of selenium-dependent peroxidase [34]. Elevated serum MDA with decreased antioxidants vitamin C and vitamin E was observed in hypertensive individuals compared to normotensive controls (p-value <0.05) [35]. In the case of hypertensive smokers the cigarette smoke induces generation of more free radicals and thus more antioxidants are utilised for scavenging these reactive oxygen species which may result in a decreased availability of antioxidants for protection of lipids by peroxidative damage and thus generation of reactive oxygen species [12,36]. This may be the possible reason for the present observation of decreased GSH in cases. GSH is a major intracellular antioxidant, which tries to normalise the free radicals generated, moreover, it also plays a role in maintaining optimum levels of vitamin antioxidants in plasma [37]. The sulfhydryl group (-SH) in GSH is responsible for this effect [37]. In this context, in a study by Dikalov S et al., it was observed that the mitochondrial oxidative stress induced by tobacco smoke is responsible to cause endothelial dysfunction, which further aggravates hypertension [38]. A list of earlier published works in this regard and the present findings may help in better understanding of the outcomes [Table/Fig-6] [11,12,25,26,28-32,35].

Thus hyperlipidaemia and oxidative stress are more in hypertensive smokers. In the present era, knowledge about such ill effects is readily available in different electronic media. Despite smokers justify themselves with interesting explanations. Some of such reasons were socialisation in younger generations, relief of pain in older people, prevention of weight gain in females, whereas, enjoyment

| S. No. | Authors name, year and ref no. | Place of the study | Sample size | Parameters assessed | Outcome |
|---|---|---|---|---|---|
| 1 | Pasupathi P et al., 2009, [12] | Tamil Nadu, India | Case-control study- 100 smokers and 100 non smokers | SBP, DBP, lipid profile, TBARS, GSH, glutathione peroxidase, superoxide dismutase, catalase, vitamin A, vitamin C, vitamin E. | Significantly elevated SBP, DBP, lipid profile, TBARS and low glutathione peroxidase, superoxide dismutase, catalase, vitamin A, vitamin C, vitamin E in smokers compared to non smokers. |
| 2 | Gepner AD et al., 2011 [30] | Wisconsin, US | One year prospective study- 1504 | TC, TAG, LDL-C, HDL-C. | Smoking cessation in women resulted in weight gain, elevated HDL-C and total HDL without significant change in LDL-C. |
| 3 | Yan-Ling Z et al., 2012 [32] | China | 870 | Fasting blood glucose, TC, TAG, LDL-C, HDL-C. | No significant changes in lipid profile except for TC among smokers and non smokers. |
| 4 | Rao Ch S and Subhash EY 2013 [11] | Andhra Pradesh, India | 75 male subjects | TAG, TC, HDL-C, LDL-C, VLDL-C. | Tobacco users showed significantly high TC, TAG, LDL-C and VLDL-C with low HDL-C compared to non users. Significant elevation of TC, TAG with low HDL-C in population who are both smokers and chewers compared to those who are smokers but non chewers. |
| 5 | Lakshmi AS et al., 2014 [28] | Tamilnadu, India | Case control study including 40 smokers and 40 nonsmokers. | Complete blood count, TC, TAG, LDL-C, HDL-C. | Smokers showed significantly elevated haemoglobin, haematocrit, total leukocyte count, TC, TAG, LDL-C and low HDL-C compared to non smokers. |
| 6 | Mahassni SH et al., 2016 [29] | Saudi Arabia | 68 | BMI, SBP, DBP, TC, TAG, LDL-C, HDL-C. | Cigarette smoking was associated with hypertension and dyslipidaemia. Smokers showed higher BMI. |
| 7 | Babandi A et al., 2017 [35] | Nigeria | Case control study – 400 with 200 each hypertensive and normotensive | Serum Calcium, vitamin C, vitamin E, TBARS | Hypertensive population showed significantly low serum calcium, vitamin C and vitamin E with high TBARS compared to normotensive population. |
| 8 | Nepal G et al., 2018 [31] | Nepal | 280 | TC, TAG, LDL-C, HDL-C. | Smoking and alcohol consumption was associated with hypercholestrolemia and hypertriglyceridemia. |

| | | | | | |
|---|---|---|---|---|---|
| 9 | Jain RB and Ducatman A 2018 [26] | Data from NHANES for the years 1999-2012, US. | Cohort study- 15267 | Dietary habits, BMI, TC, TAG LDL-C, HDL-C. | Smoking was associated with dyslipidaemia. |
| 10 | Andriani H et al., 2020 [25] | Indonesia | Cohort study- 10338 | Smoking history, BMI, SBP, DBP. | SBP in female smokers and DBP in new male and female smokers showed association with the intensity of smoking. |
| 11 | Present study, 2022 | Karnataka, India | Case-control study, 116 with 58 hypertensive smokers and 58 hypertensive non smokers respectively. | BMI, TAG,TC,LDL-C, HDL-C, VLDL-C, TBARS, GSH. | Significant association between dyslipidaemia, oxidative stress and smoking in hypertensive individuals. Significant correlation between smoking and dyslipidaemia, oxidative stress. |

**[Table/Fig-6]:** Previously published similar works [11,12,25,26,28-32,35].
TC: Total cholesterol; TAG: Triacylglycerol; LDL-C: Low density lipopotein-cholesterol; HDL-C: High density lipoprotein-cholesterol; VLDL-C: Very low density lipoprotein-cholesterol; GSH: Reduced glutathione; TBARS: Thiobarbituric acid reactive substances; BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure

and relief of stress were the main reasons in adult males [13,39]. Family history is a non modifiable risk factor for hypertension, but smoking begins purely as an enjoyable act that subconsciously turns into addiction. Chronic smokers may encounter difficulty in quitting smoking where they may experience temporary withdrawal symptoms. However, this population, especially of younger age, maybe encouraged to quit smoking by making them understand the protracted ill effects of smoking.

### Limitation(s)

Limitation of the present study was that duration of hypertension was not taken into account and the sample size was small.

## CONCLUSION(S)

The present study demonstrated that smoking further disrupted the existing dyslipidaemia in hypertensive individuals. Moreover, the oxidative stress induced by smoking causes further oxidative damage indicated by elevated oxidant and lower antioxidant levels. A more controlled study with respect to the duration of both hypertension and smoking may be more useful in understanding the extent of damage caused by smoking in individuals diagnosed with hypertension and to advise this population to quit smoking.

### Acknowledgement

Authors sincere gratitude to Ms. Preeval Shreya Crasta, Statistician and Assistant Professor, Department of Community Medicine for the timely assistance with the statistical part of the project. Authors would like to thank the Head of Deparment and faculty, Department of Internal Medicine for their support.

## REFERENCES

[1] Kukubo Y, IwashimaY, Kamide K. Hypertension: Introduction, types, causes and complications. In: Jagadeesh G, Balakumar P, Maung UK. (eds) Pathophysiology and pharmacotherapy of cardiovascular disease. Adis, Cham. https://doi.org/10.1007/978-3-319-15961-4-30.
[2] Petrie JR, Guzik TJ, Touyz RM. Diabetes, hypertension and cardiovascular disease: Clinical insights and vascular mechanisms. Canadian J Cardiology. 2018;34(5):575-84.
[3] Maharjan BR, Jha JC, Vishwanath P, Alurkar VM, Singh PP. Oxidant- antioxidant status and lipid profile in the hypertensive patients. J Nepal Health Res Counc. 2008;6(13):63-68.
[4] Baradaran A, Nasri H, Rafieian-Kopaei M. Oxidative stress and hypertension: Possibility of hypertension therapy with antioxidants. J Res Med Sci. 2014;19(4):358-67.
[5] Gebrie A, Gnanasekaran N, Menon M, Sisay M, Zegeye A. Evaluation of lipid profile and haematological parameters in hypertensive patients: Laboratory–based cross-sectional study. SAGE Open Medicine. 2018;6:01-11.
[6] Choudhury KN, Mainuddin AKM, Wahiduzzaman M, Islam SMS. Serum lipid profile and its association with hypertension in Bangladesh. Vasc Health Risk Manag. 2014;10:327-32.
[7] Hanna BE, Hamed JM, Touhala LM. Serum uric acid in smokers. Oman Medical Journal. 2008;23(4):269-74.
[8] Naik R, Deshmukh S, Dakhure S. Study of lipid profile in young smokers and non smokers. Journal of Practical Biochemistry and Biophysics. 2017;2(1):31-34.
[9] Kubihal CV, Naik HD. A study of serum lipid profile in smokers and non–smokers: Evaluation of role of smoking on lipid profile. Int J Res Med Sci. 2019;7(4):1016 -21.
[10] Moradinazar M, Pasdar Y, Najafi F, Shahsavari S, Shakiba E, Hamzeh B, et al. Association between dyslipidemia and blood lipids concentration with smoking habits in the Kurdish population of Iran. BMC Public Health. 2020;20(1):673.
[11] Rao Ch S, Subash EY. The effect of chronic tobacco smoking and chewing on the lipid profile. J Clin Diagn Res. 2013;7(1):31-34.
[12] Pasupathi P, Saravanan G, Farook J. Oxidative stress biomarkers and antioxidant status in cigarette smokers compared to nonsmokers. J Pharm Sci & Res. 2009;1(2):55-62.
[13] Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. The American J of Clinical Nutrition. 2008;87(4):801-09.
[14] Jacobs M. Adolescent smoking: The relationship between cigarette consumption and BMI. Addictive Behaviors Reparts.2019;9:100153.
[15] Audrain-Mc Govern, Benowitz NL. Cigarette smoking, nicotine and body weight. Clin Pharmacol Ther. 2011;90(10:164-68.
[16] Delacroix S, Chokka RC, Worthley SG. Hypertension: Pathophysiology and treatment. J Neurol Neurophysiol. 2014;5:250. Doi: 10.4172/2155-9562.1000250.
[17] Satyanarayana U, Chakrapani U. In: Biochemistry (with Clinical Concepts & Case Studies)-4th Ed. Publisged by Elsevier, A division of Reed Elsevier India Pvt. Ltd, New Delhi, Co-published by Books& Allied Pvt. Ltd, Kolkata. 2013;315.
[18] Stocks J, Dormandy TL. The auto-oxidation of human red cell lipids. Br J Haematol. 1971;20:95-111.
[19] Butler. Estimation of Glutathione content of RBC and whole blood. J Lab And Clin Med.1963;61:882.
[20] Vasudevan DM, Sreekumari S, Vaidyanathan K. In: Textbook of Biochemistry for Medical students- 8th Ed. 2016; 517.
[21] Girdhar S, Sharma S, Chaudhary A, Bansal P, Satija M. An epidemiological study of overweight and obesity among women in an Urban Area of North India. Indian Journal of Community Medicine. 2016;41(2):154-57.
[22] Bhatt SP, Kim YK, Harrington KF, Hokanson JE, Lutz SM, Cho MH, et al. Smoking duration alone provides stronger risk estimates of chronic obstructive pulmonary disease than pack-years. Thorax. 2018;73(5):414-21.
[23] Otsuka T, Takada H, Nishiyama Y, Kodani E, Saiki Y, Kato K, et al. Dyslipidemia and the risk of developing hypertension in a working-age male population. Journal of the American Heart Association. 2016;5:e003053.
[24] Narkiewics K. Obesity and hypertension- the issue is more complex than we thought. Nephrology Dialysis Transplantation. 2006;21(2):264-67.
[25] Andriani H, Kosasih RI, Putri S, Kuo H. Effects of changes in smoking status on blood pressure among adult males and females in Indonesia: A 15-year population-based cohort study. MJ Open. 2020;10:e038021.
[26] Jain RB, Ducatman A. Associations between smoking and lipid/lipoprotein concentrations among US adults aged ≥20 years. J Circ Biomark. 2018;7:1849454418779310.
[27] Tan XJ, Jiao GP, Ren YJ, Gao XR, Ding X, Wang XR et al. Relationship between smoking and dyslipidaemia in western Chinese elderly males. J Clin Lab Anal. 2008;22:159-63.
[28] Lakshmi AS, Lakshmanan A, Kumar GP, Saravanan A. Effect of intensity of cigarette smoking on haematological and lipid parameters. J Clin Diagn Res. 2014;8(7):BC11-13.
[29] Mahassni SH, Bukhari AA, Bukhari MA, Al-Khathami AS. Dyslipidemia and hypertension in Saudi male cigarette smokers. Journal of Basic and Applied Research International. 2016;19(1):30-37.
[30] Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipid and lipoproteins: Outcomes from a Randomised Clinical trial. Am Heart J. 2011;161(1):145-51.
[31] Nepal G, Tuladhar ET, Acharya K, Bhattarai A, Sharma VK, Raut M, et al. Dyslipidemia and associated cardiovascular risk factors among young Nepalese university students. Cureus. 2018;10(1):e2089.
[32] Yan-Ling Z, Dong-Qing Z, Chang-Quan H, Bi-Rong D. Cigarette smoking and its association with serum lipid/lipoprotein among Chinese non agenerians/centenarians. Lipids in Health and Diseases. 2012;11:94.
[33] El-Maghrabey MH, Kishikawa N, Ohayama K, Kuroda N. Analytical method for lipoperoxidation relevant reactive aldehydes in human sera by high-performance liquid Chromatography-fluorescence detection. Analytical Biochemistry. 2014;464(1):36-42.
[34] Chavez J, Cano C, Souki A, Bermudez V, Medina M, Ciszek A, et al. Effect of cigarette smoking on the oxidant/antioxidant balance in healthy subjects. American Journal of Therapeutics. 2007;14:189-93.
[35] Babandi A, Muhammed IY, Murtala Y, Ibrahim AA, Mudugu AU, Babagana K, et al. Oxidative stress indices and calcium level among hypertensive patients in Kano-Nigeria. Borno Medical Journal. 2017;2(1):01-09.

[36] Bello HA, Dandare A, Danmaliki GI. Effects of cigarette smoking on lipid peroxidation and serum antioxidant vitamins. IOSR Journal of Pharmacy and Biological Sciences. 2017;12(2):40-44.
[37] Adeoye O, Olawumi J, Opeyemi A, Christiania O. Review on the role of glutathione on oxidative stress and infertility. JBRA Assist Reprod. 2018;22(1):61-66.
[38] Dikalov S, Itani H, Richmond B, Arslanbaeva L, Vergeade A, Rahman SMJ, et al. Tobacco smoking induces cardiovascular mitochondrial dysfunction, and enhances hypertension. M J Physiol Heart Circ Physiol. 2019;316:H 639-46.
[39] Baumeister RF. Addiction, cigarette smoking, and voluntary control of action: Do cigarette smokers lose their free will? Addiction Behav Rep. 2017;5:67-84.

**PARTICULARS OF CONTRIBUTORS:**
1. Research Scholar, Department of Biochemistry, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India.
2. Professor and Head, Department of Biochemistry, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India.
3. Associate Professor, Department of Biochemistry, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India.

**NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:**
Dr. Bhagyajyothi M Bhat,
Associate Professor, Department of Biochemistry, Kanachur Institute of Medical Sciences, Mangalore-575018, Karnataka, India.
E-mail: dr.bhagyajyothi@kanachur.edu.in

**PLAGIARISM CHECKING METHODS:** [Jain H et al.]
- Plagiarism X-checker: Jan 30, 2022
- Manual Googling: Feb 07, 2022
- iThenticate Software: Mar 14, 2022 (12%)

**ETYMOLOGY:** Author Origin

**AUTHOR DECLARATION:**
- Financial or Other Competing Interests: The first author received ICMR STS grant for undertaking the project (Reference ID: 2020-07410).
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Jan 25, 2022**
Date of Peer Review: **Feb 09, 2022**
Date of Acceptance: **Mar 15, 2022**
Date of Publishing: **Apr 01, 2022**