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EFFECT OF ALCOHOL INTAKE AND CIGARETTE SMOKING ON LIPID PROFILE LEVELS AND SELECTED INFLAMMATORY BIOMARKERS

Godwin Olawoyinbn Adunmo[1], Temidayo Daniel Adeniyi[2], Oluwaloseyi Ayoposi Olorunfemi[3], Eyitayo Olutoyin Adunmo[4], David Adesoye Stephen[5], Abayomi Joseph Ajayi[6]

Department of Medical Laboratory Science[1,2,3], Department of Family Medicine[4],

Department of Environmental Health Sciences[5], Department of Physiology[6]

Faculty of Basic Clinical Sciences, University of Ilorin, Ilorin, Kwara State, Nigeria[1,2]

University of Ilorin Teaching Hospital, Ilorin, Kwara State, Nigeria[3,4]

Thomas Adewumi University, Oko, Kwara State, Nigeria[5,6]

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Address for correspondence

Dr. Godwin Olawoyin Adunmo

Department of Medical Laboratory Science

Faculty of Basic Clinical Sciences,

University of Ilorin, Nigeria

Email: adunmo.go@unilorin.edu.ng

Contact no: +234-8037902903

ABSTRACT

Background: Alcoholism and cigarette smoking pose serious health risks globally. They have a connection to several diseases that are the main causes of sickness and death. Relatively few studies have examined the effects of alcohol use and cigarette smoking on lipid profile levels and particular inflammatory biomarkers, in contrast to the substantial body of research on the effects of smoking on traditional indicators that explain the prevalence of cardiovascular diseases. Thus, we assess how lipid profile levels and certain inflammatory biomarkers are affected by alcohol consumption and cigarette smoking. Method: The study involved the recruitment of 160 participants. Data pertaining to their medical records, daily alcohol and cigarette use, and length of smoking were gathered using a structured questionnaire. The participants were divided into additional groups: smokers (40), alcoholics (40), alcoholic-smokers (40), and controls (40). Spectrophotometric analysis was used to assess the overall cholesterol, HDL-cholesterol, triglycerides, albumin, protein content in general, and CRP. LDL-cholesterol, cardiac risk Indices and atherogenic Indices were determined using Friedewald and Castelli formulas, respectively. Result: In comparison with the control group, the levels of total protein and albumin were considerably lower in smokers, alcoholics, and alcoholic-smokers ($p < 0.05$). In contrast, CRP levels were elevated in smokers, alcoholics, and alcoholic-smokers when compared with controls. But as compared to smokers, the number of alcoholics and alcoholic-smokers is significantly lower. Serum levels of low-density lipoprotein, triglycerides, and overall cholesterol were found to be considerably higher in the groups than in the control group. In relation to the control, the groups' HDL-C levels were, however, far lower. Cardiac risk indices and atherogenic indices were significantly elevated between the groups in contrast to the control. Conclusion: This present study suggests that both cigarette smokers and alcoholics may promote systemic inflammatory processes and systemic lipid disorders. These findings may give an insight to the mechanism by which smoking, and alcohol intake promote arterial sclerosis.

KEYWORDS: C-Reactive Protein, Total Cholesterol, Alcohol, Cigarette Smoking, and Cardiovascular Disease

Introduction

Social lifestyle choices like drinking alcohol and smoking cigarettes are strongly linked to a number of ailments. Globally, the rate of hypertension and cardiovascular disease has been on the rise despite the advancements in their management (1). Several factors have been reported to stimulate inflammatory processes, including cigarette smoking and alcohol consumption, which have been two of the biggest threats to world health over the past few decades, amongst others (2). Cigarette smoking and alcohol intake may affect the inflammatory process by directly affecting the liver's production of acute phase proteins and causing the accumulation of effector molecules (3). This may result in persistent inflammatory responses, which have been implicated in causing tissue damage, and illness. The

production of acute-phase proteins, which are mostly produced in the liver in response to infection and inflammation, is regulated by inflammatory cytokines (4), such as albumin, which is the primary protein in the plasma responsible for controlling blood oncotic pressure, transportation of water, ions, hormones, bilirubin, etc. (5, 6), and c-reactive protein (CRP), acting as opsonin in the serum that can identify and bind to damaged membranes and nuclear autoantigens for removal. (7, 8), Hence, inflammation indicators such as CRP, albumin, and other acute phase proteins must be extensively researched to fully understand the mechanism involved (9).

Cigarette smoking is a leading causal factor for atherosclerosis, cerebral vascular disease, and cardiovascular disease. Smoking has been linked to the facilitation of atherosclerosis progression through a number of pathophysiological processes, such as lipid peroxidation and injury to the vascular endothelium (10). Smoking affects the blood lipid profile; compared to non-smokers, smokers have been demonstrated to have greater serum concentrations of LDL cholesterol and lower concentrations of HDL cholesterol (11). Thus, alteration of blood lipid levels may be the mechanism by which smoking causes atherogenic effects. Nonetheless, it is well recognized that moderate alcohol usage lowers the incidence of coronary heart disease; this beneficial effect of alcohol use is mostly due to its increased influence on HDL cholesterol levels in the blood (12). Smoking cigarettes has been linked to increased cholesterol, decreased high-density lipoprotein cholesterol, and platelet aggregation (13, 14). Furthermore, drinking alcohol has been shown to lower LDL cholesterol(15). Based on this data, it appears that drinking alcohol and smoking cigarettes have different impacts on atherogenicity by changing the blood lipid profile. It has also been proposed that drinking and smoking affect blood lipid levels.

On the other hand, data regarding how alcohol consumption and cigarette smoking affect lipid profile levels and particular inflammatory biomarkers is still scarce. This will be the first attempt, as far as we are aware, in Nigeria to assess how smoking affects these newly identified variables. Accordingly, the effects of alcohol and cigarette use on lipid profile levels and certain inflammatory biomarkers is assessed in this study.

SUBJECTS AND METHODS

The study design was cross-sectional and population-based. Ilorin, a city in north-central Nigeria, and the cities that surround it make up the study area. The survey was conducted from June 2022 to November 2022. Sample size was determined using the global status report on alcohol and health (16). The prevalence was reported at 8.5% (16). The sample size of 120 was estimated using Charan *et al*., (2013) (17). With an anticipated 80% participation rate, we increased the final sample size to 160. Hence, a total of one hundred and sixty (160) adult subjects with not less than five (5) years of cigarette smoking and alcohol drinking were recruited from Ilorin Metropolis, Kwara State, Nigeria, containing forty (40) adult subjects who smoke cigarettes alone, forty (40) adult subjects who drink alcohol alone, and forty (40) adult subjects who are alcoholic smokers. Additionally, to act as research controls, forty (40) apparently healthy persons were recruited from the same metropolitan area.

The study only included people who provided written consent. Among the requirements for inclusion were men within the age range of 18 to 60 who have either been smoking cigarettes, consuming alcohol, or doing both for a period of five years or more. Clinical conditions including diabetes, hypertension, tuberculosis, alcoholism, and individuals taking medications known to change biomarkers for renal, hepatic, and cardiovascular illnesses were excluded from the research. Among the participants, none previously had diabetes mellitus, cardiovascular disease, or any other systemic or metabolic diseases, nor were they currently on any medications. Additionally, they had no history of substance misuse or usage of any of the following drugs: non-steroidal anti-inflammatory drugs, statins, corticosteroids, aspirin, or testosterone replacement therapy. A carefully crafted questionnaire created specifically for the study was used to collect the data.

Each participant's demographic data, anthropometric measures, smoking, alcoholism, and other lifestyle characteristics were gathered. A medical professional performed a clinical assessment on the participants, including taking their vital signs, in order to rule out an unstable clinical state. Blood pressure was measured using the Omron IntelliSense Automatic Upper Arm Blood Pressure Monitor and Weight (kg) divided by height (m^2) yields the estimated body mass index (BMI).

LABORATORY ANALYSIS

On the day of collection, each participant's blood sample was taken following an overnight fast and a low-fat, light diet. A tourniquet was employed during the venipuncture in the cubital fossa, but it was withdrawn right before the blood sample was taken in order to prevent an unnatural rise in the content of serum lipids and proteins. Each participant provided a

15 ml sample of their fasting blood, which was aseptically drawn and placed into bottles containing lithium heparin or plain water. Each sample was allowed undisturbed for an hour.

After centrifuging the blood sample for 10 minutes at ×4000 g, the serum and plasma were recovered and kept at -20°C. In two weeks, the biochemical parameters were analyzed used assay kit for enzyme-linked immunosorbent assay from Diazyme Laboratories, USA were used to detect CRP spectrophotometrically. Spectrophotometric analysis was performed using kits from Agappe Diagnostics, Switzerland, to assess the levels of total cholesterol, HDL-cholesterol, triglycerides, total protein, and albumin. Friedewald's formula was utilized to calculate LDL-cholesterol: (LDL-C (mmol/L) = Total Cholesterol - (T.G./2.2 + HDL-C)).

CALCULATION OF THE INDEX

The following formula was used to get the atherogenic index of plasma: Atherogenic Index of Plasma = Log Triglyceride/HDL-Cholesterol. Castelli Risk Index I was determined by applying the Castelli Formula (Castelli Risk Index I or Cardiac Risk Ratio = Total Cholesterol/HDL-Cholesterol).

STATISTICALANALYSIS

IBM SPPS version 23 (SPPS Inc., Chicago, IL, USA) statistical software was used to analyzed the data obtained. To investigate the group characteristics, frequency statistics were generated. For continuous variables, student's t-test was used to test the significance of the means.

ETHICALAPPROVAL

Permission for the research project was sought and obtained from the Kwara State Ministry of Health prior to its commencement.

RESULTS

The study involved 160 adult male participants in total, including forty smokers (40), forty alcoholics (40), forty alcoholic smokers (40), and forty controls (40). The study's findings indicate that there is a significant variation in the average age of the groups compared to the control. However, alcoholics and alcoholic smokers have a significantly higher ($p<0.05$) mean age when compared to individuals that smoke alone. Table 1 indicates that there was no noticeable variation in the BMIs of smokers, alcoholics, and alcoholic smokers, although the mean values of BMI were considerably higher ($p<0.05$) when comparing the groupings to the control.

The quantities of total protein and albumin in smokers, alcoholics, and alcoholic smokers are substantially less than those in the control group. On the other hand, CRP levels were greater among smokers, alcoholics, and alcoholic smokers than in controls. However, when compared to people who smoke alone, those who are alcoholics and smokers had significantly lower CRP levels (Table 2).

When compared to the control, the group's serum cholesterol level is higher, although it is not statistically significant. Similarly, compared to the control group, smokers especially alcoholic smokers had greater levels of triglycerides and LDL-C. Nevertheless, HDL-C quantities are substantially less in smokers and alcoholic smokers, although they are equivalent in controls and alcoholics (Table 3).

When compared to controls, smokers, alcoholics, and alcoholic smokers have significantly higher cardiac risk scores and atherogenic score (Table 4).

	Age (Years)	BMI (Kg/m^2)
Smokers (n = 40)	28.65 ± 1.44^a	22.95 ± 0.37^a
Alcoholics (n = 40)	32.75 ± 1.43^b	23.28 ± 0.45^a
Smokers and Alcoholics (n = 40)	33.83 ± 1.98^b	23.05 ± 0.56^a
Control (n = 40)	23.98 ± 0.25^c	21.58 ± 0.30^b
P-Value	0.000	0.026

Table 1. Study Population's Demographic Characteristics

At $p<0.05$, values with distinct superscripts exhibit were considered statistically significant.
BMI = Body Mass Index.

	Total Protein (g/l)	**Albumin (g/l)**	**CRP (ug/ml)**
Smoker (n=40)	61.00 ± 0.97^a	31.38 ± 0.46^a	4.32 ± 0.13^a
Alcoholics (n=40)	62.05 ± 0.74^a	30.20 ± 0.48^a	2.89 ± 0.12^b
Smokers and Alcoholics (n=40)	61.13 ± 0.78^a	30.05 ± 0.59^a	3.32 ± 0.15^c
Control (n=40)	70.35 ± 0.75^b	38.63 ± 0.69^b	2.46 ± 0.11^d
p-value	0.001	0.002	0.001

Table 2. The effect of Alcohol consumption and Cigarette Smoking on some Inflammatory Markers

At $p<0.05$, values with distinct superscripts exhibit were considered statistically significant.
CRP = C-reactive protein.

Subject	TC (mmol/L)	TG (mmol/L)	HDL (mmol/L)	LDL (mmol/L)
Smokers (n=40)	5.36 ± 0.21^{a}	1.52 ± 0.64^{a}	1.72 ± 0.48^{b}	2.95 ± 0.15^{a}
Alcoholics (n=40)	5.17 ± 0.79^{a}	$1.07 \pm 0.34^{b,c}$	1.98 ± 0.41^{a}	2.70 ± 1.43^{a}
Smokers and Alcoholics (n=40)	4.81 ± 0.11^{a}	1.10 ± 0.35^{b}	1.73 ± 0.38^{b}	2.58 ± 1.39^{a}
Control (n=40)	4.21 ± 0.80^{a}	0.97 ± 0.27^{c}	1.94 ± 0.40^{a}	1.83 ± 0.84^{b}
p-value	0.213	0.000	0.000	0.000

Table 3. The Impact of Smoking Cigarettes and Alcohol Consumption on the Levels of Lipid Profiles

At $p<0.05$, values with distinct superscripts exhibit were considered statistically significant.

TC = Total Cholesterol

TG = Triglyceride

HDL = High Density Lipoprotein

Subject	CRI	AI
Smokers (n=40)	3.20 ± 0.11^{a}	1.87 ± 0.11^{a}
Alcoholics (n=40)	$2.74 \pm 0.52^{a,b}$	1.60 ± 0.90^{a}
Smokers and Alcoholics (n=40)	$2.81 \pm 0.80^{a,b}$	1.78 ± 0.10^{a}
Control (n=40)	2.21 ± 0.62^{b}	0.97 ± 0.55^{b}
p-value	0.076	0.000

Table 4. The Impact of Smoking Cigarette and Alcohol Consumption on the Cardiac Risk Index and Atherogenic Index

LDL = Low Density Lipoprotein

At $p<0.05$, values with distinct superscripts exhibit were considered statistically significant.

CRI = Cardiac risk index

AI = Atherogenic index

DISCUSSIONS

The study's findings indicate that, in comparison to controls, the plasma of smokers, alcoholics, and alcoholic smokers had higher levels of LDL-C, TC, and TG. Still, the HDL-C values of smokers and alcoholic smokers were significantly lower than those of controls. Nevertheless, the HDL-C levels of the drinkers and the control group did not differ significantly. Blood lipids are vital to many bodily processes and serve as a structural element; however, unstable lipid fractions can be harmful to an individual's health. (18). This study also demonstrated the strong correlation between dyslipidemia and ongoing smoking. Nicotine is present in cigarettes; thus, smoking causes the lungs to absorb large amounts of nicotine into the bloodstream. Regular exposure to nicotine causes the body to release catecholamines by increasing lipolysis, circulating free fatty acids, and activating adenyl cyclase in adipose tissue. Elevated hepatic free fatty acid levels trigger the synthesis of TG and VLDL-C, consequently elevating blood levels of these lipids (19). Lower blood levels of HDL-C are a result of elevated levels of LDL-C and VLDL-C(11). Increased LDL-C and decreased HDL-C are mostly linked to CVDs. Impaired cholesterol is one of the causes of atherosclerotic heart disease. Our findings demonstrated that the levels of TC were considerably high in smokers, alcoholics, and alcoholic-smokers than in controls, which is in line with earlier studies (20, 21, 22, 23). As per many research, the triglyceride levels of smokers, alcoholics, and alcoholic-smokers were also higher than those of controls ($p < 0.000$) (20, 21, 22, 24, 25). Additionally, compared to anthropometrically matched controls, smokers and alcoholic smokers had lower HDL values ($p <0.000$). This result is consistent with findings from earlier studies (19, 21, 22, 24, 25).

Alcohol consumption has been reported to have the opposite effect when compared to the antherogenic effects of cigarette smoking (9). It is commonly known that drinking alcohol in moderation lowers the possibility of developing atherosclerotic heart disease, and that alcohol's beneficial effects are mostly caused by its increased effect on HDL cholesterol levels in the

blood (12). The findings from this investigation, which aligned with those of similar studies, demonstrate that there was no noticeable variation in the blood HDL-C level comparing the control group and the alcoholic group (21). However, heavy alcohol consumption has been linked to elevated systemic lipid disorders, resulting in increased lipid profile levels as it increases adipose tissue lipolysis, leading to ectopic fat disposition within the liver and the development of accumulated alcoholic fatty acids. On the other hand, the increase in lipid profile levels may be due to the high sugar level in alcohol.

The activation of inflammatory and other types of cells to defend the host against pathogens, poisons, and diseases through tissue healing and repair is the hallmark of the evolutionarily conserved process of inflammation. Immune-related cells' generation of inflammatory promoting cytokines and the ongoing stimulation of the innate immune system can result in a number of disease conditions (26). The plasma c-reactive protein levels in this study varied significantly among the groups; the control group had the lowest amount of c-reactive protein, and the single smokers had the highest level. A significant rise in c-reactive protein levels was also found in this study between alcoholic smokers and alcoholics, suggesting that alcohol consumption and cigarette smoking had antagonistic effects on the inflammatory marker. This study concurs with research previously documented by Shamima et al., (2015) (27) but contradicts studies by Kanae et al., (2021); (28) who found a synergistic effect between alcohol consumption and cigarette smoking on plasma c-reactive protein levels.

Additionally, the current investigation showed that alcohol intake and cigarette smoking are linked to noticeably decreased plasma total protein and albumin concentrations in smokers as compared to controls. The study's findings also revealed that alcoholic smokers had lower plasma albumin levels than people who either smoke or drink alone. This suggests that alcohol consumption and cigarette smoking work together to reduce plasma albumin levels even further. The reduced levels of albumin and total protein in plasma among smokers are consistent with the findings of Khaled and Rahab, (2014) (29) and the synergistic effect agrees with the study of Kanae *et al.,* (2021); (28). However, because albumin is an endogenous antioxidant, it is possible that the decreased levels of albumin found in smokers, alcoholics, and alcoholic smokers are partly due to the increased production of free radicals. Cigarette smoking and alcoholic intake have been attributed to causing oxidative stress. On the other hand, alcohol intake impairs liver function. Thus, this may ultimately affect albumin synthesis, as reported in this study (29). Furthermore, cigarette smoking, and alcohol intake have been reported to impair renal function, causing increased excretion of albumin and reduced levels of plasma albumin, as seen in this study (29).

Additionally, this study showed that, in comparison to the control group, cigarette smoking, and alcohol intake are linked to considerably increased cardiac risk indices and atherogenic indices among smokers, alcoholics, and alcoholic smokers. The elevated cardiac risk index and atherogenic index levels in smokers agree with the study carried out by Brinton, (2012); (20). Albuquerque *et al.,* (2019); (30), also reported that cigarette smoking, and alcohol intake have been reported to impair lipid synthesis or metabolism, causing increased distortion of lipid levels and ultimately the values of the atherogenic indices, as also seen in this study.

CONCLUSION

Our study confirms elevated CRI, AI, CRP, TC, TG, and LDL-C levels in smokers, alcoholics, and alcoholic-smokers. Total protein, albumin, and HDL-C were significantly reduced. These findings thus suggest the mechanisms through which cigarette smoking and alcohol intake promote atherosclerosis.

LIMITATION AND STRENGTH

The limitations of the study are that the non-compliance of the subjects with the diet prescription may affect the analysis and that a substantial part of the subjects was from the Fulani ethnic group, and the strategy for sampling, which is prone to bias in selection; yet the study's strength lies in the size of the sample.

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REFERENCES

1. Field KM. Effect of 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors on high-sensitivity C-reactive protein levels. Pharmacotherapy (Internet). 2005;25(10):1365–77. http://dx.doi.org/10.1592/phco.2005.25.10.1365
2. Linlin C, Huidan D, Hengmin C, Jing F, Zhicai Z, Junliang D, et al. Inflammatory responses and

inflammation-associated diseases in organs. Impact Journals. 2017; 56:2–7.

3. Rehm J, Gmel GE, Gmel G, Hasan SM, Imtiaz S, Popova S, et al. The relationship between different dimensions of alcohol use and the burden of disease, An update. The Lancet. 2017; 112:968–1001.
4. Koj G, Fedi S, Grassi M, Rosi E, Liotta AA, Fontanelli AD, et al. Acute phase proteins in alcoholics with or without liver injury. Italian Journal of Gastroenterology. 2015; 24:3–5.
5. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. N Engl J Med (Internet). 1999;340(6):448–54. http://dx.doi.org/10.1056/NEJM199902113400607
6. Griswold G, Fullman N, Hawley C, Arian N, Zimsen SR, Tymeson HD. Alcohol use and burden for 195 countries and territories, 1990-2016: A systematic analysis for the Global Burden of Disease Study. The Lancet. 2018; 392:1015–35.
7. Marmot M and Brunner E. Alcohol and cardiovascular disease. BMJ Journal. 2019; 303:562-565.
8. Pickles T, Liu M, Berthelet E. For the Prostate Cohort Outcomes Initiative, the effect of smoking on outcome following external radiation for localized prostate cancer. Journal of Urology. 2008; 171:1543–6.
9. Nicola S, Jason A. Role of C-Reactive Protein at Sites of Inflammation and Infection. Frontiers in Immunology. 2018; 97:24–7.
10. Barbara M, David B. Mechanisms of Endothelial Dysfunction and Early Atherogenesis. Arteriosclerosis, Thrombosis, and Vascular Biology. 2014; 34:509–15.
11. Jain RB, Ducatman A. Associations between smoking and lipid/lipoprotein concentrations among US adults aged ≥20 years. J Circ Biomark (Internet). 2018; 7:1849454418779310. http://dx.doi.org/10.1177/1849454418779310.
12. Piano MR. Alcohol's effects on the cardiovascular system. Alcohol Res. 2017;38(2):219–41.
13. Bøttcher M, Falk E. Pathology of the coronary arteries in smokers and non-smokers. Eur J Cardiovasc Prev Rehabil (Internet). 1999;6(5):299–302. http://dx.doi.org/10.1177/204748739900600504
14. Howard G, Burke GL, Szklo M, Tell GS, Eckfeldt J, Evans G, et al. Active and passive smoking are associated with increased carotid wall thickness. The Atherosclerosis Risk in Communities Study. Arch Intern Med (Internet). 1994;154(11):1277–82. http://dx.doi.org/10.1001/archinte.154.11.1277
15. Shimomura T, Wakabayashi I. Inverse associations between light-to-moderate alcohol intake and lipid-related indices in patients with diabetes. Cardiovasc Diabetol (Internet). 2013;12(1):104. http://dx.doi.org/10.1186/1475-2840-12-104
16. Global status report on alcohol and health-2018. Geneva: World Health Organization. 2018; 1:1–3.
17. Charan J, Biswas T. "How to calculate sample size for different study designs in medical research". Indian journal of psychological medicine. 2013;35(2), 121–126.
18. Samuel O, Ibrahim S, Olusegun TO. Lipid profile of cigarette smokers in an ancient City. Sch J Appl Med Sci SJAMS. 2013; 1:447–51.
19. Nakamura K, Barzi F, Lam T-H, Huxley R, Feigin VL, Ueshima H, et al. Cigarette smoking, systolic blood pressure, and cardiovascular diseases in the Asia-Pacific region. Stroke (Internet). 2008;39(6):1694–702. http://dx.doi.org/10.1161/STROKEAHA.107.496752
20. Brinton E. Effects of ethanol intake on lipoproteins. CurrAtheroscler. 2012; 14:108–121.
21. George S, John SD, George S, Thottiyil JJ. Lipid profile and alcoholism. Int J Adv Med (Internet). 2019;6(5):1408. http://dx.doi.org/10.18203/2349-3933.ijam20193595
22. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. Am Heart J (Internet). 2011;161(1):145–51. http://dx.doi.org/10.1016/j.ahj.2010.09.023
23. Nakamura M, Yamamoto Y, Imaoka W, Kuroshima T, Toragai R, Ito Y, et al. Relationships between smoking status, cardiovascular risk factors, and lipoproteins in a large Japanese population. J Atheroscler Thromb (Internet). 2021;28(9):942–53. http://dx.doi.org/10.5551/jat.56838

24. Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking intensity and lipoprotein abnormalities in active smokers. J Clin Lipidol (Internet). 2009;3(6):372–8. http://dx.doi.org/10.1016/j.jacl.2009.10.008

25. Singh D. Effect of cigarette smoking on serum lipid profile in male population of Udaipur (Rajasthan). Int J Clin Biochem Res. 2016;3.

26. David F, Judith C, Eric V, Pedro C, Sasha T, Claudio F, et al. Chronic inflammation in the etiology of disease across the life span. Nature Medicine. 2019;25(2):1822–32.

27. Shamima J, Qazi SA. Serum High Sensitive C - reactive protein in Male Smokers of Bangladesh. Journal of Bangladesh Society of Physiology. 2015; 10:36–40.

28. Kanae M, Susumu T, Minae M, Mano H, Michihiro M, Toshiyuki S, et al. The Combination of Cigarette Smoking and Alcohol Consumption Synergistically Increases Reactive Carbonyl Species in Human Male Plasma. International Journal of Molecular Sciences. 2021; 22:9038-9043.

29. Khaled SA, Rahab DA. Effect of cigarette smoking on liver functions: a comparative study conducted among smokers and non-smokers male in El-beida City, Libya. Libya International Current Pharmaceutical Journal. 2014; 3:291–5.

30. Albuquerque LG, Moreira DM, Silva RL da, Fattah T. Association between smoking and alcohol consumption and the severity of coronary artery injuries in patients with AMI. International Journal of Cardiovascular Sciences (Internet). 2019; http://dx.doi.org/10.5935/2359-4802.20190007

Orcid ID:

Godwin Olawoyinbn Adunmoi - https://orcid.org/0000-0001-7938-8092

Temidayo Daniel Adeniyii - https://orcid.org/0000-0002-8482-9136

Oluwaloseyi Ayoposi Olorunfemii - https://orcid.org/0009-0000-3589-0427

Eyitayo Olutoyin Adunmoi - https://orcid.org/0009-0006-3129-0524

David Adesoye Stepheni - https://orcid.org/0009-0004-7482-160X

Abayomi Joseph Ajayi - https://orcid.org/0009-0007-1129-9254

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