Combined Effects of Smoking Type and Dietary Patterns on Dyslipidemia Risk: A Cross-Sectional Study

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Abstract: Smoking and dietary habits are among the most important of these factors. This study aimed to examine the relationship between smoking, different dietary patterns, and blood lipid levels. Ibn Al-Nafees Teaching Hospital selected 105 adults (ranging in age from 20 to 60) for the research. The participants were grouped according to their smoking status: non-smokers, cigarette smokers, e-cigarette users, and shisha smokers, 35 individuals were assigned to each of 3 nutritional categories: healthy, moderate, and unhealthy. They fasted for at least 8 hours was required before blood samples were taken for the purpose of measuring total cholesterol, triglycerides, LDL-C, and HDL-C. When comparing the diet groups to the one that had a healthy lunch before, there were clear statistical differences. More specifically, levels of TC, TG, and LDL-C were significantly higher while levels of HDL-C were significantly lower in those with an unhealthy diet ($p < 0.05$). The results also proved a that smoking negatively affected lipid profiles, which was a major concern, in those who smoked cigarettes & hookah ($p < 0.001$). The results demonstrate that unhealthy dietary habits and smoking both elevate the hazard of cardiovascular illness via lowering lipid levels. Multimodal preventive initiatives that aim to change people behaviors, like choosing better dietary choices and initiating smoking-cessation clinics more soon, are emphasized via the results.

1 INTRODUCTION

Heart illness and metabolic syndrome include tobacco utilize as a key modifiable hazard [1], [2]. There is strong evidence that it contributes to alterations in lipid metabolism, which in turn increase levels of total cholesterol (TC), triglyceride (TG), and low-density lipoprotein cholesterol (LDL-C) while decreasing levels of high-density lipoprotein cholesterol (HDL-C). In turn, elevates the hazard of atherogenesis of heart disease. Most notably, among people, smoking has taken several forms, including traditional cigarettes, electronic cigarettes, and shisha smoking. There has been a lot of research on the harmful effects of regular cigarettes, but less on the metabolic and biochemical effects of alternative smoking methods. A recent study found that compared to cigarette smoking, the detrimental effects on lipid profiles from waterpipe smoking could be even more pronounced due to the longer exposure period and higher hazardous chemical intake [3], [4]. While the effects of electronic cigarettes on lipid metabolism are still being studied in the long run, new research suggests that they may have an impact also [5]. TC, TG, LDL, and HDL are important markers of metabolic health and cardiovascular risk. In order to understand the possible health consequences as well as to guide preventative measures, it is vital to evaluate the relationship between several smoking practices and changes in lipid profiles. Previous studies conducted in Iraqi populations demonstrated associations between metabolic and hormonal disturbances and cardiovascular risk factors. In addition, emerging endocrine and metabolic biomarkers have been linked to alterations in lipid metabolism and cardiometabolic risk [6], [7]. Therefore, the aim of this study is to assess and compare the effects of cigarette, electronic cigarettes, and shisha on lipid profile parameters in people.

2 EXPERIMENTAL

2.1 Study Design and Participants

A total of 105 adult participants aged between 20 and 60 years were recruited from Ibn Al-Nafis Teaching Hospital in this cross-sectional study. Participants were categorized into three groups according to dietary patterns: healthy, moderate, and unhealthy (n = 35 for each group). Based on smoking status, participants were further classified into non-smokers, cigarette smokers, electronic cigarette users, and shisha smokers. Direct interviews were adopted to collect demographic and clinical data through specially designed questionnaires.

2.2 Sample Collection

Blood samples were obtained after an 8-hour fast. Lipid parameters, including TC, TG, LDL-C, and HDL-C, were analyzed by using enzymatic colorimetric methods. Statistical analysis was performed using SPSS version 26.0. The data was cleaned and formatted.

2.3 Calculation of LDL-C

Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation:

$$LDL\text{-}C = TC - HDL\text{-}C - (TG/5),$$

where TC is total cholesterol, HDL-C is high-density lipoprotein cholesterol, and TG is triglycerides.

2.4 Statistical Analysis

Normality of data was assessed by using the Shapiro-Wilk test. Comparisons between groups were determined using one-way ANOVA. Data were expressed as mean and standard deviation (mean±SD). A p-value < 0.05 was considered statistically significant.

2.5 Ethical Approval

The study protocol was approved by the Ethics Committee (Approval No. 1090). Written informed consent was obtained from all participants prior to enrollment, and all procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki.

3 RESULTS AND DISCUSSIONS

Important distinctions were noted in Population as well as lipid profile parameters throughout dietary pattern groups. Age as well as BMI various considerably in addition to groups ($p < 0.05$), denoting possibility basis distinctions that may impact metabolic results. Participants with unhealthy dietary patterns exhibited markedly elevated levels of total cholesterol (297.34 ± 132.30 mg/dL) and triglycerides (366.52 ± 116.22 mg/dL) compared to those following healthy dietary patterns (TC: 152.41 ± 24.31 mg/dL; TG: 86.88 ± 30.36 mg/dL) ($p < 0.001$). This elevation may be due to high saturated fat consumption in along with simple sugars and processed foods [7], [8], which act to increase the hepatic lipid synthesis and reduce lipid metabolism, as previously reported. LDL-C levels show a significant $p<0.05$ increased in both study groups. Inversely HDL-C levels reduced significantly in the unhealthy group. Impaired reverse cholesterol transport act in reduction of HDL-C levels and its synthesis associated with poor dietary quality [9], [10]. These findings are in line with previous results indicating that dietary patterns stimulate an atherogenic lipid profile and increases the risk of cardiovascular disease. The Mediterranean diet as healthy dietary patterns, have been reported to improve lipid metabolism and reduce inflammatory markers [11].

As illustrated in Figure 1 progressive elevation was found in TC and TG levels in comparison of unhealthy to healthy dietary groups. This significant elevation indicates the dose response relation between dietary quality and lipid profile degradation. The greater variability noted in the unhealthy group may be attributed to heterogeneity in dietary consumption as well as personal metabolic responses. The demographic and lipid profile characteristics according to dietary patterns are presented in Table 1. In Table 1:

- Healthy. Participants following a balanced/healthy diet;
- Moderate. Participants with moderately balanced dietary habits;
- Unhealthy. Participants with poor dietary patterns (high-fat, sugar food intake).

Data are presented as box plots showing median, interquartile range, and minimum–maximum values. Significant differences were observed between groups ($p < 0.05$).

Table 1: Association between dietary patterns and lipid profile parameters among study participants.

Parameter	Healthy (n=35)	Moderate (n=35)	Unhealthy (n=35)	p-value
Age (years)	46.74 ± 9.81	40.29 ± 10.37	36.2 ± 10.50	<0.05
BMI (kg/m^2)	31.32 ± 5.25	27.22 ± 5.02	33.38 ± 5.42	<0.05
TC (mg/dL)	152.41 ± 24.31	262.57 ± 136.28	297.34 ± 132.30	<0.001
TG (mg/dL)	86.88 ± 30.36	342.87 ± 96.86	366.52 ± 116.22	<0.001
LDL-C (mg/dL)	113.46 ± 7.29	179.2 ± 107.92	153.75 ± 118.85	<0.05
HDL-C (mg/dL)	48.83 ± 5.62	44.83 ± 9.19	40.25 ± 9.92	<0.05

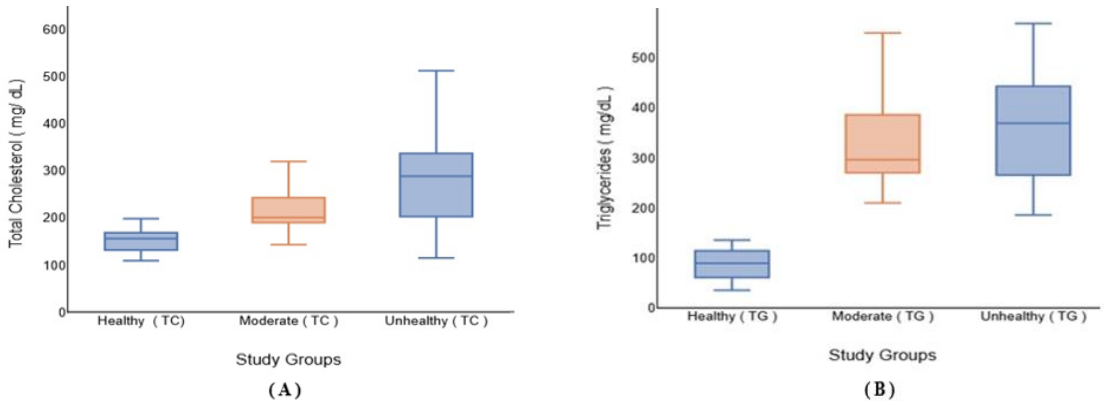

Figure 1: Distribution of lipid profile parameters across dietary pattern groups: a) Total cholesterol (TC) levels among healthy, moderate, and unhealthy dietary groups, b) Triglyceride (TG) levels among the study groups.

Table 2: Comparison of demographic and lipid profile parameters among study groups.

Parameter	Non-smokers (n=35)	Cigarette (n= 24)	E- Cigarette (n= 23)	Shisha (n= 23)	p-value
Age (years)	34.2±8.5	36.7±9.1	33.5±7.8	35.9±8.2	0.412
BMI (kg/m^2)	24.8±3.2	27.1±3.8*	26.4±3.5*	27.8±4.0*	0.021
TC (mg/dL)	172.5±28.6	210.3±35.2**	198.7±30.5**	215.9±33.8**	<0.001
TG (mg/dL)	110.4±25.7	165.8±40.3**	150.2±35.1**	170.6±38.7**	<0.001
LDL-C (mg/dL)	98.6±20.4	135.7±28.9**	125.3±26.7**	140.2±30.1**	<0.001
HDL-C (mg/dL)	52.3±9.1	41.5±7.8**	44.2±8.3**	39.8±7.5**	<0.001

Note: * $p < 0.05$ vs non-smokers; ** $p < 0.01$ vs non-smokers. Values are expressed as mean ± SD. Statistical comparisons were performed using one-way ANOVA followed by Tukey's post hoc test.

Based on smoking status, non-significant $p>0.05$ differences were found in age groups, indicating suitable group similarity. Inversely, BMI was significantly higher in smokers, particularly cigarette and shisha users, compared to non-smokers ($p < 0.05$), suggesting a potential association between smoking status and increased BMI in this population. Furthermore, significant modifications in lipid profile parameters were noted across smoking groups ($p < 0.001$). The smoker group showed raised levels of TC, TG, and LDL-C, together with a decline in HDL-C levels, compared to non-smokers [11]. This dyslipidemic status may be attributed to nicotine-induced catecholamine synthesis and release, which increases FFA mobilization, hepatic TG synthesis, oxidative stress and lipid peroxidation that impede lipid metabolism [12]-[15]. Particularly, shisha smokers recorded the most dyslipidemic profile which can be explained by the fact that smoking water pipe sessions have longer and include more harmful compounds, which led to larger metabolic impacts than regular cigarettes [16]. Despite what electronic cigarette users exemplified fewer harsh modifications, It may be concluded that e-cigarettes do not provide no metabolic risk since they continued to display adverse lipid modification. Comparison of demographic and lipid profile parameters according to smoking status is presented in Table 2.

Collectively, these findings highlight the synergistic role of dietary smoking and patterns on lipid metabolism. Individuals exposed to both smoking and an unhealthy diet exhibit a more

atherogenic lipid profile, which may substantially increase cardiovascular disease risk. These results emphasize the essential role of combined lifestyle modification strategies, including dietary improvement and smoking cessation, in preventing metabolic disorders and reducing the burden of cardiovascular diseases.

The plasma atherosclerosis index of the individuals increased dramatically, as seen in Table 3. The plasma atherosclerosis index has been well famed as a strong foreteller of cardiovascular disease and a sensitive indication of dyslipidemia that may contribute to atherosclerosis [17]-[19]. This study discovered that people manifest elevated LDL -c index values were more showed to the formation of atherosclerotic small, dense LDL particles, which are essential to the development of atherosclerosis [20]. The study indicates that those who smoke and consume shisha while adhering to an unhealthy diet have substantially elevated levels of TC, TG, and LDL, and reduced levels of HDL, in compared to other unhealthy groups with smoking habits. These results confirm other studies indicating that high-fat diets increase the risk of cardiovascular disease and lipid disorders [21].

Figure 2 shows the distribution of the AIP across various eating habits. The AIP values increased in a linear fashion from the healthy to the harmful food categories, forming a distinct gradient. The majority of observations were below the low-risk range, and those with a healthy food pattern had the lowest AIP levels. The opposite was true for those whose eating patterns were moderate: their intermediate AIP levels pointed to a change toward a lipid profile that was more atherogenic. Notably, in comparison to the other groups, those whose diets were unhealthy had the highest AIP levels, both in terms of distribution and median. There seems to be more variation and a higher propensity for dyslipidemia in this population. The higher upper quartiles and longer whiskers in the unhealthy group provide further evidence that poor eating habits raise the risk of cardiovascular disease. In sum, our results show a robust association between food pattern and AIP levels, with poor eating habits leading to an even more atherogenic lipid profile.

Table 4 shows that there is a strong positive link with TG ($r=0.71$, $p<0.001$), a weaker link with TC ($r=0.62$, $p<0.001$) as well as LDL-c ($r=0.55$, $p<0.001$), and a negative link with HDL-c ($r= -0.48$, $p<0.001$). These findings enhance prior research indicating that detrimental dietary practices, particularly those rich in fat, elevate the danger of atherosclerosis and cardiovascular disease by diminishing HDL-c levels and augmenting TG production in the liver [21], [22]. The study indicates that elevated levels of detrimental fats and reduced HDL are linked to an inadequate diet. Our findings align with previous research, indicating that smoking increases circulating free fatty acids, promotes oxidative stress, and contributes to the development of atherosclerosis [23]-[25]. Smoking is strongly linked to higher levels of TC, TG, and LDL-C and lower levels of HDL-C. Smoking is strongly linked to higher levels of TC, TG, and LDL-c ($r=0.60$, $p<0.001$) and lower levels of beneficial HDL-c ($r= -0.52$, $p<0.001$). The correlate between BMI, eating habits, and smoking status was weak. The data show that smoking and eating badly together raises the likelihood of heart disease. The strong links suggest lifestyle changes that help people quit smoking and eat well [26], [27].

Table 3: Atherogenic Index of Plasma (AIP).

Group	AIP (Mean ± SD)
Healthy Diet	0.24 ± 0.15
Moderate Diet	0.88 ± 0.21
Unhealthy Diet	0.96 ± 0.25
Non-smokers	0.24 ± 0.15
Cigarette Smokers	0.95 ± 0.22
E-cigarette	0.89 ± 0.20
Shisha Smokers	0.98 ± 0.23

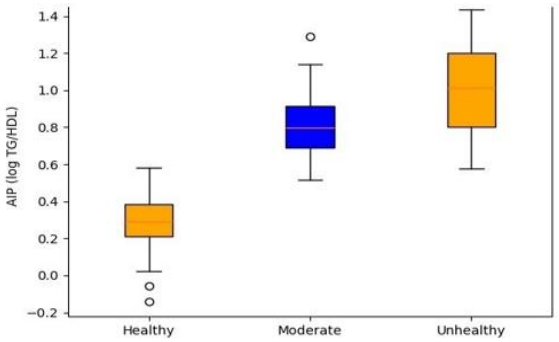

Figure 2: AIP by dietary pattern.

Figure 3 shows a connection heat map that shows how the lipid profile parameters, such as TC, TG, LDL-C, and HDL-C, are connected to each other. A robust positive correlation indicates that TC, TG, and LDL-C, three atherogenic lipid markers, often increase together. For instance, dyslipidemia is often characterized by a coordinated increase in lipids; TG was positively associated with both TC and LDL-C. Conversely, HDL-C consistently exhibited a negative connection with TC, TG, and LDL-C. This negative connection shows that HDL-C protects lipid metabolism and implies that those with higher HDL-

C levels have lower levels of atherogenic lipids. The correlation pattern supports the hypothesis that the research group has an unhealthy lipid profile since it shows that HDL-C is low.

Table 4: Correlation analysis (Pearson r).

Variable	TC	TG	LDL	HDL
Diet Pattern	0.62**	0.71**	0.55**	-0.48**
Smoking Status	0.66**	0.69**	0.60**	-0.52**
BMI	0.41*	0.46*	0.38*	-0.35*

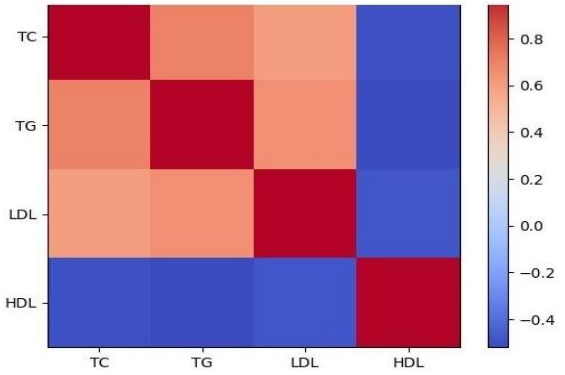

Figure 3: Correlation heatmap.

Table 5 shows the linear regression and some of the patterns it shows include dietary pattern (β= 0.52, <0.001), smoking status (β= 0.48, <0.001), BMI (β= 0.21, p= 0.02), and age (β= 0.09, p= 0.18). The findings showed that the food pattern had the most effect on TG. Smoking had the second most effect. Their results agree with what prior studies have found: smoking greatly raises the risk of heart disease. After adjusting for other characteristics, the effect was negligible for and almost BMI for age [28]-[30].

Table 5: Multiple linear regression (Outcome: TG).

Variable	β (Beta)	p-value
Diet Pattern	0.52	<0.001
Smoking	0.48	<0.001
BMI	0.21	0.02
Age	0.09	0.18

Figure 4 illustrates the linear regression analysis between the independent variable and triglyceride levels (TG). The scatter plot demonstrates a clear positive relationship, indicating that TG levels increase as the independent variable increases. The fitted regression line shows an upward trend, supporting the presence of a positive association. Although some variability is observed around the regression line, the overall pattern suggests a moderate linear relationship between the variables.

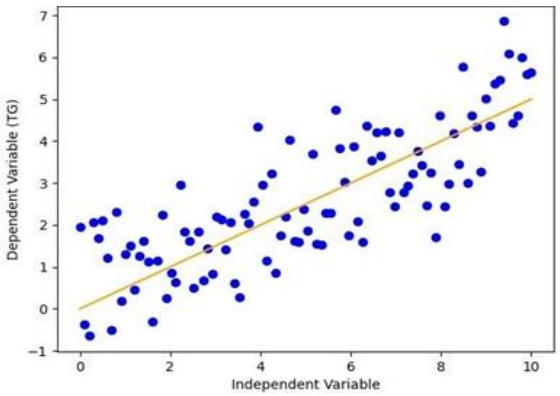

Figure 4: Regression plot.

According to Table 6 a significant majority of the research participants had atherogenic lipid profiles which raise the risk of CVD, as shown by AIP values more than 0.24. The research emphasizes that dietary manners particularly diets rich in refined carbs and saturated fats, have a greater impact on triglyceride levels than smoking does. Modifying one's diet and quitting smoking are crucial for decreasing the danger of CVD, but smoking also has a detrimental effect by lowering HDL-C and encouraging an atherogenic profile. Age and BMI don't seem to have as big of an impact as lifestyle variables. Although further longterm studies are needed to determine causation, this study concludes that lifestyle modifications are important for lowering heart disease risk and improving lipid profiles [28]-[32].

Table 6: Cardiovascular risk classification based on AIP.

Category	AIP Range	% Participants
Low Risk	<0.1	22%
Medium Risk	0.1–0.24	18%
High Risk	>0.24	60%

4 CONCLUSIONS

This research found that smoking and bad eating habits are major risk factors for dyslipidemia. People who smoke, especially those who use hookahs and cigarettes, have lower levels of HDL-C and higher levels of total cholesterol, triglycerides, and LDL-C. Lipid diseases as well as the cardiovascular problems they cause are more likely to strike people with this trait. It seems that this metabolic imbalance is worsened when a bad diet is combined with smoking. These results highlight the need of reducing

cardiovascular risk via the adoption of healthy eating habits and the implementation of smoking cessation programs.

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