A Study of the Association of Smoking with Cardiometabolic Risk

Fouzy Mohammad Lesloom[1], Jaber Ali Saad Bani Humayyim[2], Fahad Zamil Alyami[2], Obeid Mohammed Laslom[3], Mohammed Ali Bani Hamaim[4], Mohamed Hussien Bany Hammem[5], Manal Mohammed Ali Alqahtani[6], Hasan Hommadi[6], Latifah Hommadi[7], Samar Sultan[8]

(1) Laboratory Specialist, Thar General Hospital, Saudi Arabia
(2) Laboratory technician, Thar General Hospital, Saudi Arabia
(3) Nursing Technician, Forensic Medical Services Center, Najran, Saudi Arabia
(4) Dental Technician, Habuna General Hospital¸ Saudi Arabia
(5) Radiology technician, Habuna General Hospital¸ Saudi Arabia
(6) Armed Forces Hospital - Southern Region, Saudi Arabia
(7) Ahad Rufaidah General Hospital, Saudi Arabia
(8) Associate Professor, Clinical Biochemistry, Medical Laboratory Sciences Department, Faculty of Applied Medical Sciences, King Abdulaziz University, Saudi Arabia

Corresponding Author:
Fouzy Mohammad Lesloom,
Thar General Hospital, Saudi Arabia
Email: fawzy7755@gmail.com

Received: September 2023. Accepted: October 2023; Published: November 1, 2023.
Citation: Fouzy Mohammad Lesloom et al. A Study of the Association of Smoking with Cardiometabolic Risk. World Family Medicine. November 2023; 21(10): 17-27. DOI: 10.5742/MEWFM.2023.95256210

Abstract

Aim of the study: To explore the effects of cigarette smoking on cardiometabolic risk in Saudis in Jeddah City.

Methods: Following a descriptive case-control approach, the study included 160 healthy Saudi adults who were sampled consecutively (100 smokers [60 males and 40 females] and 60 non-smokers [36 males and 24 females]). Personal characteristics, smoking patterns, and cardiometabolic risk assessment were included in a questionnaire. Each participant's cardiometabolic functions were assessed by drawing blood.

Results: Among participant smokers, 80% smoked one pack daily, while 20% smoked more than one pack daily. Smokers had insignificantly higher levels of total cholesterol, LDL-cholesterol, and non-HDL, significantly higher vWF functional activity, and high-sensitivity cardiac troponin I ($p<0.001$ for both), but significantly lower albumin and total bilirubin levels than non-smokers ($p=0.026$, and $p<0.001$, respectively). The number of daily cigarettes consumed correlated positively and significantly with plasma levels of LDL-cholesterol ($r=0.225$, $p=0.004$), non-HDL cholesterol ($r=0.220$, $p=0.005$), vWF function activity ($r=410$, $p<0.001$), high-sensitivity cardiac troponin I ($r=0.686$, $p<0.001$), but negatively correlated with total bilirubin ($r=-0.459$, $p<0.001$). Moreover, the cigarette smoking intensity correlated positively and significantly with the participant's systolic blood pressure ($r=0.303$, $p<0.001$) and diastolic blood pressure ($r=0.300$, $p<0.001$), body mass index ($r=0.448$, $p<0.001$), and waist-to-hip ratio ($r=0.493$, $p<0.001$). Those who had smoked for more than 10 years had significantly higher plasma levels of triglycerides ($p=0.031$), total cholesterol ($p=0.023$), LDL cholesterol ($p=0.011$), non-HDL cholesterol ($p=0.008$), vWF functional activity ($p<0.001$), systolic blood pressure ($p=0.011$), and diastolic blood pressure ($p=0.023$). Moreover, heavy smokers had a significantly higher BMI ($p=0.001$) and waist-to-hip ratio among male smokers ($p=0.003$).

Conclusions: Cigarette smoking is associated with increased dyslipidemia, body mass index, and central obesity, in addition to higher vWF functional activity. Increased hs-cTnI levels in smokers indicate a higher risk of heart failure and cardiovascular death.

Keywords: smoking, cardiometabolic risk, lipid profile, liver function, troponin.

Introduction

Smoking is considered a leading public health problem (1), with a direct heavy toll on lives, and indirectly, via passive inhalation of tobacco smoke (2). The World's "No Tobacco Day" on May 31st is a reminder of its hazards and the importance of quitting smoking (3).

The "tobacco epidemic" is globally considered one of the worst public health problems, annually killing more than seven million people, with almost six million deaths among smokers due to direct tobacco use, in addition to about one million non-smokers who die as a result of exposure to secondhand smoke (4). It is a risk factor for several chronic diseases, infections, tumors, and cardiac and respiratory diseases. Nevertheless, cigarettes continue to be highly consumed worldwide and remain among the commonest forms of addiction (5).

Products of tobacco are commonly used worldwide. Besides their addictive properties, tobacco manufacturers enjoy the best marketing skills and strategies that actively promote their tobacco products. At the same time, adults, the elderly, and even children are progressively attracted to these extremely harmful products (4).

Tobacco use remains highly prevalent (6) due to reasons that are not completely clear and may be attributed to its addictive nature (7). Nicotine fulfills the criteria for drug dependency by promoting its compulsive use, its psychoactive effects, and reinforcing its use (8).

Smoke's constituents are either particulate (e.g., tar, poly-nuclear hydrocarbons, phenols, cresol, catechol and trace elements, nicotine, indole, carbazole, and 4-aminobiphenyl), or gaseous (e.g. CO, HCN, NH3, CH2O, C2H4O, C3H4O, and oxides of nitrogen) (9).

Tobacco consumption is an etiological agent for several chronic diseases, infections, tumors, heart diseases, and respiratory illnesses. Lighting a cigarette is responsible for several harmful adverse effects on most body organs, including the lungs, the heart, and even those without direct contact with the smoke, including the liver. The liver is a vital organ with many important tasks, such as processing drugs and toxins and removing them from the body (9).

Skranes et al. (10) recently screened subjects with stable coronary artery disease by applying the troponin test to assess cardiac-specific troponin blood levels to detect heart injury. They found lower concentrations of circulating "high-sensitivity cardiac troponin" among cigarette smokers. Still, the association between high-sensitivity cardiac troponin T and high-sensitivity cardiac troponin I and the incidence of cardiovascular events in current smokers with stable coronary artery disease is weak and not statistically significant.

"Cardiometabolic diseases" is a term that includes common cardiovascular and metabolic diseases. Cardiometabolic diseases result from primary or metabolic drivers (11). Despite being mostly preventable and treatable, cardiometabolic diseases are the first cause of mortality worldwide, with 22.4 million deaths. Almost half of them were described as "premature deaths" (i.e., before 60 years of age), and a considerable number of years of life are lost as a result of time lived in states of less than full health (12-13). However, risk factors contributing to the development of cardiometabolic diseases (e.g., obesity, dyslipidemia, and smoking) are modifiable and preventable. Estimates of the magnitude and distribution of these risk factors remain relatively scarce (14).

Cigarette smoke contains over 4,000 harmful compounds, including over 200 toxicants, 80 carcinogens, and other compounds that provoke oxidative stress. Even though tobacco has a deleterious effect on health, it continues to be highly used worldwide and remains one of the most common addictions (15).

Cigarette smoking is a significant risk factor for cardiometabolic diseases, the most considerably preventable cause of cardiovascular diseases (12), and an independent risk factor for diabetes, probably via promoting insulin resistance. It is also associated with hypertension, dyslipidemia, and low HDL levels (16).

Smoking also influences von Willebrand factor (vWF) protein, facilitating platelet aggregation and adhesion to the sub-endothelium of injured vessel walls. Smoking causes disturbances to the endothelium, associated with increased vWF levels (17). Fibrinogen is another inflammatory marker that becomes elevated in smokers. The high levels of fibrinogen affect the viscosity of blood, aggregation of platelets, and fibrin formation (18).

Despite the great efforts that the Saudi government has undertaken to fight tobacco smoking, the prevalence of smoking is still growing (19). Between 2013 and 2018, the prevalence of tobacco smoking among the Saudi population substantially increased from 12.2% to 21.4% (20). Moreover, several studies conclude that cigarette smoking and impaired liver function are associated with lowered health-related quality of life (21).

Therefore, the cardiometabolic profile of current and former smokers should be actively evaluated and compared with that among non-smokers to lower the health and economic burdens of these medical conditions.

Aim of Study

This research aims to explore the possible effects of cigarette smoking on the cardiometabolic risk of apparently healthy Saudi individuals in Jeddah City.

Methodology

This study followed a descriptive case-control research design at a primary health care center at East Jeddah Hospital, Jeddah City, Saudi Arabia. Those attending the primary health care center at East Jeddah Hospital, Jeddah City, Saudi Arabia, for vaccination or with their children at the well-baby clinics were included. Following consecutive sampling, 100 smokers and 60 non-smokers were enrolled in the present study.

Inclusion and Exclusion Criteria
The study included Saudi adults, aged 20-60 years, apparently healthy, attending the study setting. Those with a family history of cardiac diseases and those who smoke other than cigarettes (e.g., shisha or e-cigarettes) were excluded.

A study questionnaire was developed by the researcher based on a review of relevant literature. It consists of personal characteristics, smoking patterns, and cardiometabolic risk assessment. Using the data collection tool, participants were interviewed by the researcher at the outpatient clinics. All potential participants were clearly informed about the study's objectives and were invited to participate. A blood sample was drawn from each participant to assess his/her cardiometabolic functions.

Blood Sampling Procedures and Biochemical Assays
One hundred fasting smokers (60 males and 40 females) and 60 non-smokers (36 males and 24 females) had 9 mL of venous blood collected. The samples were split into three tubes: 4 mL in tubes containing lithium heparin, 3 mL in tubes containing no anticoagulant, and 2 mL in tubes containing tri-sodium citrate. The lithium heparin tubes (green top) samples were analyzed for ALT, albumin, total bilirubin, lipid profile, and fasting blood glucose levels. In contrast, serum tube samples (red top) and tri-sodium citrate samples (blue top) were employed to examine the high-sensitive troponin I and vWF, respectively. The Heraeus Labofuge centrifuge (Thermo ScientificTM, PA-USA) was used to centrifuge all green- and red-capped tubes once at 4400 g for 5 minutes at room temperature, while blue-capped tubes were centrifuged at 2000 g for 15 minutes.

After an enzymatic reaction, Alinity c (Abbott, Wiesbaden, Germany) evaluated the amounts of liver function tests, lipid profile, and fasting blood glucose by spectrophotometrically measuring light absorption. The high-sensitive troponin I was measured using Alinity I (Abbott, Wiesbaden, Germany). This assay is a two-step immunoassay using chemiluminescent microparticle immunoassay (CMIA) technology to measure cardiac troponin I in human serum. The concentrations of vWF were assessed using STA R Max3 (Stago, Parsippany-USA), which relies on assessing the clotting time in the presence of cephalin and activator.

Statistical Analysis
The Statistical Package for Social Sciences (IBM, SPSS, version 25) was used to describe and compare the demographic, clinical, and laboratory findings of the three compared groups. Data were expressed as frequency and percentage for qualitative variables and arithmetic mean and standard deviation for numerical continuous variables.

The Kolmogorov-Smirnov and Shapiro-Wilk tests were applied to explore the normality of quantitative variables. Accordingly, parametric or non-parametric statistical tests of significance were applied. A power of 0.8 (i.e., 80% probability of correctly rejecting the null hypothesis) was chosen within the present study. The effect size (d) was calculated by dividing the estimated difference between two comparison groups by their pooled estimated standard deviation (22).

The minimum sample size for the study groups was determined according to the Raosoft Online sample size calculator to be 92 in the study group, with a 0.1 margin of error, 95% confidence level, and a reported 39.8% prevalence of cardiometabolic risk among the Saudi population (23). However, the sample size in the study group was increased to 100 participants, with a 2:1 proportion between the study group and the control group.

The Chi-square test was used to compare qualitative variables, and the Student t-test was used to compare the arithmetic means of continuous variables between two or more different groups, respectively. Pearson's correlation coefficient (r) was used to describe the strength and direction of a linear relationship between the number of cigarettes smoked per day and the participant's age as well as the results of cardiometabolic tests. By using the independent variable t-test, the results of cardiometabolic tests according to participants' smoking status and smoking duration were statistically analyzed. Results with p-values of <0.05 were considered statistically significant.

The official ethical approval for conducting the present study (A0289) was obtained from the Institutional Review Board at the Directorate of Health Affairs in Jeddah on February 10th, 2022. Before the interview, all participants were clearly informed about the study's objective, that their participation was completely voluntary, and that they had the full right to withdraw without the need to state any reasons. Anonymity and privacy were completely respected during data collection. Participants' verbal consent was obtained before the start of the study.

Results

Table (1) shows that participants' age group and gender did not differ significantly according to their smoking status. However, participants' body mass index differed significantly according to their smoking status, with significantly more obese smokers than non-smokers (21% and 8.3%, respectively, p=0.017).

Figure (1) shows that among smokers, 80% smoked one pack daily (up to 20 cigarettes/day), while 20% smoked more than one pack daily (more than 20 cigarettes daily).

Table (2) shows that smokers had higher lipid profile parameters, with higher plasma total cholesterol, LDL cholesterol, non-HDL levels, and plasma HDL cholesterol. However, the differences between smokers and non-smokers were not statistically significant. Regarding liver function, smokers had significantly lower plasma albumin (p=0.026) and significantly lower total bilirubin levels (p<0.001). Regarding the hematological findings, smokers had significantly higher vWF functional activity (p<0.001). Fasting blood glucose did not differ significantly according to smoking status. Cardiac troponin I was significantly higher among smokers than non-smokers (p<0.001). Moreover, smokers had significantly higher systolic and diastolic blood pressures than non-smokers (p=0.003 and p=0.004, respectively). Smokers also had a significantly higher waist-to-hip ratio than non-smokers (p<0.001 for both males and females).

Table (3) shows that the number of cigarettes smoked per day correlated positively and significantly with participants' plasma LDL cholesterol (r=0.225, p=0.004), plasma non-HDL cholesterol (r=0.220, p=0.005), vWF functional activity (r=0410, p<0.001), cardiac troponin I serum level (r=0.686, p<0.001), systolic blood pressure (r=0.303, p<0.001), diastolic blood pressure (r=0.300, p<0.001), body mass index (r=0.448, p<0.001), and waist-to-hip, ratio (r=0.493, p<0.001). Moreover, the number of cigarettes smoked per day correlated negatively and significantly with HDL-cholesterol (r=-0.177, p=0.025), and total bilirubin (r=-0.459, p<0.001). However, other results of cardiometabolic function tests did not correlate significantly with participants' number of cigarettes smoked per day.

Table (4) shows that participants' high smoking intensity (>1 pack/day) was higher among older smokers aged >40 years and those aged 30-40 years than younger smokers aged <30 years or 30-40 years (30.6%, 24.4%, and 10.5%, respectively). However, the differences were not statistically significant. The prevalence of high smoking intensity (>1 pack/day) was significantly higher among male than female smokers (31.7% and 12.5%, respectively, p=0.028). Moreover, the prevalence of high smoking intensity (>1 pack/day) was significantly higher among obese than normal and overweight participants (57.1%, 25.0%, 14.7% and 12.5%, respectively, p<0.001)

Table (5) shows that smokers who have been smoking for more than ten years had significantly higher plasma total cholesterol (p=0.023), significantly higher triglycerides (p=0.031), significantly higher LDL-cholesterol (p=0.011), and non-HDL-cholesterol (p=0.008). Regarding the results of liver function tests, participants who smoked for more than ten years had significantly lower plasma total bilirubin (p=0.003). Regarding the hematological findings, participants who smoked for over ten years had significantly higher vWF functional activity (p<0.001). Moreover, those who smoked for more than ten years had significantly higher BMI (p=0.001), higher systolic blood pressure (p=0.011), higher diastolic blood pressure (p=0.011), and a higher waist-to-hip ratio among males (p=0.003). Other laboratory parameters (i.e., HDL-cholesterol, ALT, albumin, fibrinogen level, and Cardiac troponin I), in addition to waist-to-hip ratio, did not differ significantly according to the duration of smoking.

Table (6) shows that smokers who smoked more than one pack/day had higher lipid profile parameters, with significantly higher plasma total cholesterol (p=0.014), LDL cholesterol (p=0.011), and non-HDL levels (p=0.017). Regarding liver function, participants who smoked >1 pack/day had significantly lower plasma albumin (p=0.021). Regarding the hematological findings, participants who smoked >1 cigarette pack/day had significantly higher vWF functional activity (p<0.001). Fasting blood glucose was higher among those who smoked >1 cigarette pack/day, but the difference was not significant. Cardiac troponin I was higher among those who smoked >1 pack/day than those who smoked <1 pack/day. However, the difference was not statistically significant. Systolic and diastolic blood pressures were significantly higher among those who smoked >1 cigarette pack/day (p=0.004 for both). Moreover, the waist-to-hip ratio for males was significantly higher among those who smoked >1 cigarette pack/day (p=0.020).

Table 1: Participants' personal characteristics according to their smoking status

Personal Characteristics	Non-smokers		Smokers		P value
	No.	%	No.	%	
Age groups					
• <30 years	17	28.3	19	19.0	
• 30-40 years	25	41.7	45	45.0	0.378
• >40 years	18	30.0	36	36.0	
Gender					
• Male	36	60.0	60	60.0	
• Female	24	40.0	40	40.0	1.000
Body mass index					
• Normal (<25 kg/m²)	8	13.3	4	4.0	
• Overweight (25-29.9 kg/m²)	47	78.3	75	75.0	0.017†
• Obese (≥30 kg/m²)	5	8.3	21	21.0	

† Chi-square test (statistically significant, p<0.05)

Figure 1: Participants' cigarette smoking intensity (pack/day)

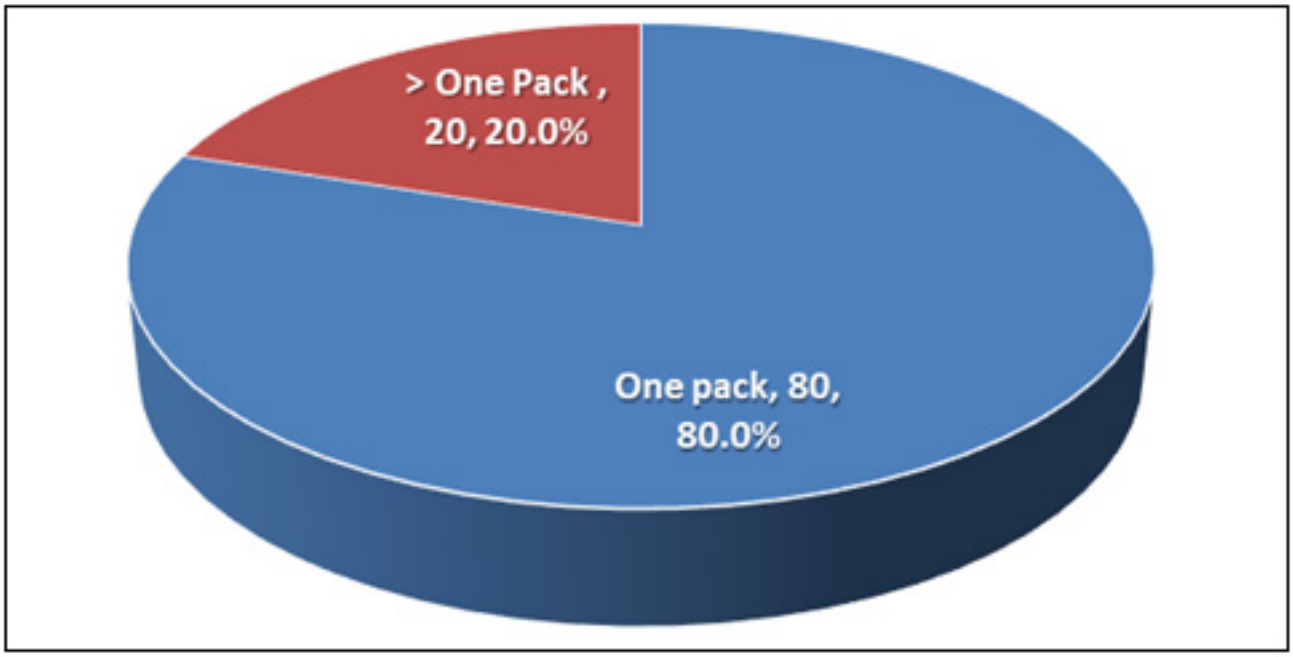

Table 2: Results of cardiometabolic tests according to participants' smoking status

Parameters	Nonsmokers (n=60)		Smokers (n=100)		
	Mean	SD	Mean	SD	P-value
Triglycerides	147.98	10.02	147.57	13.89	0.841
Total cholesterol	198.95	11.90	199.40	14.11	0.836
LDL cholesterol	113.58	17.40	116.07	17.35	0.382
HDL cholesterol	48.73	6.01	50.87	8.24	0.082
Non-HDL cholesterol	120.23	16.61	123.73	18.92	0.238
ALT	29.62	2.23	29.69	2.14	0.835
Albumin	4.74	0.28	4.63	0.32	0.026‡
Total bilirubin	0.96	0.05	0.92	0.05	<0.001‡
Fasting blood glucose	89.10	5.99	89.56	5.85	0.634
Fibrinogen level	3.51	0.54	3.59	0.86	0.498
vWF functional activity	56.45	6.59	65.87	19.07	<0.001‡
Cardiac troponin I (ng/mL)	0.0147	0.0105	0.0382	0.0077	<0.001‡
Systolic blood pressure	130.8	9.2	135.5	9.8	0.003‡
Diastolic blood pressure	91.6	6.4	94.8	7.0	0.004‡
Waist-to-Hip ratio					
Males	0.90	0.10	1.00	0.12	<0.001‡
Females	0.82	0.07	0.90	0.07	<0.001‡

‡ Independent samples t-test (statistically significant, p<0.05)

Table 3: Pearson's correlation coefficient (r) between the number of smoked cigarettes per day and participants' cardiometabolic tests

Variables	Correlation coefficient	P-value
Triglycerides	0.058	0.456
Total cholesterol	0.148	0.061
LDL cholesterol	0.225	0.004†
HDL-cholesterol	-0.177	0.025†
Non-HDL cholesterol	0.220	0.005†
ALT	0.129	0.103
Albumin	-0.098	0.216
Total bilirubin	-0.459	<0.001†
Fasting blood glucose	0.090	0.257
Fibrinogen	0.116	0.142
vWF functional activity	0.410	<0.001†
Cardiac troponin I (ng/mL)	0.686	<0.001†
Systolic blood pressure	0.303	<0.001†
Diastolic blood pressure	0.300	<0.001†
Body mass index	0.448	<0.001†
Waist-to-hip ratio	0.493	<0.001†

† Pearson's correlation coefficient (statistically significant, p<0.05)

Table 4: Participant smokers' personal characteristics according to their smoking intensity (pack/day)

Personal Characteristics	≤1 pack/day		> 1 pack/day		P Value
	No.	%	No.	%	
Age groups					
• <30 years	17	89.5	2	10.5	
• 30-40 years	34	75.6	11	24.4	0.254
• >40 years	25	69.4	11	30.6	
Gender					
• Male	41	68.3	19	31.7	
• Female	35	87.5	5	12.5	0.028†
Body mass index					
• Normal (<25 kg/m²)	3	75.0	1	25.0	
• Overweight (25-29.9 kg/m²)	64	85.3	11	14.7	<0.001†
• Obese (≥30 kg/m²)	9	42.9	12	57.1	

† Chi-square test (statistically significant, p<0.05)

Table 5: Results of laboratory findings according to participants' duration of smoking

Parameters	≤10 years (n=61)		>10 years (n=39)		P-value
	Mean	SD	Mean	SD	
Triglycerides	149.25	12.43	142.25	16.96	0.031†
Total cholesterol	197.61	12.48	205.08	17.47	0.023†
LDL-Cholesterol	113.61	17.33	123.88	15.29	0.011†
HDL-Cholesterol	50.05	7.36	53.46	10.31	0.077
Non-HDL Cholesterol	120.93	16.05	132.58	24.349	0.008†
ALT	29.50	2.22	30.29	1.76	0.115
Albumin	4.07	0.19	4.11	0.21	0.359
Total bilirubin	0.92	0.04	0.89	0.05	0.003†
Fasting blood glucose	89.07	5.36	91.13	7.067	0.133
Fibrinogen level	3.53	0.52	3.78	1.51	0.230
vWF functional activity	60.91	9.49	81.58	30.55	<0.001†
Cardiac troponin I (ng/mL)	0.038	0.008	0.039	0.008	0.484
BMI	28.03	1.70	29.53	2.39	0.001†
Systolic blood pressure	134.08	9.24	139.83	10.41	0.011†
Diastolic blood pressure	93.86	6.46	97.88	7.29	0.021†
Waist-to-hip ratio:					
• Males	0.96	0.10	1.05	0.13	0.003†
• Females	0.90	0.07	0.89	0.07	0.720

† Independent variable t-test (statistically significant)

Table 6: Results of cardiometabolic results according to participants' smoking intensity (pack/day)

Parameters	1 pack/day (n=80)		>1 pack/day (n=20)		P value
	Mean	SD	Mean	SD	
Triglycerides	149.79	11.50	144.33	17.34	0.248
Total cholesterol	198.39	11.79	209.00	16.78	0.014†
LDL-cholesterol	113.36	17.99	127.58	15.17	0.011†
HDL cholesterol	49.04	7.20	53.92	11.37	0.109
Non-HDL level	118.61	16.23	135.92	27.29	0.017†
ALT (U/L)	29.57	2.33	31.00	1.76	0.065
Albumin	4.68	0.29	4.41	0.39	0.021†
Total bilirubin	0.95	0.05	0.92	0.05	0.360
Fasting blood glucose	88.50	5.99	91.08	8.16	0.270
Fibrinogen level	3.49	0.56	3.76	7.12	0.433
vWF functional activity	57.25	7.12	81.42	28.51	<0.001†
Cardiac troponin I (ng/mL)	0.037	0.008	0.041	0.008	0.192
Systolic blood pressure	134.1	9.2	139.8	10.4	0.004†
Diastolic blood pressure	93.9	6.5	97.9	7.3	0.004†
Waist-to-hip ratio (males)	0.97	0.11	1.05	0.13	0.020†
Waist-to-hip ratio (females)	0.90	0.07	0.89	0.01	0.672

† Independent variable t-test (statistically significant)

Discussion

The term "cardiometabolic disease" includes common cardiovascular and metabolic diseases. Cardiometabolic diseases result from primary or metabolic drivers (11). Risk factors for developing cardiometabolic diseases, such as dyslipidemia and smoking, are modifiable and preventable. However, estimates of the magnitude and distribution of these risk factors remain relatively scarce (14).

Despite the strong and consistent epidemiological evidence that links cigarette smoking with several cardiovascular and liver diseases, the exact mechanisms of these links remain inadequately understood (17).

Therefore, this study aimed to explore the possible effects of cigarette smoking on cardiometabolic risk in apparently healthy Saudi individuals in Jeddah City. Laboratory findings regarding lipid profiles, liver function, fasting blood glucose, high sensitivity troponin I, and some hematological tests were compared according to participants' smoking status.

This study included 100 healthy smokers (of whom 36, 60% were males), and 60 control nonsmoker subjects (of whom 36, 60% were males). One-fifth of smokers were heavy smokers, who smoked more than one pack daily. Heavy smoking was significantly higher among males (p=0.028).

It is to be noted that although the prevalence of smoking is decreasing in developed countries, it is still increasing in Saudi Arabia. Ansari & Farooqi (24) reported that the prevalence of smoking among the Saudi population is still high, 37.6% among males and 6% among females. Abdalla et al. (25) argued several reasons for the growing spread of smoking among Saudi females, including imitation, curiosity, relieving anxiety, high socioeconomic status, and contact with other female smokers.

This observed male gender predilection for smoking has been attributed mainly to social reasons; in Saudi Arabia, the conservative community norms still disapprove and deplore female smoking, especially in public. Nevertheless, Gastaldelli et al. (16) noted that, although smoking prevalence rates were higher in men than women for most of the past century, the gender gap has recently narrowed.

The present study showed that participants' laboratory findings generally were worse among smokers than non-smokers and correlated with their smoking intensity, particularly among those who have smoked for more than ten years and heavy smokers (i.e., those who smoked more than one pack of cigarettes daily). The prevalence of dyslipidemia was significantly higher in those who smoked more than one pack of cigarettes daily than in those who smoked less than one pack per day. Plasma total bilirubin levels were significantly lower in smokers (p=0.0103). The functional activity of vWF was also significantly elevated (p<0.001). In addition, smokers had significantly greater levels of cardiac troponin I than nonsmokers (p<0.001); a positive correlation was identified between its level and the number of cigarettes smoked (p<0.001).

According to Gastaldelli et al. (16), dyslipidemia commonly seen in smokers arises from elevated LDL, HDL, and triglyceride concentrations. Confirmation for this comes from the recovery of lipid metabolism that occurs with smoking cessation; specifically, LDL levels decline, and

HDL levels typically increase soon after smoking has stopped. The disruption to lipid metabolism is attributed to the various components of tobacco smoke. For example, Gastaldelli et al. (16) stated that nicotine stimulates lipolysis, resulting in the elevation of free fatty acids released into the blood. Nicotine also upregulates the synthesis of lipoproteins and the pro-atherosclerotic LDL in particular; meanwhile, oxidative stress and platelet aggregation increase in response to carbon monoxide and other smoking-generated oxidant gases.

In their research, Alsalhen and Abdalsalam (26) stated that smokers' mean serum concentration of total bilirubin is considerably lower. Cigarette smoking has a positive relationship with hemoglobin, which is responsible for bilirubin concentration. Consequently, the differences in bilirubin concentrations between smokers and non-smokers may be linked to differing hemoglobin concentrations. As agreed with this study, Alsahlen and Abdalsalam (26) concluded that the association between total blood bilirubin concentration and smoking was independent of hemoglobin and that bilirubin levels were consistently lower in smokers than in non-smokers. The researchers hypothesize that the drop in serum bilirubin levels may be caused by the increase in free radicals caused by cigarette smoking.

The effects of cigarette smoke on body organs extend beyond those that come into direct contact with the smoke (27). For example, smoke affects the liver as it processes and eliminates smoke-related toxins. Albumin binds bilirubin, cations, fatty acids, hormones, thyroxine, water, and many drugs metabolized in the liver. Consequently, an inverse relationship exists between an individual's serum concentration of albumin and their state of health (28).

As explained by Clerici et al. (29), the biosynthesis of albumin occurs in the liver; in heavy smokers, serum albumin levels are significantly lower than in control subjects. Chemicals in cigarette smoke cause the carbonylation of albumin, which alters albumin's antioxidant properties. Furthermore, proteolytic activity is elevated by the high level of free radicals. The work by Mehjabeen and Ashraf (30) found that smokers excrete around 2.8 times more albumin excretion rate, which is about 2.8 times higher in smokers than non-smokers with the development and progression of diabetic renal disease damage.

Ge et al. (31) argued that a plausible explanation for the significantly lower level of plasma albumin, observed in the heavy smokers who took part in this present study, is that most of the proteins are synthesized in the liver. Albumin is the most abundant serum protein; therefore, an early indicator or impairment of liver function could be a significant increase in serum albumin level.

The effects of the chemicals in cigarette smoke on the serum protein profile can be exerted both directly and indirectly (32). Serum albumin, which is involved in the transport of biomolecules, is at normal levels in non-smokers. The chemicals in the smoke interfere directly with albumin's binding properties, causing the albumin to be broken down by the liver and eliminated via the kidneys (i.e., hypo-albuminuria).

Blood sugar levels are higher in smokers than they are in non-smokers or past smokers (33). In diabetic patients, stopping smoking yields immediate health benefits that build as the smoke-free duration gets longer. Gastaldelli et al. (16) stated that cigarette smoking is an independent risk factor for the development of type 2 diabetes and insulin resistance. The evidence that smoking stimulates insulin resistance is overwhelming.

Early identification of damage to the endothelium helps diagnose atherosclerosis (18). As vWF is unique to endothelial cells and is a reliable indicator of endothelial damage, there is value in evaluating vWF's functional activity. Al-Awadhi et al. (17) think that O2 free radicals cause the formation of cytotoxic lipid peroxidase. CO, nicotine, and other chemicals in the smoke also cause vWF to be released.

Moreover, compared to non-smokers, Sinha et al. (34) noted that smokers also have higher levels of fibrinogen, which is also a marker of inflammation elevated in smokers. Wannamethee et al. (18) said that high fibrinogen affects how thick the blood is, how fibrin forms, and how platelets stick together. This makes smokers more likely to get heart disease. In this study, persons who had smoked for more than ten years had higher fibrinogen levels. Long-term smoking may result in cardiovascular disease.

The present study indicated that cardiac troponin I levels were higher among heavy smokers who smoke >1 pack/day than mild smokers who smoke <1 pack/day, but the difference in mean troponin I levels between both groups was not significant.

Our findings are not in agreement with those of a recent study by Skranes et al. (10), who reported that non-smokers had significantly higher concentrations of high-sensitivity cardiac troponin I than smokers ($P<0.001$). They added that an elevated cardiac troponin I concentration is a marker of subclinical damage to the myocardium. High levels are considered strongly predictive of heart failure and death from cardiovascular malfunction. Therefore, the high levels of troponin I that this study found in heavy smokers could be a sign of early endothelial damage.

The observed difference between our findings and those of Skranes et al. (10) regarding concentrations of high-sensitivity cardiac troponin I according to smoking status may be attributed to differences in characteristics of included samples in both studies, sampling technique, or even differences in types of cigarettes smoked by participants. However, more research is needed to determine how smoking affects high-sensitivity cardiac troponin I level.

The present study showed a significant impact of smoking on both body mass index and waist-to-hip ratio. There were significantly more obese smokers than non-smokers.

Smokers also had a significantly higher waist-to-hip ratio than non-smokers ($p<0.001$ for both males and females). There were significantly positive correlations between the number of cigarettes smoked daily and both body mass index and waist-to-hip ratio. Moreover, those who smoked for over ten years had a significantly higher body mass index.

Graff-Iversen et al. (35) showed a positive association between current smoking and waist-hip ratio, concluding that smoking enhances abdominal obesity as an unhealthy outcome. Also, Morris et al. (36) used a Mendelian randomization approach to indicate a causal effect of tobacco smoking on abdominal fat accumulation.

Our study showed that participant smokers had significantly higher systolic and diastolic blood pressures than non-smokers. Moreover, the number of daily cigarettes correlated positively and significantly with participants' systolic and diastolic blood pressures. In addition, those who smoked for over ten years had significantly higher systolic and diastolic blood pressures. Furthermore, systolic and diastolic blood pressures were significantly higher among those who smoked >1 pack of cigarettes daily.

Similarly, in the study of Primatesta et al. (37), 24-hour ambulatory blood pressure monitoring for participants revealed that smokers maintained a higher mean daytime ambulatory systolic blood pressure than non-smokers. They stressed that smoking causes an acute increase in blood pressure and is associated with malignant hypertension. The high blood pressure among smokers was explained by nicotine acting as an adrenergic agonist, mediating local and systemic catecholamine release and possibly the release of vasopressin. However, several epidemiological studies have found that blood pressure levels among cigarette smokers were the same or lower than non-smokers.

Study Limitations

Some limitations are to be considered in the present study. First, the smoking habit was determined through self-reports. Therefore, further studies should include confirmation by objective assessment, such as exhaled carbon monoxide and nicotine testing. Second, the generalizability of results is limited by the consecutive non-random sampling method used, which had a limited sample size, and was a single-center study.

Based on the findings of the present study, it can be concluded that heavy cigarette smoking is significantly higher among males than females. Heavy smokers and those who smoke for longer durations have a significantly worse lipid profile and higher vWF functional activity. Heavy smoking is associated with significantly higher plasma total cholesterol, LDL cholesterol, non-HDL cholesterol, and vWF activity but significantly lower plasma albumin. The number of cigarettes smoked daily correlates significantly with plasma levels of total cholesterol, LDL, non-HDL cholesterol, and vWF activity. Cardiac troponin I levels are higher among smokers than non-smokers, indicating higher susceptibility to heart failure and cardiovascular mortality among smokers.

Therefore, smoking cessation programs should be implemented, and comprehensive health promotion programs need to be widely applied. Primary healthcare providers could actively participate in reducing smoking-induced consequences related to cardiometabolic diseases by initiating innovative health promotion programs. Since the present study followed a cross-sectional design, further prospective studies with larger sample sizes are needed to supplement the results of this study.

References

1. Asaria P, Chisholm D, Mathers C, Ezzati M, Beaglehole R. Chronic disease prevention: health effects and financial costs of strategies to reduce salt intake and control tobacco use. Lancet 2007; 370:2044–2053.
2. Nilsson PM, Fagerström KO. Smoking cessation: it is never too late. Diabetes Care. 2009; 32 Suppl 2: S423-5.
3. Al-Doghether MH. Do we need national guidelines for smoking cessation? Annals of Saudi Medicine 2001; 21(1-2):3-4.
4. Khanagar SB, Siddeeqh S, Khinda V, Khinda P, Divakar DD, Jhugroo C. Impact of electronic cigarette smoking on the Saudi population through the analysis of literature: A systematic review. J Oral Maxillofac Pathol 2019; 23(3): 473.
5. Mehta H, Nazzal K, Sadikot RT. Cigarette smoking and innate immunity. Inflamm. Res. 2008; 57(11): 497-503. Mercado C, Jaimes EA. Cigarette smoking as a risk factor for atherosclerosis and renal disease: novel pathogenic insights. Curr Hypertens Rep 2007; 9:66–72.
6. Mannino DM. Why won't our patients stop smoking? The power of nicotine addiction. Diabetes Care. 2009; 32 Suppl 2: S426-8.
7. Lucan SC, Katz DL. Factors associated with smoking cessation counseling at clinical encounters: The Behavioral Risk Factor Surveillance System (BRFSS) 2000. Am J Health Promot 2006; 21:16–23
8. Benowitz NL. Neurobiology of nicotine addiction: implications for smoking cessation treatment. Am J Med 2008; 121: S3–S10.
9. El-Zayadi A. Heavy smoking and liver. World J Gastroenterol 2006;12(38):6098-101.
10. Skranes JB, Lyngbakken MN, Hveem K, Rosjo H, Omland T. Tobacco Consumption and High-Sensitivity Cardiac Troponin I in the General Population: The HUNT Study. J Am Heart Assoc. 2022; 11: e021776. Doi: 10.1161/JAHA.121.021776.
11. Mechanick JI, Rosenson RS, Pinney SP, Mancini DM, Narula J, Fuster V. Coronavirus and Cardiometabolic Syndrome. Journal of the American College of Cardiology 2020; 76(17):2024-2035.
12. Ezzati M, Lopez AD. Regional, disease-specific patterns of smoking-attributable mortality in 2000. Tob Control 2004; 13: 388-95.
13. World Health Organization. Global Action Plan for the Prevention and Control of Noncommunicable Diseases

A 2013-2020. Geneve, Switzerland: World Health Organization, 2013.
14. de Heens GL, Kikkert R, Aarden LA, Van der Velden U, Loos BG. Effects of smoking on the ex vivo cytokine production in periodontitis. J. Periodontal. Res. 2009; 44(1): 28-34.
15. Zhong CY, Zhou YM, Pinkerton KE. NF-kappa B inhibition is involved in tobacco smoke-induced apoptosis in the lungs of rats. Toxicol. Appl. Pharmacol. 2008; 230(2): 150-158.
16. Gastaldelli A, Folli F, Maffei S. Impact of Tobacco Smoking on Lipid Metabolism, Body Weight and Cardiometabolic Risk. Current Pharmaceutical Design, 2010, 16, 2526-2530.
17. Al-Awadhi AM, AlFadhli SM, Mustafa NY, Sharma PN. Effects of Cigarette Smoking on Hematological Parameters and von Willebrand Factor Functional Activity Levels in Asymptomatic Male and Female Arab Smokers. Med Princ Pract 2008; 17:149–153. DOI: 10.1159/000112970.
18. Wannamethee SG, Shaper AG. Cigarette smoking and serum liver enzymes: the role of alcohol and inflammation. Ann Clin Biochem 2010; 47: 321–326.
19. Alrabah M, Gamaleddin I, Allohidan F. International approaches to tobacco-use cessation programs and policy for adolescents and young adults in Saudi Arabia. Curr Addict Rep. 2018;5(1):65-71. doi:10.1007/s40429-018- 0188-9
20. Moradi-Lakeh M, El Bcheraoui C, Tuffaha M, Daoud F, Al Saeedi M, Basulaiman M, et al. Tobacco consumption in the Kingdom of Saudi Arabia, 2013: Findings from a national survey. BMC Public Health. 2015;15(1):611- 621. doi:10.1186/s12889-015-1902-3.
21. Algabbani AM, Almubark R, Althumiri N, Alqahtani A, BinDhim N. The prevalence of cigarette smoking in Saudi Arabia in 2018. FDRJ. 2018;1(1):1-13. doi:10.32868/rsj.v1i1.22.
22. Cohen J. Statistical power analysis for the behavioral sciences (2nd ed.). Hillsdale, 1988; NJ: Lawrence Earlbaum Associates.
23. Al-Rubeaan K, Bawazeer N, Al Farsi Y, Youssef AM, Al-Yahya AA, AlQumaidi H, et al. Prevalence of metabolic syndrome in Saudi Arabia—A cross sectional study. BMC Endocr. Disord 2018; 18: 3–9.
24. Ansari K, Farooqi FA. Comparison and prevalence of smoking among Saudi females from different Departments of the College of Applied Medical Sciences in Dammam. Int J Health Sci (Qassim). 2017; 11(5): 56–62.
25. Abdalla AM, Al-Kaabba AF, Saeed AA, Abdulrahman BM, Raat H. Gender differences in smoking behavior among adolescents in Saudi Arabia. Saudi Med J. 2007; 28:1102–8.
26. Alsalhen KS, Abdalsalam RD. 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.
27. Sokolowska M, Wszelaka Rylik, M, Poznanski J. And Bal, W. Spectroscopic and thermodynamic determination of three distinct binding site for Co (II) in human serum albumin. J. Inorg. Biochem. 2009; 103(7): 1005-1013.
28. Farrugia, A., 2010. Albumin Usage in Clinical Medicine: Tradition or Therapeutic? Transfus. Med. Rev., 24(1): 53-63.
29. Clerici, M., Colombo, G., Secundo, F., Gagliano, N., Colombo, R., Portinaro, N., Giustarini., D., Milzani, A., Rossi, R. And Dalle-Donne, I., 2014. Cigarette smoke induces alterations in the drug-binding properties of human serum albumin. Blood Cells Mol. Dis., 52(4): 166-74.
30. Mehjabeen NR, Ashraf S. Effects of cigarette smoking on serum proteins profile in male active and passive smokers. Punjab Univ. J. Zool., 2017; 32 (2): 209-215.
31. Ge P, Yang H, Lu J, Liao W, Du S, Xu Y, et al. Albumin Binding Function: The Potential Earliest Indicator for Liver Function Damage. Gastroenterol Res Pract. 2016; 2016: 5120760. doi: 10.1155/2016/5120760.
32. Ramamurthy, V, Raveendran, S, Thirumeni, S. And Krishnaveni, S, 2012. Biochemical changes of cigarette smokers and non-cigarette smokers. Int. J. Adv. Lif. Sci., 1: 12-22.
33. Shaktour AT, Najjar AK, Alhabrush RA. Effect of smoking on fasting blood glucose level. Lebda Medical Journal 2019; 6:235-237.
34. Sinha S, Luben RN, Welch A, Bingham S, Wareham NJ, Day NE, Khaw KT: Fibrinogen and cigarette smoking in men and women in the European Prospective Investigation into Cancer in Norfolk (EPIC-Norfolk) population. Eur J Cardiovasc Prev Rehabil 2005;12: 144–150.
35. 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: 1439.
36. Morris RW, Taylor AE, Fluharty ME, et al. Heavier smoking may lead to a relative increase in waist circumference: evidence for a causal relationship from a Mendelian randomization meta-analysis. The CARTA consortium BMJ Open. 2015; 5: e008808. Doi: 10.1136/bmjopen-2015-008808.
37. Primatesta P, Falaschetti E, Gupta S, Marmot MG, Poulter NR. Association Between Smoking and Blood Pressure: Evidence from the Health Survey for England. Hypertension 2001; 37:187–193. Doi: 10.1161/01.HYP.37.2.187.