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Study of the Relationship between the Nicotine and Lipid Profile with Some Hematology Parameters in Serum of Smoker and Non-Smoker Blood Samples

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Abstract

The effect of cigarette smoking on lipid profile and CBC (Complete Blood Count) of blood was investigated in this study. Spectrophotometric methods were used to estimate the amounts of nicotine, cholesterol, triglycerides, and HDL-cholesterol. The findings revealed that cigarette smokers and the duration of smoking had significantly higher levels of nicotine and cholesterol when compared to non-smokers. The data also revealed differences in the contents of hematological parameters between smokers' and non-smokers' blood samples, with high counts of WBC, MCHC, HGB, and PLT clearly visible in the smokers' samples. However, there was no discernible effect on RBC, MCV, or MCH counts in the trial. Smoker samples had high PLT values (243.8 ± 84.26), while non-smoker samples had lower PLT values (229.3 ± 30.07 on average).

Keywords

Non-Smoker, Smoker, Relationship between the Nicotine and Lipid

1. Introduction

High blood levels of total cholesterol (TC 240 mg/dL), low-density lipoprotein cholesterol (LDL-C 160 mg/dL), and triglyceride (TG 200 mg/dL) and a low amount of high-density lipoprotein cholesterol (HDL-C 40 mg/dL) characterize dyslipidemia [1]. Both individually and in combination, serum lipid components are linked to the risk of cardiovascular disease (CVD) [2]. Cigarette smoking is a

well-known primary risk factor for atherosclerosis and cardiovascular disease [3]. These lipids that have been damaged may induce atherogenesis and atherosclerosis [4]. However, the findings varied between research, and the impact of smoking on lipid levels is not well established [5].

Dyslipidemia affects about four out of ten (40.5 percent) persons over the age of 30, with the severity being substantially higher in men. The current study aims to explore the independent connection between cigarette smoking and blood lipid levels in smokers and non-smokers, according to the Organization for Economic Cooperation and Development Health Statistics 2017, which is still higher than the average of 23 percent [6]. The aim of this study was to determine the impact of smoking on the lipid profile and a few blood parameters in serum samples taken from a Libyan population.

2. Materials and Methods

2.1. Sampling and Preparation of Blood Samples

In this study, two separate groups of serum blood samples were used. The first group consisted of fifty samples taken from male smokers, whereas the second was made up of non-smokers. Blood is extracted from veins and placed in blood tubes. The serum was extracted from the cells by centrifugation at 3000 rpm for a few minutes and then stored in the freezer after the Complete Blood Count (CBC) was measured. There were no major health issues among the participants.

2.2. Laboratory Tests

2.2.1. Nicotine Content

The nicotine concentration of blood samples was tested using the following method: Based on [7], the extraction processes were carried out with a little modification. A screw-capped glass test tube was used to aliquot 0.5 mL of plasma. 200 l of 2.5 M NaOH was added, and the mixture was stirred for 5 minutes at 3000 rpm. For one-step single extraction, a 10 ml mixture of dichloromethane-diethylether (1:1 v/v) was utilized, followed by vortex mixing at 3000 rpm for 5 minutes. After centrifugation at 3000 rpm for 2 - 4 minutes, the organic layer was transferred to a fresh glass tube containing 40 l of 0.25 M HCl.

The organic phase was then evaporated at 35˚C until dry, and then reconstituted to 2 ml with a mixture of 0.2973 g KH2PO4, 820 ml distilling water, and 180 ml methanol. The plasma samples were then analyzed using a UV-Visible Spectrophotometer at 258 nm, and the nicotine content of the samples was calculated using the standard curve of standard nicotine solutions.

2.2.2. Complete Blood Count (CBC)

At several laboratories in El-beida city, Complete Blood Count was measured using (DIAGON D-CELL 60). White blood cells (WBC), red blood cells (RBC), platelets count (PCT), mean platelets volume (MPV), hematocrit (HCT), hemoglobin (Hb), Mean Corpuscular Hemoglobin (MCH), and Mean Corpuscular Hemoglobin Concentration (MCHC) were all measured during a complete blood count.

2.2.3. Lipid Profile Test

Cholesterol, Triglycerides, and HDL-Cholesterol values in the blood were provided. All lipid profile tests and complete blood counts (CBC) were calculated using a kit approach on an automated analyzer in all patients (Spectrophotometer, 4040 V5).

2.3. Statistical Treatments

The soft ware computer program of (**R** program, version, 2013) was used for draw the Figure and calculated the correlation coefficient values of the obtained results.

3. Results and Discussion

3.1. Nicotine

For the smokers and non-smokers blood tests, respectively, the nicotine substance was modified within the ranges of (11 - 52 μg/ml) and (0.9 - 9.07 μg/ml), as shown in Table 1 & Table 2 and Figure 1. The findings revealed high levels of nicotine in smokers' blood tests, which can be attributed to a variety of factors

Table 1. The Nicotine contents of the collected blood samples of smokers (μg/l).

Sample No.	*Smoking Period* (*Year*)	Nicotine (μg/l)	*Sample No.*	*Smoking Period* (*Year*)	Nicotine (μg/l)	*Sample No.*	*Smoking Period* (*Year*)	Nicotine (μg/l)
1	Few months	12.5	20	2	18	42	30	52
2	1	11	21	6	19.8	43	11	32.1
3	2	16	22	12	29.5	44	8	19
4	8	33.5	23	13	33.9	45	7	25
5	4	28.8	24	10	36	46	12	33
6	6	20	25	11	30.3	47	8	28.9
7	4	22	26	18	34.9	48	4	17
8	4	28	27	18	46	49	3	27
9	1	30	28	9	30	50	7	44.6
10	9	29.6	29	16	37.0	42	30	52
11	6	29	30	28	42	43	11	32.1
12	6	19	31	12	38	44	8	19
13	8	38	32	18	40.9	45	7	25
14	10	34.9	33	23	43.6	46	12	33
15	9	30	34	32	40.7	47	8	28.9
16	9	33	35	4	21	48	4	17
17	12	28.2	36	13	37	49	3	27
18	8	29.4	37	20	38	50	7	44.6
19	7	29	38	10	20			

Table 2. The Nicotine contents of the collected blood samples of non-smokers (μg/l).

Sample No.	Nicotine (μg/l)
1	2.33
2	4.45
3	3.15
4	8.8
5	5.8
6	6.9
7	3.60
8	7.1
9	2.11
10	4.08
11	8.1
12	6.7
13	4.0
14	0.9
15	5.3
16	1.9
17	3.0
18	9.07
19	2.8
20	7
Average	4.85
SD	2.45

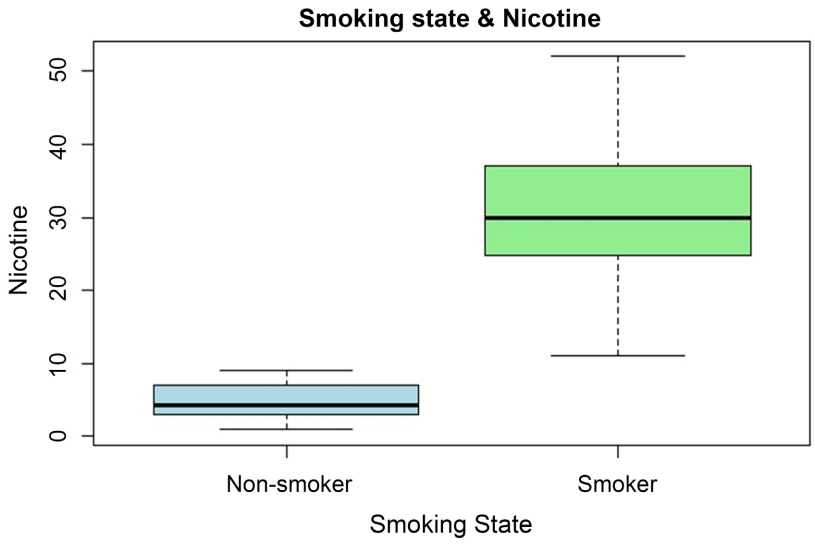

Figure 1. The distribution of nicotine contents (Box blot) for smokers and non-smokers blood samples.

such as cigarette type, age, nicotine intake, and certain disorders. This study used the smoking period as the most important factor in comparing the nicotine substance in the various tests. As a result, the study recorded the relative impact of the smoking period on the nicotine substance. On the other hand, in non-smokers' tests, moo substance of nicotine emerged, as did the proximity of high amounts of nicotine [8].

It has been claimed that meals or foods may have an impact on nicotine levels in the human body; nonetheless, Because of the high degree of hepatic extraction, nicotine clearance should be influenced by liver blood flow. As a result, physiological events such as meals, posture, exercise, or medicines that disrupt hepatic blood flow are expected to alter nicotine metabolism. Meals ingested during a steady state nicotine infusion result in a continuous decrease in nicotine concentrations, with the maximum effect occurring 30 - 60 minutes after the meal ends [9]. After a meal, hepatic blood flow increases by around 30%, and nicotine clearance increases by about 40%.

The kidney is also one of the most important components that has a significant impact on nicotine levels in human body tissues. According to some research, nicotine is expelled by glomerular filtration and tubular secretion, with varying re-absorption depending on pH. Renal clearance of nicotine ranges between 35 and 90 ml min1 when pH is not regulated, accounting for around 5% of total clearance. Nicotine is mainly ionized in acid urine, and tubular re absorption is reduced; renal clearance can reach 600 ml min1 depending on urinary flow rate [10]. A greater proportion of nicotine is unionized in alkaline urine, allowing net tubular re absorption with a renal clearance as low as 17 ml min 1 (urine pH 7.0).

Furthermore, several researches have found a link between nicotine and genetic variables, with hereditary contributions to nicotine and cotinine renal clearances being assessed in a twin study. These data show that, even in the presence of net re absorption, nicotine and cotinine re absorption are active processes regulated by the genetics of re absorptive transporters, or that the active secretor component of renal clearance has a significant impact on clearance. It's possible that the hereditary component of nicotine re absorption clearance is determined by the matching variable in re absorption transporters. Renal dysfunction, as previously indicated, affects overall renal clearance as well as nicotine and cotinine metabolic clearance [9].

In this investigation, the contents of nicotine differed by smoking group and smoking period, with significant amounts of nicotine detected in several samples after ten years of smoking (**Table 1**), and a high level (52 g/ml) detected after thirty years of smoking (**Table 2**). Low levels of nicotine have also been linked to disorders such as (nicotine metabolism), which may be exacerbated in the elderly by lower liver blood flow, despite the fact that no decrease in liver protein levels or nicotine metabolism in liver microsomes has been identified as a result of age. The nicotine levels in non-smoking blood samples indicated no variations in steady nicotine plasma levels, as illustrated in **Figure 1**, Box blot, where the

nicotine contents were between (1 and 2).

According to some research, dietary sources of nicotine may be a potential confounder of cotinine (one of nicotine's isomers) levels utilized in secondhand smoke exposure testing. Nicotine can be found in a variety of foods [8]. Nicotine levels in meals, on the other hand, are fairly modest. The amounts of cotinine produced by a diet high in nicotine-containing foods are lower than those reported in individuals exposed to moderate levels of secondhand smoke, according to nicotine levels in foods and the normal daily consumption of various nicotine-containing meals. In general, the new study supports prior research that found a direct link between smoking and nicotine levels in blood samples [10].

3.2. The Hematological Parameters

The collected findings showed that the contents of various CBC values in the smokers blood samples were ranged as follows: (10.1 - 17.5), (4.0 - 23.3), (3.5 - 6.6), (139 - 603), and (28.7 - 52.5) for Hemoglobin, WBC, RBC, PLT, and HCT, respectively (**Table 3**). The aforementioned values in the blood of non-smokers samples, on the other hand, were ranged as follows: (13.1 - 16.1), (3.5 - 10.0), (4.9 - 5.9), (168 - 285), and (39.0 - 50.0), for the above parameters, respectively, (**Table 4**).

Table 3. The hematological parameters of the collected blood samples of smokers.

Samples No.	The Smoking Period years	Parameters							
		WBC $\times 10^9$/L	RBC $\times 10^{12}$/L	HGB g/dl	HCT %	MCV fl	MCH pg	MCHC g/dl	PLT $\times 10^9$/L
1	Few months	10.1	5.48	15.4	44.1	80.5	28.1	34.9	140
2	1	10.8	6.61	16.2	46.9	71.0	24.5	34.5	180
3	2	13.3	3.95	12.0	33.5	84.8	30.4	35.8	350
4	8	8.9	6.13	12.1	37.2	60.8	19.7	32.5	295
5	4	18.8	5.08	12.7	36.3	71.5	25.0	35.5	515
6	6	15.5	5.11	15.2	44.9	87.9	29.7	33.9	217
7	4	5.6	5.37	15.2	44.9	83.7	28.3	33.8	169
8	4	6.3	5.57	14.8	43.5	78.1	26.5	34.0	146
9	1	5.5	4.94	14.0	40.0	81.0	28.3	35.0	173
10	9	5.2	4.76	13.8	40.9	86.0	28.9	33.7	221
11	6	6.5	5.90	16.1	47.3	80.3	27.2	34.0	240
12	6	13.3	4.26	11.6	32.0	75.1	27.2	36.3	256
13	8	9.8	5.33	16.0	44.9	84.3	30.0	35.6	139
14	10	13.8	5.14	14.5	43.3	84.4	28.2	33.4	274
15	9	8.6	5.22	14.8	44.1	84.5	28.3	33.5	295
16	9	4.6	4.95	13.3	40.5	82.0	26.8	32.8	221

Continued

17	12	6.2	5.18	15.0	43.7	84.5	28.9	34.3	288
18	8	13.7	4.96	13.8	39.7	80.0	27.8	34.8	283
19	7	6.5	5.18	14.5	42.9	83.0	27.9	38.7	160
20	2	8.6	5.11	13.8	44.7	87.6	27.0	30.8	267
21	6	5.4	5.31	16.0	51.4	96.9	30.1	31.1	136
22	12	16.3	5.95	10.4	32.8	55.1	17.5	31.7	312
23	13	7.6	5.09	13.7	39.1	76.8	26.9	35.0	250
24	10	5.2	5.78	16.1	46.7	80.9	27.8	34.4	184
25	11	18.0	3.51	10.1	28.7	81.8	28.8	35.2	603
26	18	7.8	5.44	15.3	45.0	82.9	28.1	34.0	197
27	18	4.0	4.76	14.5	41.8	88.0	30.4	34.6	289
28	9	13.8	4.13	11.1	31.1	75.3	26.9	35.7	251
29	16	8.2	5.03	14.6	43.5	86.5	29.0	33.5	278
30	28	17.3	5.44	16.5	44.8	82.4	30.3	36.8	190
31	12	10.2	4.65	12.0	33.8	72.7	25.8	35.5	361
32	18	14.1	5.14	17.5	47.6	92.6	34.0	36.8	226
33	23	18.3	3.67	13.7	40.2	86.1	29.3	34.1	199
34	32	13.4	4.75	13.2	37.9	79.8	27.8	34.8	282
35	4	7.2	5.30	15.0	44.5	84.0	28.3	33.7	207
36	13	8.1	5.57	16.4	52.5	94.3	29.4	31.2	256
37	20	5.8	5.26	15.5	45.6	86.7	29.4	33.9	168
38	10	5.4	5.06	14.0	42.8	84.6	27.6	32.7	169
39	5	8.2	4.98	15.60	38.50	75.10	27.18	33.17	200
40	4	9.4	5.01	15.20	30.40	77.20	26.20	35.20	199
41	15	8.6	5.62	16.80	37.40	71.18	31.70	30.45	255
42	30	6.7	5.40	16.45	44.70	83.16	33.30	37.18	230
43	11	10.5	4.75	14.60	40.25	79.15	31.18	36.60	260
44	8	7.9	5.21	13.55	39.17	78.20	29.50	34.12	215
45	7	8.4	5.30	14.25	38.20	70.40	28.40	38.14	218
46	12	9.8	5.45	15.40	37.30	69.15	29.80	31.50	195
47	8	7.9	4.85	16.45	34.15	80.75	27.50	30.60	258
48	4	8.6	4.70	14.80	39.20	69.44	28.30	32.16	260
49	3	9.4	4.85	15.07	47.10	72.20	29.22	33.14	281
50	7	8.12	4.78	14.30	42.15	65.16	30.70	37.50	233
Average	-	9.82	5.09	14.45	41.07	79.78	28.18	34.24	243.8
ST.D	-	4.33	0.56	1.65	5.31	8.02	2.69	1.92	84.26

Table 4. The hematological parameters of the collected blood samples of non-smokers.

Samples No.	Parameters							
	WBC $\times 10^9$/L	RBC $\times 10^{12}$/L	HGB g/dl	HCT %	MCV fl	MCH pg	MCHC g/dl	PLT $\times 10^9$/L
1	3.5	5.95	15.4	46.2	77.7	25.8	33.3	223
2	9.2	4.95	13.1	39.0	78.9	26.4	33.5	285
3	6.5	5.03	16.0	47.0	93.5	31.8	34.0	206
4	7.3	4.91	14.1	43.5	88.6	28.7	32.4	264
5	5.7	5.59	16.1	50.0	89.5	28.8	32.2	229
6	6.2	5.03	13.9	44.0	87.5	27.6	31.5	226
7	5.8	5.26	15.5	45.6	86.7	29.4	33.9	168
8	5.4	5.06	14.0	42.8	84.6	27.6	32.7	169
9	4.6	4.95	13.3	40.5	82.0	26.8	32.8	221
10	10.0	5.11	14.2	41.7	81.7	27.7	34.0	216
11	6.5	4.50	14.8	44.30	80.10	27.20	31.70	202
12	6.20	5.18	15.2	40.20	79.20	26.18	33.20	210
13	7.19	5.20	13.8	41.18	73.18	28.70	34.15	260.5
14	8.4	4.95	14.5	47.17	77.40	28.15	31.20	245
15	7.9	4.70	13.75	45.20	73.70	26.60	30.70	257.6
16	8.5	5.15	14.30	46.18	80.20	27.40	33.15	260
17	6.40	5.30	14.80	44.20	85.14	28.50	32.18	247
18	8.50	4.75	13.75	41.90	82.20	29.20	30.20	220.7
19	6.2	5.20	14.60	47.18	78.14	30.40	33.18	235.5
20	5.8	4.98	15.01	40.20	79.16	64.14	32.19	240.9
Average	6.78	5.08	14.50	43.90	81.95	29.85	32.60	229.3
ST.D	1.59	0.31	0.84	2.93	5.30	8.20	1.12	30.07

3.2.1. WBC

Increases in WBC and platelet (PLT) counts have been seen in several studies as one of the acute effects of smoking on the hematological system [11]. A relationship was discovered between smoking and WBC levels. WBC counts were found to be greater in smokers' blood samples. This was also observed in this investigation, where greater WBC (9.82 4.43) values were found in smokers' blood samples compared to WBC (6.78 1.59) in non-smokers' blood samples, (Table 3). Additionally, the correlation matrix revealed a relative positive connection between nicotine and WBC (0.12), Table 5. Many studies have concluded that the WBC count is arguably the most useful, inexpensive, and straightforward biomarker for endothelium injury, and the current findings are in line with that conclusion. In certain research, it was also discovered that the median total leukocyte.

Table 5. The correlations matrix between nicotine and the studied hematological parameters.

	Nicotine	PLT	MCHC	MCH	MCV	HCT	HGB	RBC	WBC
Nicotine	1								
PLT	0.12	1							
MCHC	0.39	0.05	1						
MCH	−0.03	−0.08	0.02	1					
MCV	−0.08	−0.23	−0.06	0.27	1				
HCT	−0.2	−0.49	−0.021	0.13	0.52	1			
HBG	0.07	−0.57	−0.08	0.29	0.39	0.68	1		
RBC	−0.03	−0.39	−0.14	−0.16	−0.17	0.47	0.5	1	
WBC	0.26	0.51	0.31	−0.11	−0.18	−0.46	−0.4	−0.38	1

According to several research, nicotine, which is a component of cigarette smoke, promotes catecholamine release and causes cortisol levels to rise [12]. Direct damage resulting from modifications in epithelial and endothelial surfaces and/or cytokine levels (particularly IL-6) produced by components of cigarette smoke can cause increases in peripheral blood WBC counts and alterations in WBC function (Smith *et al.*, 2003). The link between smoking and the number of white blood cells in the blood has been well documented [13]. Smokers have larger white blood cell counts than nonsmokers, according to a number of studies [14].

3.2.2. Hemoglobin (HGb)

In this study, the hemoglobin values for smokers and non-smokers blood samples varied between (10.1% - 17.5%) and (13.1% - 16.1%). However, many smokers' blood samples showed relative higher hemoglobin values, such as 17.5, 16.80, 16.50, 16.45, and 16.20, whereas most non-smokers' samples showed hemoglobin values below 16 percent. The high HGb values are due to smoking, and the correlation matrix showed a relative positive (0.07) relationship between nicotine and hemoglobin (**Table 5**), which is consistent with many studies [15].

Smoking active tobacco cigarettes, both acute and chronic, has been shown to raise white blood cell counts. In addition, past epidemiological researchers have found that long-term passive tobacco cigarette smoking can raise white blood cell counts.

WBC, red blood cells, Hb, and HTC levels were shown to be significantly higher in a study on the effects of smoking on hematological parameters, but MCV and MCH counts were reduced [16]. These changes have been linked to atherosclerosis, polycythemia vera, chronic obstructive pulmonary disease, and cardiovascular disorders, as well as an increased risk of atherosclerosis, polycythemia, COPD, and cardiovascular disease in smokers [17].

3.2.3. Hematocrit (HCT) %

Ulmonary disease and cardiovascular disease are two of the most common dis-

eases. The hematocrit values of smokers' blood samples ranged from 28.7 to 52.5%, while non-smokers' blood samples had hematocrit values ranging from 39.0 to 47. The effects of smoking on HCT levels were also observed in certain smokers' blood samples, with values such as 52.5, 51.4, 47.3, 46.9, 46.7, and 44.9 being reported. On the other hand, the HCT of non-smoker blood samples was often below 45 percent, with the exception of one sample (50 percent). In addition, there is an increased risk of atherosclerosis. Some studies indicated that smoking has a direct effect on hematocrit levels in blood samples; it was also noted that smokers' hematocrit values were significantly higher than non-smokers', which was consistent with prior findings [18]. Carbon monoxide (CO) is formed by incomplete combustion of carbon-containing materials in cigarette smoking, according to reports. In comparison to oxygen, CO has a very high affinity for hemoglobin. In smokers, higher levels of hematocrit and hemoglobin have been seen, and these increases are likely compensatory for CO exposure. Smokers have higher hematocrit and hemoglobin levels, which may lead to a hypercoagulable state [19].

3.2.4. RBC, PLT and Other CBC Parameters

For smoker blood samples, RBC and PLT values varied from (3.5 - 6.6) and (139 - 603), respectively, while non smoker blood samples had RBC and PLT values of (4.9 - 5.9) and (168 - 285). Smoking has been found to raise neutrophil, lymphocyte, monocyte, platelet counts (PLT), and RBC indexes in both men and women. This is consistent with the results of the current study, which show that high PLT values were found in many samples (243.8 ± 84.26), while non-smokers' PLT values fell on average (229.3 ± 30.07).

The correlation matrix (**Table 5**) revealed that PLT and nicotine (0.12) had relative positive values. In addition, the length of time spent smoking has an effect on some RBC levels in smokers' blood samples. When comparing smokers to nonsmokers, RBC values are generally higher in smokers [20]. The current CBC parameters agree with many studies on the effect of smoking on hematology blood analysis, where some studies comparing smokers and nonsmokers have shown increases in Hb, HCT, RBC, MCV, WBC, neutrophil, lymphocyte, eosinophil, and monocyte counts in both groups [21], investigated the impact of smoking on RBC, WBC, and Hb, and indicated increases in WBC counts and decreases in RBC, With a small reduction in the daily number of cigarettes smoked, the detrimental effects of smoking on hematological parameters improve. The correlations matrix between nicotine and the examined hematological parameters revealed that nicotine had a positive association with WBC, HGB, and MCHC, but was somewhat negative with RBC, MCV, and MCA. Different correlation coefficient values between nicotine and hematology blood measures show the presence of additional chemicals in cigarettes that may influence hematology blood parameters.

3.2.5. The Lipid Profile Parameters Results

The contents of lipid profile parameters of smokers and non-smokers are listed

in (Table 6 & Table 7) and box plots, (Figures 2-4) show that the contents of Triglycerides in smokers' blood samples ranged from 35 to 260, Cholesterol values from 88 to 220, and HDL contents from 20 to 109. On the other hand, the same values in non-smokers' blood samples ranged from 18 to 156, 95 to 167, and 36 to 79, respectively, as shown in (Table 7).

Table 6. The lipid profile of the collected blood samples of smokers.

Samples No.	The smoking Period	Parameters		
		Triglycerides Mg/dl	Cholesterol Mg/dl	HDL Mg/dl
1	Few Months	73	98	56
2	1	87	122	93
3	2	90	120	65
4	8	260	216	30
5	4	90	88	70
6	6	83	176	109
7	4	64	94	61
8	4	70	171	40
9	1	45	151	30
10	9	50	127	30
11	6	95	220	40
12	6	134	180	95
13	8	75	182	40
14	10	150	175	55
15	9	88	230	58
16	9	80	167	53
17	12	40	125	50
18	8	72	100	59
19	7	39	102	45
20	2	35	113	30
21	6	113	184	56
22	12	110	132	89
23	13	62	134	59
24	10	63	181	26
25	11	64	88	75
26	18	140	121	20
27	18	80	152	44
28	9	99	120	84
29	16	217	214	40

Continued

30	28	72	112	90
31	12	102	185	82
32	18	84	122	95
33	23	120	164	103
34	32	48	102	30
35	4	84	152	50
36	13	70	145	62
37	20	230	150	69
38	10	76	115	63
39	11	58	160	58
40	9	90	192	69
41	13	110	210	73
42	20	115	220	91
43	17	100	180	75
44	16	95	136	68
45	21	87	120	73
46	18	65	131	70
47	16	120	159	65
48	20	115	162	80
49	14	109	180	75
50	11	120	215	105
Average		96	151.9	59.1
ST.D		45.58	39.3	22.4

Table 7. The lipid profile parameters of the collected blood samples of non-smokers.

Samples No.	Parameters		
	Triglycerides Mg/dl	Cholesterol Mg/dl	HDL Mg/dl
1	54	130	61
2	125	137	60
3	95	137	36
4	156	140	59
5	87	170	60
6	57	95	47
7	38	95	52
8	76	115	63
9	80	167	53
10	18	116	79
11	101	155	83
12	120	148	70

Continued

13	99	147	68
14	115	161	65
15	93	160	66
16	110	168	73
17	105	157	59
18	102	158	62
19	130	171	65
20	99	160	58
Average	93	144.3	61.9
ST.D	32.5	23.5	10.5

Figure 2. The distribution of Cholesterol contents for smokers and non-smokers blood samples.

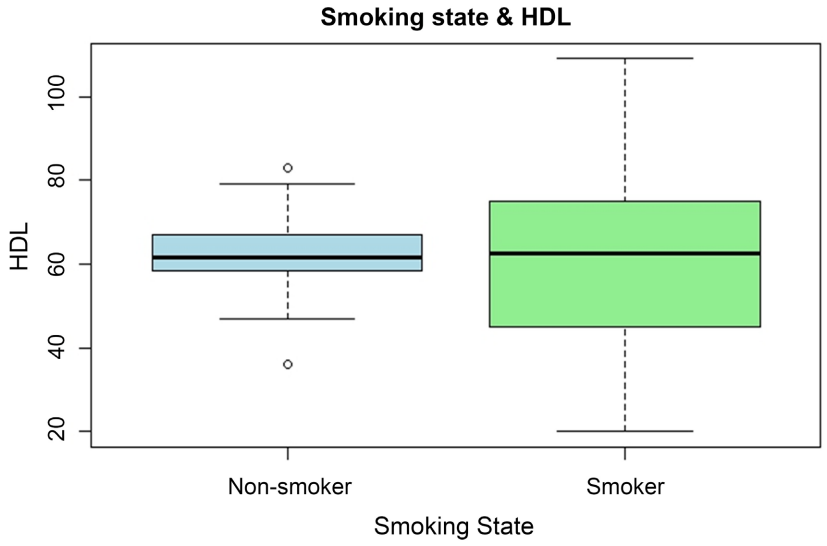

Figure 3. The distribution of HDL contents for smokers and non-smokers blood samples.

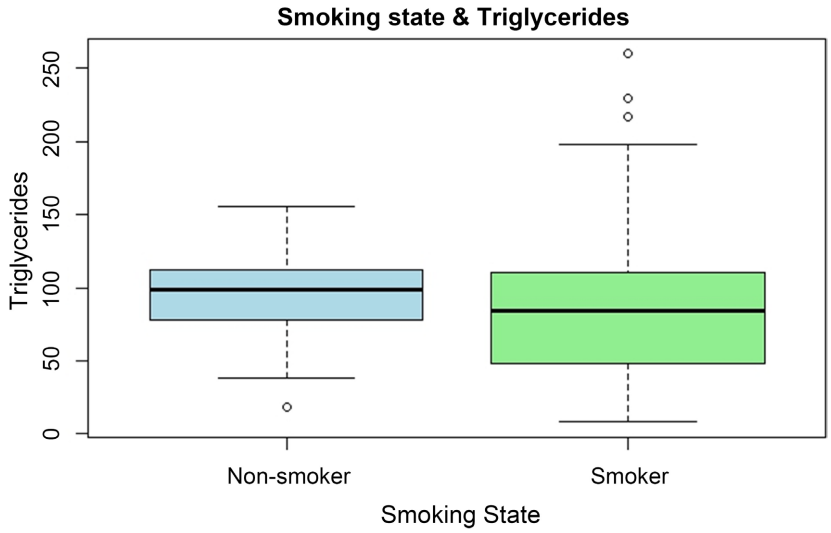

Figure 4. The distribution of triglycerides contents for smokers and non-smokers blood samples.

3.3. Effect of Cigarette Smoking on Lipid Profile

Lipids are involved in practically every element of biological existence. Serving as hormones or hormone precursors that aid digestion, provide energy, storage function, and metabolic fuels; operating as functional and structural chemicals in bio membranes; and producing insulation to facilitate nerve conduction or prevent heat loss are just a few of these roles. The results of this investigation revealed differences in lipid profile contents between smoker and non-smoker blood samples, with high values of TG and cholesterol in smoker blood samples (96 ±45.88) and (151.90 ± 39.3), respectively, compared to non-smoker blood samples (93 ± 32.50) and (144.30 ± 23.5). In comparison to non-smoking blood samples (61.90 ±10.5), there was a proportionate drop in HDL values in smoker blood samples (59.1± 22.4). Cigarette smokers had significantly higher serum triglycerides than non-smokers, according to the current study. This was also discovered in a study, which discovered a significant decrease in HDL levels in smokers' blood samples. According to this study, nicotine raises the quantity of bad fats (total cholesterol (TC) and triglycerides (TG) circulating in the blood arteries while lowering the amount of good fat (high-density lipoprotein cholesterol (HDL-C) available. It has been claimed that smoking has a negative impact on plasma lipid concentrations and lipoprotein levels [22] [23].

In addition, total cholesterol, HDL C and TG levels in smokers are significantly higher than in nonsmokers, and the correlation matrix (**Table 8**) revealed a significant positive correlation between cholesterol and nicotine (0.25), as well as positive correlations with TG and HDL with values (0.06 and 0.04), respectively. It was shown that cigarette smokers have a higher risk of cancer. When the results are analyzed in terms of cigarette smoking duration, it is discovered that there is a significant increase in serum cholesterol levels as the duration and intensity of cigarette smoking increases. Our findings are contradicted by the

Table 8. The correlations matrix between nicotine and the studied lipid profile parameters.

	Nicotine	Cholesterol	Triglyceride
Nicotine	1		
Cholesterol	0.25	1	
Triglyceride	0.063	0.47	1
HDL	0.04	0.075	0.12

above findings. A diagram depicting a proposed method by which nicotine absorbed from cigarette smoke raises plasma lipids and cholesterol levels [24].

Cigarette Smoking

↓

Absorption of nicotine into the body

↓

Secretion of catecholamines, cortisol and growth hormones

↓

Activation of adenyl cyclase in adipose tissue

↓

Lipolysis of stored TG and release of FFA into plasma

↓

Release of FFA from adipose tissue TG into plasma bound to albumin

↓

Increased Hepatic synthesis of TG, VLDL-C

↓

Increased Plasma TG, VLDL-C

↓

HDL-Cholesterol

In this study, smokers had significantly higher TC and TG levels than nonsmokers, while smokers had significantly lower blood HDL levels than nonsmokers. Our findings are consistent with those of many other researchers. The difference in serum cholesterol and lipoprotein levels were more pronounced as the number of years spent smoking increased. This conclusion has been supported by [23] [24]. Contrary to the findings of the previous study [24], no significant changes in serum TG, or HDL levels were seen between smokers and nonsmokers. In several studies, no significant differences in triglycerides or total cholesterol were reported between smokers and nonsmokers. Previous studies [25] have suggested that these disparities are linked to ethnic variance in the population.

4. Conclusion

The data revealed that the contents of hematological parameters differed between smokers' and nonsmokers' blood samples, with high counts of WBC,

MCHC, HGB, and PLT plainly visible in smokers' samples. However, there was no discernible effect on RBC, MCV, or MCH counts in the trial. Our research clearly demonstrates a robust link between serum cholesterol and HDL levels and cigarette smoking. It also emphasizes that as the duration and intensity of smoking increase, so do the changes in serum lipids.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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