

## **Analysis of serum lipid profile in adult female smokers in Erbil city, Kurdistan region of Iraq.**

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### **Abstract**

**Background:** There are few studies demonstrated the association between smoking and lipid profile in female adult smokers.

**Materials and Methods:** This study conducted to determine and compare the serum lipid profile of female adult smokers with non-female smokers, known as controls. In 180 female subjects, the level of serum lipid profile measured.

**Results:** Of these, 110 were smokers and 70 non-smokers (control) aged between 25 and 50 years. The study involved only smokers who had smoked for more than 5 years. Our result revealed that mean serum of total cholesterol ( $275.2 \pm 32.6$  mg/dl), triacylglycerol ( $188.4 \pm 56.42$  mg/dl), very low density lipoprotein ( $36.6 \pm 14.2$  mg/dl), low density lipoprotein ( $133.21 \pm 9.81$  mg/dl) were significantly higher in female smokers as compared to non-female smokers with mean of serum total cholesterol ( $172.3 \pm 18.6$  mg/dl), very low density lipoprotein ( $21.8 \pm 9.6$  mg/dl), triglyceride ( $108 \pm 8.84$  mg/dl), low density lipoprotein ( $94.54 \pm 8.5$  mg/dl). However, the mean of serum value for high density lipoprotein cholesterol in chronic female smokers was lower ( $44.6 \pm 4.6$  mg/dl) than in non-female smokers ( $55.3 \pm 8.2$  mg/dl).

**Conclusion:** This study indicated that smoking cigarettes in female cause's dyslipidemia, resulting in increased risk of cardiovascular disease among smokers.

**Keywords:** Cholesterol, LDL, HDL, Smoking.

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### **Introduction**

Smoking, a known myocardial infarction risk factor. It influences various mechanisms, which can accelerate atherogenesis and it has been confirmed that is related to active smoking [1]. Smoking and cardiovascular disease have a strong relationship, where a study confirmed the total cholesterol, Low-Density Lipoprotein (LDL) cholesterol and Triglyceride (TG) tend to be elevated and the HDL level decreased in smokers [2]. In another study, the total cholesterol and TG rates decrease following cessation of smoking [3]. The exact process by which cardiovascular risk increase is unknown. The cause of smoking sickness is most likely due to these harmful and carcinogenic chemicals, because of tobacco contains around 5000 of chemical materials [4]. It has been revealed that the

common smoking-related disease includes cardiovascular disease, chronic obstructive disease and lung cancer [5]. In addition, nicotine is the key ingredient of cigarette smoke, which is an alkaloid derived from nicotine tobacco leaves that activates the body [6]. Cigarette use is associated and effects on the lipid profile thereby raising the risk of Coronary Heart Disease (CHD), atherosclerosis and atherosclerosis [3].

There are different mechanisms, which causes alteration in lipid by smoking for example (nicotine). In general, the nicotine leads to increase secretion of hepatic free fatty acids and Triglycerides (TG) in the bloodstream together with Very-low-Density Lipoprotein-Cholesterol (VLDL-C) by increasing catecholamine secretion. Then it leads to stimulate sympathetic adrenal and causes an increase in lipolysis in the body. A cigarette smoking is also considered

to have an increased levels of homocysteine that induces oxidative Low-Density Lipoproteins Cholesterol (LDL-C) alteration and decreases HDL cholesterol, multiple studies have documented homocysteine inhibiting Apolipoprotein A1 (Apo A-I) which is measure an important protein compositions of high-density lipoproteins-cholesterol (HDL-C) in plasma [7].

Nicotine increases the amount of bad fats in the blood vessels for example Total Cholesterol (TC), LDL, and TG, it may also decreases the amount of good fat for example total lipoprotein cholesterol (HDL-C) [8]. Nicotine also induces oxidative stress, creates free radicals that target the member lipids following by Malondialdehyde (MDA). This MDA can cause peroxidative damage to the body tissues [9]. Oxidation of lipoproteins was believed to occur in the bloodstream and can create superoxide radicals. Hydrogen peroxide or lipid peroxides outside the cell may contribute to LDL oxidation [9]. However, there is much debate about the components of the lipid profile are primarily altered in response to cigarette smoking. It is not exactly clear whether certain components of the lipid profile directly or indirectly affect other components.

In this study, we hypothesized and analysed the effect of smoking in female community on the lipid profile in Erbil city, Kurdistan region of Iraq.

### Materials and Methods

In this study, 180 female smokers were evaluated in Erbil city, Kurdistan region of Iraq. For this study 110 female lifelong smokers who had been smoking for more than 5 years. Female aged between 25 and 50 years, were included as a (Group A). 70 non-smokers in female nearly matching in their weigh and age with the female smokers in (Group A) were subjected as controls (Group B). The control group was considered a healthy and without history of smoking that matches the background of cases.

Blood samples were taken on fasting for 10-12 hrs. Each blood sample was moved with plastic centrifuge tube to Rizgari Teaching Hospital laboratory. For both groups the following criteria were used:

1. Ex-smokers
2. Hepatic dysfunction
3. Endocrine disorders
4. Obesity
5. Renal problem
6. Alcoholics
7. Family history and previous heart disease
8. Hypertension
9. Diabetes mellitus
10. Lipid lowering drug or any drug

Blood serum was collected, centrifuged and then samples

were subsequently aliquoted and stored at -20 °C until analyzed. Levels of lipid profile such as cholesterol, TG, LDL-C and HDL-C were examined by Cobas 400 integra colorimetric method. The range of normal, and high total cholesterol was <200, 200 to 250, >250 mg/dl respectively. TG estimation method was based on Wahlefeld's and the normal value for TG was <150 mg/dl. Examination of LDL-C depends on the cholesterol ester breakdown into free cholesterol and cholesterol esterase fatty acids. The normal range of LDL-C was <150 mg/dl. The normal range of HDL-C was 34-52 mg/dl in females.

### Statistics

Statistical analysis was performed by using of IBM SPSS Statistics software version 20. Data were presented as mean values ± SD. Statistical evaluations were performed by the use of t-test. P value ≤ 0.05 was considered significant and the difference between mean total cholesterol, TG, HDL and LDL levels of the two classes was statistically examined by student t-test.

### Results

As shown in Table 1, the levels of Total Cholesterol (TC), TG, VLDL-Cholesterol, LDL-Cholesterol, were significantly increased in female smokers compared to control group. However, the levels of HDL-Cholesterol in group A were significantly lower compared to group B.

Table 1. Parameters for the lipid profiles.

Lipid profile mg/dl	Smokers group A mean ± SD	Non-smokers group B mean ± SD	P-value
TC	275.2 ± 32.6	172.3 ± 18.6	<0.05
TG	188.4 ± 47.6	108 ± 8.84	<0.05
VLDL-Cholesterol	36.6 ± 14.2	21.8 ± 9.6	<0.05
LDL-Cholesterol	133.21 ± 9.81	94.54 ± 8.5	<0.05
HDL-Cholesterol	44.6 ± 4.6	55.3 ± 8.2	<0.05

### Discussion

The rate of CHD in female smokers was higher compared to non-female smokers in Erbil city. This might due to cigarette will leads to increase the levels of lipoprotein. According to this rate, we evaluated the levels of lipoprotein among female smokers with non-smokers in different age. In general, in cigarette smokers the risk of CHD is higher than in non-smokers and can be interpreted by different interactions among degradation of the integrity of Arterial Walls (AW), blood lipid, lipoprotein derangements and blood coagulation. The result data of lipid profiles such TC, TG, VLDL-C; LDL-C levels in smokers of group compared were higher than to non-smokers in group B with p values as shown in Table 1. It may because of nicotine's, the nicotine leads to secrete high levels of Hepatic Free Fatty Acids (HFFA) and TC along with VLDL-C in the blood stream by increasing

the secretion of catecholamines. This catecholamine's then stimulating sympathetic adrenal system followed by in increased lipolysis. Or the spike in lipids in female smokers may be because of consuming tobacco induces the absorption of nicotine into the bloodstream. This absorption may induces lipolysis. Then free fatty acids will pass through into the bloodstream via activation of adenylyl cyclase in the body tissue and then nicotine will induces catecholamine secretion [10]. It is important to mention that elevated free fatty acids in the liver contribute to decreased hepatic TG and VLDL synthesis and it may raising the blood production of TG and VLDL-C [11]. Our data has also shown a significant reduction in HDLC levels ( $P < 0.05$ ) in female smokers compared to non-female smokers (Table 1). In general, HDL biosynthesis involves in synthesis of ApoA-I (Apoa-1) followed by the acquisition of liver cholesterol and phospholipids, forming nascent HDL particles. Smoking cigarettes can decrease the ApoA-I levels. Lack of ApoA-I results in defective synthesis of HDLs. Smoking cessation regained attention at ApoA-I. This data is in line with many studies that have reported high homocysteine plasma levels in smokers [12,13], and stated that in plasma homocysteine negatively associated with HDL-C and Apo A-I. Increased levels of homocysteine may result in decreased levels of HDL-C via multiple mechanisms as described by Huang F., et al. [12]. Genetic study to evaluate the influence of blood lipid levels on the risk CHD are more recommended.

## Conclusion

Smoking-induced can increased release of catecholamine may also justify further reduction of HDL-C in lifelong smokers, resulting in decreased VLDL-C and reduced concentrations of HDL-C. However, our data clearly showed a very strong relationship between smoking and lipid profile increase. The risk of an increase in serum cholesterol in female smokers with an increase in LDLC is associated with coronary heart disease.

## Conflict of interest

The authors state no conflict of interest.

## Author Contributions

All authors contributed equally in this study.

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