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Original Research Article

# Study of Lipid Profile and Hb Level in Smoker's Males

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## Abstract

**Introduction:** Smoking is a significant contributor to atherosclerosis and coronary artery disease. Nicotine growths the release of hepatic free fatty acids and triglycerides, as well as VLDL-C, in the bloodstream via boosting catecholamine secretion, which stimulates the concerned adrenal organization, resulting in enhanced lipolysis. Over the course of a decade, 337 million Indians have ingested tobacco. Tobacco-associated fatalities in India strength beat 1.5 million per year by 2020, giving to the WHO. Cigarette smoking is a significant and liberated danger issue for atherosclerosis, heart illness, cardiovascular difficulties, and other cardiovascular disorders, and several studies have shown that nicotine is significantly linked to lipid profile alterations.

**Aim:** Study of Lipid Profile and Hb Level in Smoker's Males

**Material and Method:** The study conducted Department of Medicine this study included 50 healthy male subjects and 50 CAD subjects of age 18 -45 years subjects who were referred to Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine

**Result:** This research clearly shows a link among cigarette smoking and a growth in serum lipids. The danger of a increase in serum Cholesterol with an growth in LDL-Cholesterol and a reduction in HDL-Cholesterol in persistent smokers carries a lot of weight because this is the outcome linked to Coronary Heart Disease.

**Conclusion:** Compare to non-smokers and smokers subjects Hb level are raised in smokers subjects compare to non-smokers subjects the valve shows that statistically significant p-valve is $P < 0.0001$

**Keywords:** Heart Disease, Hb, Tobacco, WHO, Cigarette, HDL



## Introduction

Over the course of a decade, 337 million Indians have ingested tobacco. Tobacco-associated fatalities in India strength beat 1.5 million per year by 2020, giving to the WHO. Cigarette smoking is a significant and liberated danger issue for atherosclerosis, heart illness, cardiovascular difficulties, and other cardiovascular disorders, and several studies have shown that nicotine is significantly linked to lipid profile alterations.

Aside from all of this knowledge, there is still a lot of discussion over which aspects of the lipid profile are greatest influenced by smoking, and if some sections of the lipid profile directly or indirectly impact extra areas of the lipid profile, and vice versa. Divergent findings are reached by various investigators [1].

Cigarettes are the leading source of hazardous chemical poisoning and chemical-induced disorders in people. Tobacco is still one of the world's maximum significant concerns, assassination about eight million people each year. Furthermore, tobacco usage is responsible for more than 7 million fatalities, with 1.2 million of them being non-smokers (SMS) [2].

For model, Siekmeier *et al.* [3] found that HDL-C levels are the similar in smokers and non-smokers, but Ito *et al*. [3] found that HDL-C stages are different in smokers and non-smokers. HDL-C levels were shown to be lower among smokers. The hypothalamic-pituitary-thyroid axis and thyroid gland purpose are both affected by smoking. As a result of smoking exposure, thyroid hormone production, binding, distribution, storage, and elimination are all changed, resulting in the formation of hormone concentrations. Smoking is one of the greatest influential and extensive addictive performs, impacting human behavior. Smoking is progressively on the increase in the rising domain, and it is one of the maximum severe risks to current and upcoming worldwide condition. Smoking is responsible for over 20% of all coronary heart disease fatalities. Tobacco habit is an important reason of coronary artery disease, atherosclerosis, and peripheral vascular disease [4]. A advanced atherogenic lipid profile is related to smoking. It raises blood total Cholesterol, triglycerides, LDL-Cholesterol, and VLDL-Cholesterol levels while lowering HDL-Cholesterol levels. As a result, smoking is a significant contributor to atherosclerosis and coronary artery disease. Nicotine growths the release of hepatic free fatty acids and triglycerides, as well as VLDL-C, in the bloodstream via boosting catecholamine secretion, which stimulates the concerned adrenal organization, resulting in enhanced lipolysis. (b) Smokers' ingesting of a diet low in fibre and cereals but great in fat and cholesterol as likened to non-smokers [5,6].

## Aim

Study of Lipid Profile and Hb Level in Smoker's Males

## Material and Method

The study conducted Department of Medicine this study included 50 healthy male subjects and 50 CAD subjects of age 18 -45 years subjects who were referred to Patients went directly to the Observed Treatment Short-course focus in the Dept. of Medicine

## Sample Collection

A 5ml blood sample from each subject was obtained and divided in a simple tube. The Lipid Profile and Testosterone Levels Were Estimated Using the Sample.

## Biochemical Analysis

Lipid profile was estimated on AU480 Analyser and Hb, Beckman coulter analyser.

## Result

**Table 1**

| Parameters | Smokers | Non-smokers | P-valve |
|---|---|---|---|
| Cholesterol | 280.5±36.4 | 174.2±18.9 | P < 0.0001 |
| Triglyceride | 194.6±42.6 | 120.8±17.6 | P < 0.0001 |
| HDL | 31.7±3.21 | 44.7±4.44 | P < 0.0001 |
| LDL | 209.88±13.7 | 105.34±21.3 | P < 0.0001 |
| VLDL | 38.92±9.87 | 24.16±6.11 | P < 0.0001 |
| HB | 17.2±2.60 | 14.20±1.1 | P < 0.0001 |

Compare to non-smokers and smokers subjects Total cholesterol, Triglyceride, LDL, VLDL, are more than non-smokers to smokers subjects is statistically significant but was reverse the case with HDL-Cholesterol. HDL-Cholesterol was suggestively lesser in smokers than in non-smokers. The valve shows that statistically significant p-valve is $P < 0.0001$

Compare to non-smokers and smokers subjects Hb level are raised in smokers subjects compare to non-smokers subjects the valve shows that statistically significant p-valve are $P < 0.0001$

## Discussion

Smoking increases catecholamine release, resulting in a rise in VLDL-C and a decrease in HDL-C, which may explain why HDL-C levels in chronic smokers continue to decline. As a result, smoking contributes to coronary artery disease and atherosclerosis by lowering the anti-atherogenic component HDL-C while increasing the atherogenic lipoproteins LDL-C, weakening the vascular endothelium. Smokers had significantly higher total cholesterol, triglycerides, LDL-C, VLDL-C, and a much lower level of HDL-C than non-smokers, according to recent research. These findings were in line with those published by Devaranavadgi BB *et al*. [7].

In humans and animals, too much carbon monoxide (CO) can induce polycythemia. In the body, CO has a half-life of 3-5 hours. When someone smokes on a regular basis, carboxy Hb levels rise, creating gradual hypoxia, and active anaemia rises as CO binds to Hb. Tissue volume decreases, and haematological parameters change as a result [8].

Similar findings were seen in a study of lipid and lipoprotein profiles in middle-aged male smokers in southern India, where total cholesterol, triglycerides, and LDL-C levels were dramatically enhanced while HDL-C levels were significantly lowered. In another study, the same link was found between smoking and HDL-C levels; however, levels of Triglyceride and LDL-C were not substantially higher in smokers, although there was a significant rise in levels of Triglyceride and LDL-C in a meta-analysis. In our study, however, the levels of total cholesterol, triglycerides, and LDL-C all increased significantly. The following process might explain why smokers' lipid levels are higher: Nicotine is taken into the body through cigarette smoking, which causes lipolysis and the release of free fatty acids into the circulation due to nicotine-stimulated catecholamine synthesis stimulating adenylcyclase in adipose tissue. Increased hepatic Triglyceride and VLDL synthesis is caused by an increase in free fatty acids in the liver, which raises blood levels of Triglycerides and VLDL-C [9,10].

According to Dunga *et al*. [11], hypertensive smokers have an increased chance of developing severe hypertension because smoking activates the sympathetic nervous system. Sympathetic nerve activation is not caused by CO. In a 7-day single-blind experiment, the effect of CO on sympathetic activation was investigated using three intervention groups: CO, cigarette smoke, and air. There was no change in blood pressure across the groups, according to this study. When compared to air, cigarette smokers had higher urine epinephrine and norepinephrine levels with no effect from CO. According to the findings of this investigation, there was no significant link between blood pressure and CO levels. However, whereas BP is linked to SD and increasing smoke contact (BI), there is no link between BP and the number of CD.

Although blood lipids play a crucial role in the human body, contributing to a variety of biochemical processes and serving as a important section, unbalanced lipid portions pose a health risk. This study also found that

continuing to smoke is strongly linked to dyslipidemia. A number of factors can contribute to smoking-related dyslipidemia. In line with prior studies, our data demonstrated that smokers had significantly greater TC levels than non-smokers. Smokers had higher TG, LDL, and VLDL levels than non-smokers (p0.001), and our findings are consistent with those of other research.

Furthermore, smokers had lower HDL levels (p=0.016) than anthropometrically matched non-smokers, which is consistent with previous research. Furthermore, smokers had a considerably higher TC:HDL ratio than nonsmokers (p0.001). Despite rising TG and reducing HDL, a large Japanese cohort revealed no change in smokers' TC and LDL-C. This might be due to a number of competing factors such as lipid metabolism, dietary lipid consumption, culture, or other aspects such as exercise [12,13].

The results of this study showed that smokers' HDL-C levels are significantly lower than non-smokers' (p0.001), which is in line with earlier studies. CVDs are connected to an increase in LDL-C and VLDL-C, as well as a reduction in HDL-C. One of the reasons of atheroma and coronary artery disease is an imbalance between good and bad cholesterol. Serum lipid levels in smokers might rise for a variety of reasons. During smoking, large quantities of nicotine are taken into the circulation via the lungs.

Nicotine causes the body to release catecholamines, which are created by the activation of adenyl cyclase in adipose tissue, an increase in lipolysis, and the release of free fatty acids into the circulation.

Increased amounts of free fatty acids in the liver cause more TG and VLDL-C to be produced, resulting in higher TG and VLDL-C levels in the blood. 3 Increased LDL-C and VLDL-C levels in the blood produce a decline in HDL-C levels. In addition to nicotine-mediated catecholamine discharge, researchers have uncovered another mechanism that contributes to smokers' lower HDL-C levels. Smokers have higher homocysteine levels than nonsmokers. Increased homocysteine is known to lower HDL-C levels in smokers and has a detrimental influence on HDL-C levels [14,15].

## Conclusion

This research clearly shows a link among cigarette smoking and an growth in serum lipids. The danger of a increase in serum Cholesterol with an growth in LDL-Cholesterol and a reduction in HDL-Cholesterol in persistent smokers carries a lot of weight because this is the outcome linked to Coronary Heart Disease.

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