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# Effects of Smoking on Serum Lecithin: Cholesterol Acyltransferase Activity

***Corresponding Author:***
Dr. K K Kshitiz,
Assistant Professor, Biochemistry LHMC, G Block Patel Nagar, 11OO67 - India

***Submitting Author:***
Dr. K K Kshitiz,
Assistant Professor, Biochemistry LHMC, G Block Patel Nagar , 11OO67 - India

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# Effects of Smoking on Serum Lecithin: Cholesterol Acyltransferase Activity

***Author(s):*** Kshitiz K

## Abstract

Smoking is a established cardiovascular disease risk factor. But the mechanism how it causes cardiovascular risk is still not properly understood. This study was aimed to evaluate effects of smoking on lecithin:cholesterol acyltransferase (LCAT) activity ,a key factor in the esterification of plasma cholesterol and reverse cholesterol transport. 30 male cigarette smokers and 30 male non-smokers were enrolled. Serum LCAT activity was determined as a function of the decrease of free cholesterol which is esterified during incubation at 37, as described by Hitz et al (15). Serum LCAT activity was lower in smokers, but the difference was statistically non significant. So we got the idea that LCAT activity tended to be lower in smokers compared to nonsmokers and this adverse effect may lead to serious metabolic diseases like atherosclerosis.

## Introduction

Cigarette smoking is the most common type of tobacco use. By 2030, if current trends continue, smoking will kill more than 9 million people annually(1). Smoking is considered as a major cardiovascular risk factor (2) .There is a dose response relationship between the number of cigarettes smoked and cardiovascular morbidity and mortality (3). The mechanism by which cigarette smoking causes atherosclerosis remains obscure, but cigarette smoking have been found to alter the level of lipoproteins (4-8). Plasma lipoprotein abnormalities are said to be the underlying major risk factors and may even be essential for the common occurrence of atherosclerotic vascular diseases

The role of LCAT in atherosclerosis is not clearly established, studies have yielded conflicting results. Sethi et al. demonstrated that low lecithin-cholesterol acyltransferase (LCAT) activities and high pre-ß1-HDL concentrations are strong positive risk markers for ischemic heart disease and independent of HDLcholesterol.(9) .Holleboom et al. showed that low plasma LCAT levels (reflecting low LCAT activity) are not associated with an increased risk of future CAD in the general population(10). Other studies showed a positive association of LCAT levels with carotid atherosclerosis in patients with the metabolic syndrome as well as in control subjects, LCAT activity was reduced in patients with CAD and in patients with acute myocardial infarction. It can be reasoned that LCAT activity might be reduced in the acute phase of a myocardial infarction, but may normalize over time. we investigated the effects of smoking on the levels of LCAT activity in fasting blood from healthy smokers and nonsmokers, matched for age and body mass index (BMI).

## Materials And Methods

The study was conducted on 30 healthy male cigarette smokers in age group of 25-35years and it was compared with 30 healthy age, diet and Body mass index(BMI) matched nonsmokers . Informed consent was obtained from all subjects. Only those persons were included in the study, who had no other existing diseases. Persons taking vitamin C or any other antioxidants were also not included in the study. Persons smoking 5 cigarettes or more per day continuously for a year were considered as smokers. A detailed physical examination of the subjects of both groups was done. After 12 hrs overnight fasting, venous blood samples were drawn and subjected to laboratory investigations including blood glucose, blood urea ,serum alkaline phosphatase, haemogram, lipid profile and LCAT activity.

The data between control and test groups was compared using unpaired student's t test. Correlation was determined by Pearson's correlation coefficient. A two tailed p value less than 0.05 was considered to be statistically significant.

## Results

**Table 1**

Showing clinical characteristics (Mean ± SEM) in non-smokers and smokers.

See Illustration 1

**Table 2**

LCAT activity between smokers and non- smokers.

See Illustration 2

**Table 3**.

Serum LCAT activity, based on exposure to cigarette smoke.

See Illustration 3

The study groups comprised of 30 male smokers (mean age ± SD, 31.20± 1.33) and 30 male non-smokers (31.23± 1.35 ). The body mass indexes were 20.96± 0.60 and 20.99± 0.60 kg/m2 in smokers and non-smokers, respectively (Table 1).. Serum LCAT activity was lower in smokers than in non-smokers, although this difference was statistically non significant (Table 2).

# Discussion

In the study, the two groups of subjects (smokers and non-smokers) were of comparable sex, age, BMI and diet. They were non-diabetic, non-alcoholic, normotensive subjects, mostly (70%) belonging to middle socioeconomic status. There are several studies showing lower serum LCAT concentrations or activities in smokers compared to non-smokers (11). Haffner et al. reported a negative correlation between smoking and LCAT mass (12). The positive correlation between LCAT concentration and TC and LDL-C was also reported in these studies (11-13). These findings are in accordance with the suggestion that LCAT plays an important role in the reverse transport of cholesterol. In addition to in vivo studies, in vitro studies have shown that cigarette smoke inhibited LCAT activity (14). In another study, it was reported that rats given nicotine had lower LCAT activity (15). However, in several studies, LCAT activity was found not to be different in smokers and non-smokers ( 14, 16). In our study, serum LCAT activity was found to be 19.13% lower in smokers than in non-smokers, but this difference was not statistically significant. LCAT activity tended to be lower in smokers compared to non-smokers. So, it may be considered that the reverse cholesterol transport can be diminished and cholesterol can accumulate in the peripheral tissues, more in the smokers than non-smokers.

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# Illustrations

## Illustration 1

Showing clinical characteristics (Mean Ã‚Â± SEM) in non-smokers and smokers.

| Parameter | Non –smokers (n= 20) | Smokers (n= 60) |
|---|---|---|
| Age | 31.20± 1.33 | 31.23± 1.35 |
| Body weight (kg) | 58.90± 1.91 | 59.20 ± 1.91 |
| Height (cm) | 167.40± 1.1 | 167.90± 0.90<br>B |
| BMI (kg/m2) | 20.96± 0.60 | 20.99± 0.60 |

All parameters were not significantly different between groups.

# Illustration 2

LCAT activity between smokers and non-smokers.

| Parameter | Non smoker | Smoker |
|---|---|---|
| LCAT(µmol.L-1.h-1) | 80.30±54.90 | 65.00±48.80 |

LCAT activity was not significantly different between groups.

## Illustration 3

Serum LCAT activity, based on exposure to cigarette smoke.

| | Brinkmann index<300 | Brinkmann index > 300 | P |
|---|---|---|---|
| | N=30 | N=30 | |
| LCAT activity (µmol.L-1.h-1) | 78.4±48.00 | 57.2±43.67 | >0.05 |

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