Ann. For. Res. 65(1): 10562-10567, 2022
ISSN: 18448135, 20652445

ANNALS OF FOREST RESEARCH
https://www.e-afr.org/

IMPACT OF SMOKING ON HUMAN LIPOPROTEIN

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ABSTRACT

Introduction: The most frequent form of fat in the body is triglycerides. They store any extra energy you eat and high-density lipoprotein Cholesterols (HDL-C) is known as the "good" cholesterol so it can prevent you from cardiovascular problems if you have a healthy amount. Cigarette smoking damages all organ in the body causes a variety of ailments and lowers smokers' overall health, as well as Cigarette smoking, can alter triglyceride and high-density lipoprotein Cholesterols in the blood, causing smokers to have increased triglyceride levels and decreased high-density lipoprotein Cholesterols versus people who smoke.

Aim: The purpose of the study is to determine how cigarette smoking affects triglyceride and HDL-C levels when compared to non-smokers.

Materials and Methods: An outpatient clinic in Baghdad, Iraq, was the subject of a cross-control study. This outpatient clinic serves as the city's primary general center, with a sizable population. The research took place from March 2021 to September 2021. Adult smokers and non-smokers of both males and females were enrolled in this study, and serum samples were pooled from blood collections from each subject. Following that, each serum product was analyzed for triglycerides and HDL-C concentration.

Results: The results demonstrated that there was a significant difference in triglyceride levels and HDL-C between non-smokers and smokers. Daily cigarette smoking increased triglyceride levels in exclusive cigarette users (303.533±18.059; $p < 0.05$) compared to non-smokers (138.080±7.401; $p < 0.05$) and HDL-C was lower smokers (33.400±2.011; $p < 0.05$) compared to non-smokers' values (66.206±1.876; $p < 0.05$).

Conclusion: Current smokers' triglyceride levels are stimulating for cigarettes as compared to non-smokers, which is consistent with studies in other populations. Furthermore, current cigarette smokers have lower levels of good total cholesterol than non-smokers, which has been associated

with harmful cardiovascular outcomes. The direct cause of the observed differences in our study is still unknown, but future research will perhaps shed some light on it.
Keywords: blood human, triglyceride, HDL-C, smoking.

INTRODUCTION:

Cigarette smoking has been linked to heart disease, atherosclerosis, and impaired glucose tolerance. Other studies have investigated the links between smoking tobacco and serum triglyceride levels, postprandial blood glucose, and levels of HDL cholesterol [1-4]. Smoking has a physiological impact on pathogenic, hematologic, and metabolic variables [5]. In the long run, smoking causes more health problems, such as an elevated risk of stroke and neurological damage [6]. Abnormal lipid levels are firmly linked to a higher risk of atherosclerosis. Triglycerides, another type of lipid particle, are ubiquitous in the human body and have been detected in the arterial wall. Recent observational studies have found a link between high triglyceride levels and an increased risk of heart cases in the future [7]. Following the advent of statins, clinical attention was initially focused on lowering LDL cholesterol, then on the possibility to raise HDL cholesterol, with less attention paid to lowering triglycerides. The discovery, based on genetic studies and negative outcomes from the systematic review, that low HDL cholesterol may not produce coronary heart disease as previously thought has reignited interest in elevated triglyceride levels [8]. Triglycerides are an element of lipid profiles that are frequently examined. The metabolic syndrome includes high triglyceride levels [9]. HDL functioning is far more significant than systemic HDL-C levels in atheroprotection [10]. Smoking tobacco affects lipid transport enzymes, reducing lecithin: cholesterol acyltransferase action and modifying cholesterol ester transfer protein and hepatic lipase function, which contributes to HDL breakdown and subfraction diffusion [11]. In the opposite transport and removal of cholesterol, HDL-particles perform a critical function [12].

MATERIALS AND METHODS:

Participants in this study comprised adult smokers and non-smokers of all genders, as well as subjects with triglyceride and HDL-C. The first group consisted of non-smokers who served as healthy controls, while the second group consisted of smokers who smoked 15 to 25 cigarettes per day. People who smoke and non-smokers who worked at the Outpatient Setting as patients, assistants, donors, or staff. People in the study were picked from among the employees. Pressure, hyperglycemia, and kidney or liver illness are all disorders that might cause death in some people. A sampling technique using a purposive approach was used to select members from various age categories. In total, 50 people were contacted in a succession as being eligible to participate in the study. Interviews were conducted with those who were available in the outpatient clinic. After describing the project's goals to all participants, they signed the document informed consent form. The individuals' demographic information was obtained via a personality assessment. Their smoking habits, and also their medical histories, were gathered to see if they had any chronic conditions or were using any lipid-lowering medications. Additionally, all patients were obliged

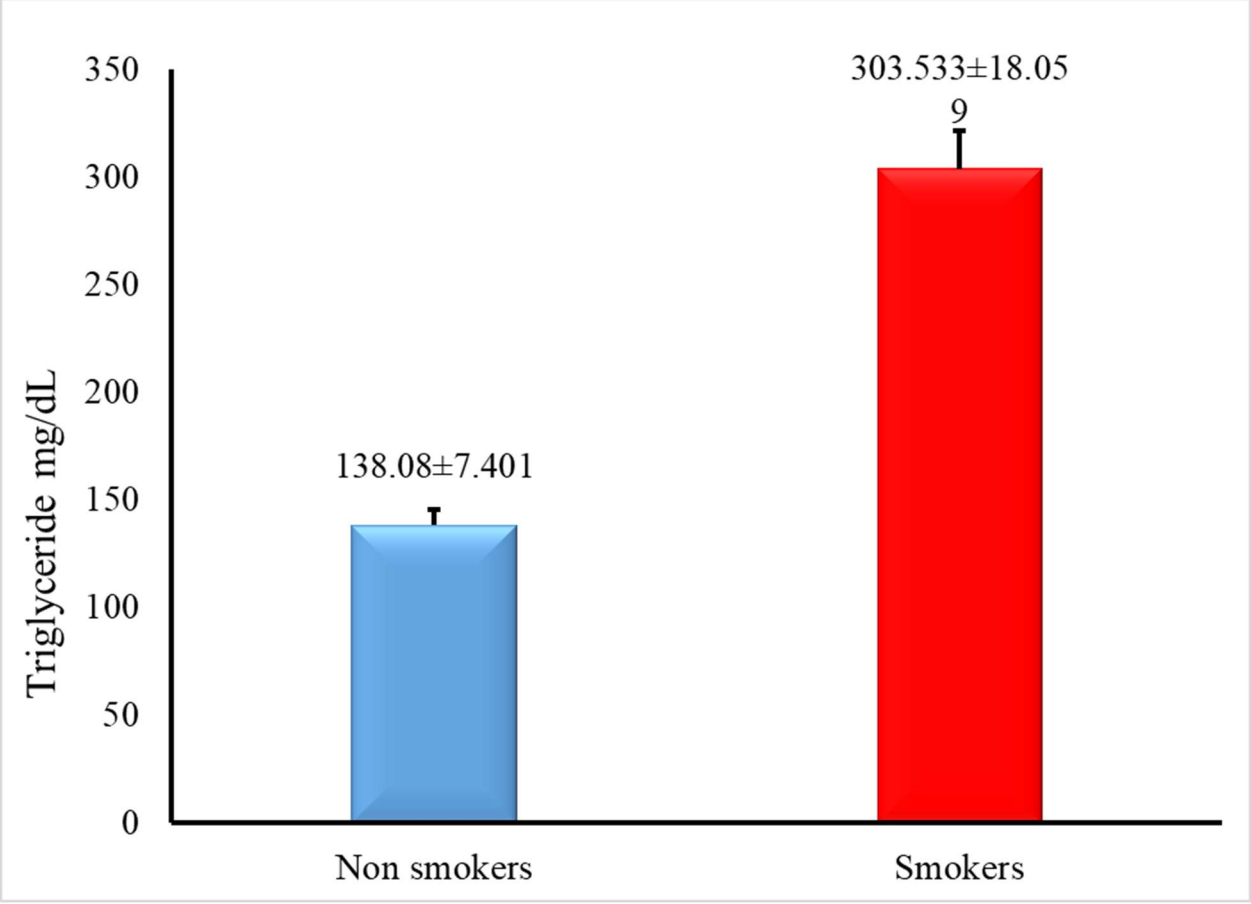
to perform their blood tests while fasting for a minimum of 14 hours before collecting samples, so each test was subject to the company's method. Blood was taken into basic tubes after a 14-hour fast and centrifuged at 3000 rpm (China) for 15 minutes to extract serum. All specimens were tested for triglyceride and HDL using an Automatic Chemistry Analyzer and the standard homogeneous enzymatic approach (Linear), with the calibration and internal controls supplied by the Linear Company with Spectrophotometer (EMCLAB, Germany).

STATISTICAL ANALYSIS:

For statistical analysis, Excel and Statistics (SPSS version 18) were utilized. Continuous data were expressed using the mean and standard deviation. Paired The Student's t-test was used to compare the differences in Cholesterol concentration measured by the direct method and other formulae between smokers and non-smokers. The significance level was set at $P < 0.05$ [16] .

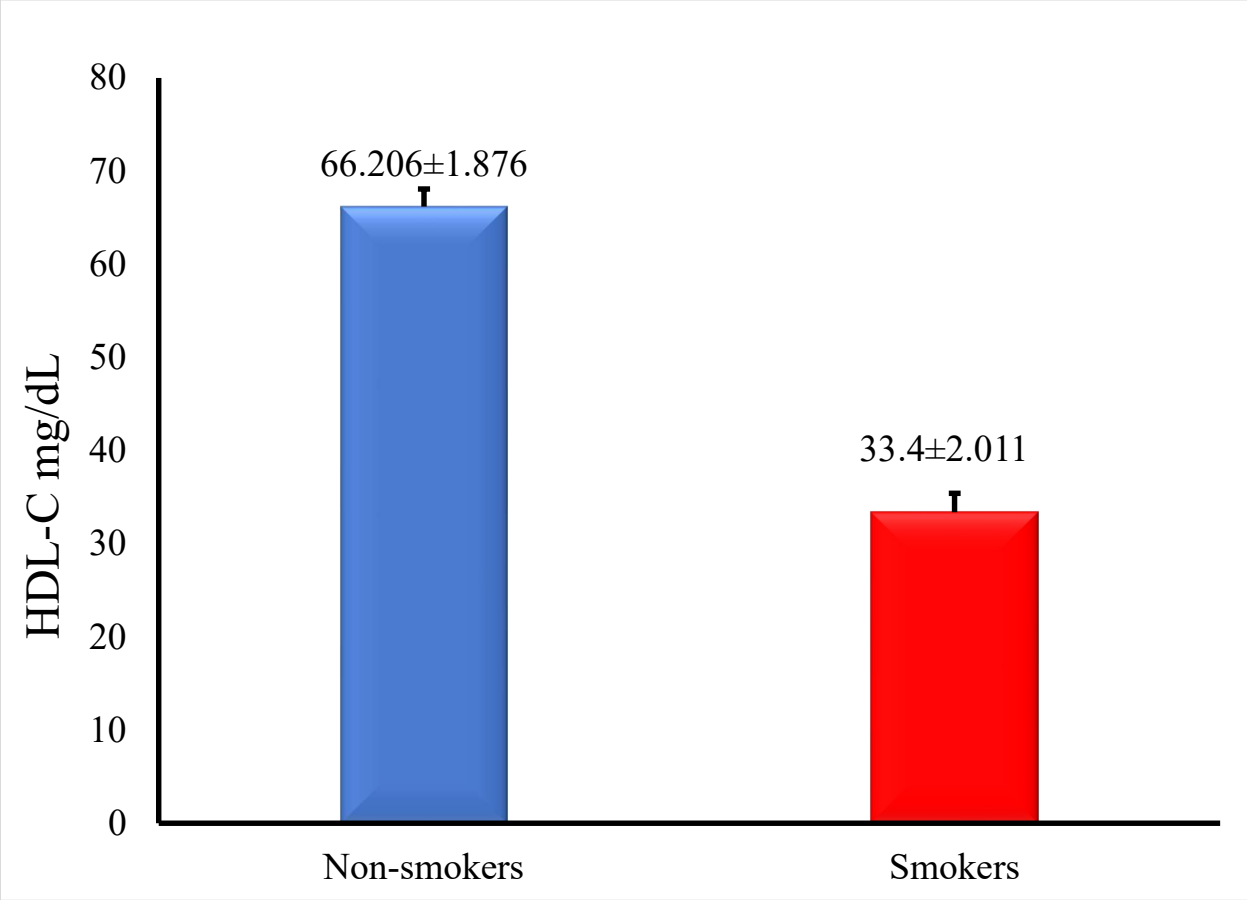
RESULTS:

The study's findings demonstrated that there was a significant difference between non - smokers and smokers in mean the triglyceride values and showed triglyceride levels compared to smokers a greatly upregulated (303.533±18.059; $p < 0.05$) compared to non-smokers (138.080±7.401; $p < 0.05$) [Table/Fig-1].



[Table/Fig-1]: Triglyceride levels in studied individuals

Furthermore, HDL-C was downregulated smokers (33.400±2.011; $p < 0.05$) compared to non-smokers' values (66.206±1.876; $p < 0.05$), [Table/Fig-2].



[Table/Fig-2]: HDL-C levels in studied individuals

DISCUSSION:

This is the strongest chronic, completely randomized design that we are aware of that has looked at the impact of quitting smoking on cholesterol and lipoprotein ratios. Smoking has been proven to change the levels of lipids and lipoproteins [13]. Cigarette smokers have an atherogenic lipoprotein profile with reduce HDL-C and modestly elevated triglycerides, according to this large cohort study [14]. In our study, it was discovered that cigarette smoking was linked to higher triglyceride levels, which coincided with the discovery of Kuzuya et al. [15]. Also our study explained smokers had lower amounts of high-density lipoprotein cholesterol (HDL-C) according to the study of Gossett et al. [14]. In our investigation, we found dose-dependent interactions with lipoproteins as well; although, the impacts of smoking activity were less for HDL-C.

CONCLUSION(S):

Elevated cigarette consumption is linked to minor increases in triglycerides. Recent smoke exposure is linked to a slight drop in HDL-C levels. Smoking is unlikely to be an effective therapy for treating dyslipoproteinemia in smokers due to the small dose-effect of smoking frequency on lipoproteins. Recent regulations propose complete cigarette smoking as a more effective method for enhancing lipoproteins and lowering Cardiovascular risk in smokers.

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