Vol 11 No 2 Year 2026

BULETIN FARMATERA

E-ISSN: 2528-410X

RESEARCH ARTICLES

Comparison of High-Density Blood Lipoprotein Levels in Active Smokers, Passive Smokers, and Non-Smokers

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Abstract: Tobacco is one of the factors known to increase the risk of disease and premature death globally. Nicotine is one of the harmful substances in cigarettes that can result in the release of hormones such as catecholamines, cortisol, and growth hormone. The release of these hormones can alter the lipid profile in the blood, including increased total cholesterol levels, *Very Low-Density Lipoprotein* (VLDL), *Low-Density Lipoprotein* (LDL), and decreased High-*Density Lipoprotein* (HDL) levels. This study aims to determine the comparison of HDL levels in active smokers, passive smokers, and non-smokers. The total sample in this study was 45 people who were divided equally into three different groups, namely active smokers, passive smokers, and non-smokers. Data analysis was processed using SPSS. Research shows there is a difference in HDL levels between active smokers, passive smokers, and nonsmokers ($p<0.05$). Smoking can significantly lower HDL levels in the blood.

Keywords: *High density lipoprotein*, cigarettes, active smokers, passive smokers, non-smokers.

INTRODUCTION

Tobacco is one of the factors known to increase the risk of disease and premature death globally. However, the relative risk of death and consequences from smoking varies in different countries. Indonesia, as one of the largest tobacco cigarette markets in the world, experiences a significant impact of tobacco-induced diseases.[1] Every year, more than 8 million lives end due to smoking, with 1.3 million of them being people who do not smoke but are passively exposed to secondhand smoke [2]

Nicotine is one of the harmful substances in cigarettes that can result in the release of hormones such as catecholamines,

E-ISSN: 2528-410X

cortisol, and growth hormone. The release of these hormones can alter the lipid profile in the blood, including increased total cholesterol levels, *Very Low Density Lipoprotein* (VLDL), *Low Density Lipoprotein* (LDL), and decreased High *Density Lipoprotein* (HDL) levels. In addition, the Carbon Monoxide (CO) released by cigarette smoke can cause narrowing of blood vessels, which in turn can increase blood pressure and damage the walls of blood vessels. Carbon monoxide can also affect haemoglobin saturation, reduce the oxygen supply to the body, and accelerate the occurrence of atherosclerosis.[3]

HDL (High Density Lipoprotein) is the type of lipoprotein with the highest protein content compared to other types of lipoproteins, so it has a high density. These lipoproteins are produced through synthesis in the liver. HDL is often referred to as a beneficial fat because of its role in clearing excess cholesterol from the blood vessels and returning it to the liver to be excreted through the gallbladder as liquid bile acids. Lack of HDL in the body can contribute to various diseases, one of which is coronary heart disease.[4]

According to a research study by Dina Fahmawati (2019) involving 13 smokers and 13 non-smokers, it was shown that the average nutritional intake and cholesterol levels in the blood of smokers and non-smokers had insignificant differences.[5] Meanwhile, according to Qasim, Falih and Al Husaini's (2020) research, significant differences in HDL levels were found between individuals who smoked and individuals who did not smoke. In individual smokers, the average HDL level was 28.9 mg/dL, while in non-smokers, the average HDL level was 50.6 mg/dL.[6] Both of these studies used cigarette butts. Therefore, this study aims to compare blood HDL cholesterol levels between three different groups, namely active smokers, passive smokers, and nonsmokers. It is hoped that the results of this study can provide further understanding of the relationship between smoking habits and blood HDL levels, as well as their contribution to cardiovascular risk factors.

METHOD

This study is an experimental quantitative study with *a cross-sectional* approach; the research is completed at a specific time, and no research is conducted at various times to compare.

Data collection of HDL cholesterol levels was carried out by a quantitative method, namely CHOD-PAP, using a spectrophotometer.

Data analysis was processed using SPSS. First, the data will be tested for normality. The normality test used is the *Shapiro-Wilk* test. After the normality and homogeneity test, the *Mann-Whitney test* is continued if the data is abnormal/homogeneous, but if the data is normal/homogeneous, then use *the one-way ANOVA* test to compare.

E-ISSN: 2528-410X

RESULT

Table 1. Sample Demographic Overview

Characteristics	Frequency (n)	Percentage (%)
Age		
18	0	0
19	0	0
20	8	17,7
21	19	42,2
22	7	15,5
23	5	11,1
24	3	6,6
25	3	6,6
Total	**45**	**100**

Based on Table 1, the demographic picture of the sample with a total sample of 45 males. Based on age, it was found that none of the samples were 18-19 years old. A total of 8 people (17.7%) were 20 years old. A total of 19 people (42.2%) were 21 years old. A total of 7 people (15.5%) were aged 22 years. A total of 5 people (11.1%) were 23 years old. A total of 3 people (6.6%) were 24 years old. A total of 3 people (6.6%) were 25 years old.

Table 2. Distribution of sample characteristics based on smoking habits

Types of Smokers	Frequency (n)	Percentage (%)
Active		
Light	15	33,3
Moderate	0	0
Heavy	0	0
Passive		
Light	11	24,4
Moderate	3	6,6
Heavy	1	2,2
Non-Smoking	15	33,3
Total	45	100

Based on Table 2, the distribution of sample characteristics was based on smoking habits, with a total of 45 people. Active smokers are divided into 3 levels, namely light, moderate, and severe. The number of samples of mild active smokers was 12 people (26.6%), moderate active smokers were 2 people (4.4%), and heavy active smokers were 1 person (2.2%). Passive smokers are divided into 3 levels, namely light, moderate, and heavy. The number of samples of light passive smokers was 11 people (24.4%), moderate passive smokers were 3 people (6.6%), and heavy passive smokers were 1 person (2.2%). The number of non-smoking samples was 15 people (33.3%).

Table 3. Distribution of Active Smokers by Brinkman Index

Smoking Habits	Frequency (n)	Percentage (%)
Number of cigarettes smoked per day		
< 10	0	0
10 – 20	15	100
> 20	0	0
Total	**15**	**100**
Long Smoking		
< 5 years	11	73,3
5 – 10 years	4	26,6
> 10 years	0	0
Total	**15**	**100**
Categories by Brinkman Index		
Light active smoker	15	100
Moderate active smokers	0	0
Heavy active smokers	0	0
Total	**15**	**100**

BULETIN FARMATERA
E-ISSN: 2528-410X

Based on Table 3, to clarify the classification of active smokers with the Brinkman index. It was found that 15 people (100%) smoked 10-20 cigarettes per day. A total of 11 people (73.3%) had a long history of smoking for < 5 years, and as many as 4 people (26.6%) had a long history of smoking for 5-10 years. Of the total active smokers in this study, they are light active smokers.

Table 4. Sample Distribution Based on High-Density Lipoprotein Levels

Group	Mean(±SD)
Active	36,8(± 2,3)
Passive	49,2(± 2,2)
Non-Smoking	57,3(± 3,2)
Total	**47,7(± 8,9)**

Based on Table 4, it is shown that active smokers have a mean of 36.8 mg/dL with a standard deviation of 2.3. In passive smokers, the mean was 49.2 mg/dL with a standard deviation of 2.2. Non-smokers had a mean of 57.3 mg/dL with a standard deviation of 3.2.

Table 5. One-Way ANOVA Test

Kelompok	Mean(±SD)	*p*
Active	36,8(± 2,3)	
Passive	49,2(± 2,2)	<0,001
Non-Smoking	57,3(± 3,2)	
Total	**47,7(± 8,9)**	

If a data has a Sig. < 0.05, then it can be concluded that there is a significant difference. Based on the table above, the data has a Sig. < 0.001, so it can be concluded that there is a significant difference between the variables.

DISCUSSION

This study involved as many as 45 respondents with gender, smoking habits, the number of cigarettes consumed per day, and a varied history of exposure to cigarettes. Men dominate in sex characteristics. Research conducted by Stenly and colleagues in 2019 produced similar findings, where of the 30 patients who experienced Coronary Heart Disease (CHD) with low HDL levels, there were 10 men (33.4%) and 8 women (26.7%). HDL levels tend to decrease in men over the age of 50 due to the influence of atherosclerosis caused by cholesterol. However, comparisons with women who have gone through menopause show that there is no significant decrease in HDL. This is due to a decrease in estrogen hormone levels in women after menopause, which causes the body to lose its ability to protect itself from atherosclerosis and cholesterol.[7]

In smoking habits, 15 people from active smokers are light active smokers, 15 people from passive smokers are mostly light passive smokers, and 15 people are non-smokers. According to Qasim, Falih, and Husaini (2020) shows that HDL levels are lower in active smokers than in non-smokers. Smoking can lead to a decrease in apoA-1 levels, resulting in a disruption in HDL formation. Not only that, the habit of smoking also has the potential to affect the restructuring of HDL in blood vessels. In

E-ISSN: 2528-410X

smokers, there is an increase in CETP activity, which significantly increases triglyceride levels while lowering HDL levels. In individuals who smoke, HDL can oxidise, inhibit the process of cholesterol transport and in turn increase LDL oxidation, which can lead to inflammation in the vascular system [6]

From the number of cigarettes consumed per day, it is known that 15 people (100%) of active smokers consume cigarettes if they consume 10-20 cigarettes per day. Research conducted by Kubihal and Naik in 2019 showed that a person's smoking levels significantly correlated with decreased HDL levels. Individuals who smoked in the heavy category (more than 24 cigarettes per day) had an average HDL level of 34.4 mg/dL, while those who smoked in the light category (1-10 cigarettes per day) had HDL levels of 47.2 mg/dL. Nicotine entering the body can result in deregulation of the heart's autonomic function, an increased heart rate, and activate the sympathetic nervous system, thereby causing vasoconstriction of the coronary and peripheral blood vessels, as well as an increased workload on the myocardium.[8]

Jika dilihat dari riwayat paparan asap rokok, didapatkan bahwa 15 orang (100%) perokok pasif terpapar asap rokok selama 1 jam per harinya. Abror dan Putri pada tahun 2021 mengindikasikan bahwa paparan asap rokok dapat memicu perkembangan aterosklerosis melalui akumulasi ester kolesterol dalam plak ateroma dan penyerapan kolesterol LDL serta makrofag.[9]

The results of the analytical test from this study obtained a p value of $p < 0.001$ ($p < 0.05$), so that it can be concluded that the Ha hypothesis is acceptable, where there is a significant difference in HDL levels between active smokers, passive smokers, and non-smokers. Based on a review of the literature and the researcher's knowledge, it is known that this study is the first study to directly explore the difference in HDL levels among three groups at once, namely active smokers, passive smokers, and non-smokers.

The relationship between cigarettes and cholesterol levels in the blood is closely related to the chemical content in cigarettes. The nicotine-like content in cigarettes is known to stimulate the release of catecholamines, cortisol, and growth hormones, which ultimately lead to changes in serum lipid profiles, including increased levels of TC, VLDL, and LDL, as well as decreased HDL levels. This is compared to the study of Najiyah et al. (2020), which explained that out of 15 participants, as many as 11 people showed low HDL levels, with an average of around 49.3 mg/dL.[10]

Another study conducted by Qasim, Falih and Al Husaini (2020) revealed that there was a significant decrease in HDL levels between individuals who smoked and individuals who did not smoke. The average HDL level in individual smokers was 28.9 mg/dL, while in non-smokers, the average HDL level reached 50.6 mg/dL.[6]

A decrease in HDL levels in the blood circulation can result in blood clots that are more likely to occur, increasing the risk of

E-ISSN: 2528-410X

arterial blockage. This can increase resistance to the flow of air into the lungs, resulting in an increase in haemoglobin levels in the body to meet oxygen needs. However, this increase in haemoglobin levels can cause thickening of blood in the body, inhibiting blood flow and disrupting the supply of nutrients and oxygen to various parts of the body.[11]

In the process of HDL metabolism, it is known that smoking habits can affect HDL biosynthesis and maturation. Smoking has the potential to decrease the production of apoA-1, hindering the formation of HDL particles. In addition, in the transformation of free cholesterol into ester cholesterol, cigarette smoke can cause damage during HDL ripening, which can lead to rapid cleansing of the circulation for newly formed HDL. Smoking can also cause HDL dysfunction, including atheroprotective disorders due to oxidative modifications. Oxidised HDL can inhibit the process of reverse cholesterol transport, increase HDL levels, and increase inflammation in blood vessels. In addition, smoking can reduce the activity and concentration of antioxidant enzymes in HDL, including paraoxonase, which plays a role in protecting lipoproteins from oxidative modification [11,12]

CONCLUSION

Based on the results of the research and analysis of the One-Way ANOVA test, it can be concluded that there is a significant difference between HDL cholesterol levels between active smokers, passive smokers, and non-smokers.

ACKNOWLEDGMENTS

For the next researcher, it can add variables of factors that affect HDL and more samples. It is hoped that the research can be further developed by using more accurate and modern measuring instruments.

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