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The Difference of Lipid Profile among Adolescent Smokers and Non-Smokers at Urban Area in Developing Country

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Abstract **Objective** Analyze the differences in TC, LDL, and HDL levels between smokers and non-smokers adolescent. **Study Design** This analytical cross-sectional study was performed during February to April 2016 in several senior high schools in Bandung. Subjects were adolescent, aged 15–18 years, who were divided into two groups: smokers and non-smokers using questionnaire. There were 206 boys included in this study, 162 met the inclusion criteria and 44 were excluded. Simple random sampling was performed to obtain 50 smokers and 50 non-smokers for our study. All data were analyzed for mean serum lipid profiles using chi square (x^2) and MANOVA test with a p value <0.05 considered significant. **Results** The x^2 analysis showed association between abnormal serum TC, LDL, and HDL levels with smoking status respectively (p=0.006, p=0.025, and p=0.006). MANOVA test results showed significant differences between smokers and non-smokers group in terms of mean±SD TC level (161.7±32.2 and 150.6±19.5), LDL (107.6±29.2 and 92.6±92.6), HDL (38,76±6.39 and 42.8±7.08)) with p value <0,05.**Conclusion** This study shows that serum TC, LDL, and HDL levels in smokers are statistically different compared to those in non- smokers adolescent.

Keywords: *smoking, adolescent, lipid profile*

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1. Introduction

Smoking is one of the leading causes of death in the world that can be prevented. Most smokers begin smoking in adolescence and continue into adulthood [1]. The recent WHO report shows that smoking in the Indonesian teenagers has increased from 12.6% in 2006 to 23.5% in 2010 [2]. *Pusat Data dan Informasi* (Pusdatin) Indonesian Ministry of Health estimates the number of smoking >10 years citizen everyday in 2013 is 48.400.332 lives [3]. Global Youth Tobacco Survey 2014 stated that Indonesia is a country with the highest number of teenage smokers in the world [4]. Smoking in adolescent can be a gateway to other types of drug abuse and can cause various health problems, including frequent upper respiratory infections, delayed lung development, decreased maximum vital capacity, and lung cancer [5,6]. Smoking can also affects fat and lipoprotein metabolism. Smokers tend to have higher total cholesterol (TC), triglyceride (TG), and low density lipoprotein (LDL) level, and lower high density lipoprotein (HDL) than non smokers [7]. Studies on lipid levels of adolescent smokers have been conducted by Waqar, Afrin et al., and Prastyanto et al. [8,9,10]. A case control study, aged 12–19 years old, by Afrin et al. compared HDL level between adolescent smokers and non smokers, showing a significantly lower level of HDL in adolescent smokers [9]. A cross sectional studies by Waqar (aged 14–19 years) and Prastyanto et al. (aged 15–18 years) showed higher levels of TG and LDL, and lower HDL in adolescent smokers [8,10]. Although studies regarding lipid profile in adolescents have been conducted previously, those studies did not analyze lipid profile simultaneously. This study analyzed lipid profile of adolescents simultaneously using multivariate analysis. The aim of this study was to determine the difference of TC, LDL, and HDL levels between adolescent smokers and non-smokers.

2. Methods

2.1. Setting

This cross-sectional study was performed from February to April 2016 in several middle class high schools in Bandung city, the second most densely populated city in country.

2.2. Inclusion and Exclusion Criteria

Subjects were adolescent, aged 15–18 years, who were divided into two groups: smokers and non-smokers. The smokers were defined as those who had smoke at least once every week for one last year. Subjects who were obese, had chronic illness such as diabetes mellitus, liver

and kidney dysfunction, thyroid disease, on a diet program, and had a history of using lipid-lowering medications and alchohol consumption were excluded.

2.3. Data Collection

Body weight and height were measured and the BMI was calculated as weight/height^2 (kg/m^2). Sample size was estimated to be 50 subjects per group, calculated by unpaired categorical analysis with α= 0.05, β=0.20, and power=0.8. Simple random sampling was performed to obtain 50 smokers and 50 non-smokers for our study. Main outcomes were levels of serum TC, LDL, and HDL. Based upon the American Academy of Pediatric guidelines for children and adolescent, total cholesterol was defined as normal (<170 mg/dL), borderline (170 to <200 mg/dL) or as abnormal (200 mg/dL or greater). The LDL cholesterol was defined as normal (<110 mg/dL), borderline (110 to <130 mg/dL) or abnormal (130 mg/dL or greater) [11]. Based on American Heart Association normal level of HDL cholesterol for children was defined as greater than 35 mg/dL [12]. Data on smoking status, number of cigarettes per day and duration of smoking were collected using WHO Monica questionnaires [13]. Three mililiters of venous blood samples were drawn into EDTA (*ethylene diamine tetra acetic acid*) tubes and were processed using direct or homogenous methods using commercial kits Architec C-4000 (Abbot®).

2.4. Statistical Analysis

We analyzed the mean differences using MANOVA test. The chi square analysis (x^2) is used to analyze association between abnormality serum TC, LDL, HDL levels with smoking status. P values of <0.05 were considered to be statistically significant. The data obtained were evaluated using by SPSS for Windows version 15.00 statistical program

The study has been approved by the Research and Ethics Committee of the Universitas Padjadjaran Medical School, Indonesia, and written both subjects and parental consents were obtained.

Table 1. Characteristics of the subjects of smokers and non-smokers group

	Groups	
	Smokers (n=50)	**Non-smokers (n=50)**
Age (year)		
15	9	13
16	14	16
17	27	21
X (SD)	16.8 (0.82)	16.6 (0.89)
Median	17.1	16.7
Range	15–17.9	15.1–17.90
Body Mass Index		
X (SD)	20.9 (3.01)	20.0 (3.22)
Median	20.2	19.80
Range	16.40–27.10	15.30–27.40
Z-score		
X (SD)	-0.32 (1.01)	-0.44 (1.14)
Median	-0.4	-0.47
Range	-2.00–1.82	-2.74–1.92

Note: n: total subjects, SD: standard deviation.

3. Results

There were 426 subjects included in the study, which consisted of 226 girls and 206 boys. There was only 2 girls who smoke. From 206 boys, 162 matched the inclusion criteria (97 smokers and 65 non-smokers) and 44 were excluded (30 obese, 1 using lipid lowering medication, 4 drinks alcohol, 9 refuse blood taken). A total of one hundred subjects participated in this study, which consisted of 50 smokers and 50 non-smokers. Characteristics of the subjects were similar between the two groups as shown in Table 1.

In the smoking group, a mean age of first smoking was 14.4 (10–16) years. The smokers smoked 1–12 cigarettes per day with an avarage 5 cigarettes/day and smoked almost every day that is 5 (1–7) days in one week. Based on the duration of smoking, the average duration of smoking is 2.4 months.

The comparison of lipid profile categorized as normal, borderline, and abnormal between both groups is shown in Table 2. We found more subjects with abnormal serum levels of TC, LDL, and HDL in the smoking group compared to the non smoking group.

Table 2. Comparison of parameter between smokers and non-smokers

Variable	Groups		p value*
	Smokers (n=50)	Non-smokers (n=50)	
TC (mg/dL)			**0.006**
Normal (<170)	29	41	
Borderline (170–199)	14	9	
Abnormal (>200)	7	0	
LDL (mg/dL)			
Normal (<110)	31	40	**0.002**
Borderline (110–129)	8	8	
Abnormal (≥130)	11	2	
HDL (mg/dL)			
Normal (≥35)	36	47	**0.020**
Abnormal (<35)	14	3	

* The chi square analysis (x^2).

The chi square analysis (x^2) showed association between abnormality serum TC, LDL, HDL levels with smoking status (p=0.006, p=0.025, and p=0.006) respectively. The adolescent smokers had significantly higher TC and LDL, as well as lower HDL level compared to the non-smokers (Table 3).

Table 3. Comparison of lipid between smokers and non-smokers

Variable	**Groups**		**p value***
	Smokers (n=50)	**Non-smokers (n=50)**	
TC (mg/dL)			
X (SD)	161.7 (32.2)	150.6 (19.5)	**0.045**
Range	93–259	94–187	
LDL (mg/dL)			
X (SD)	107.6 (29.2)	92.6 (19.8)	**0.003**
Range	54–195	44–143	
HDL (mg/dL)			
X (SD)	38.7 (6.39)	42.8 (7.08)	**0.003**
Range	26–55	32–66	

*F Hotteling's = 1,690.21; p<0.001.

MANOVA test results showed significant differences between the two groups in terms of TC (p= 0.045), LDL (p= 0.003), and HDL (p= 0.003) levels.

4. Discussion

The subjects of this study were adolescent boys, because we only found 2 girls who smoke. We classified lipid profile levels in both groups into abnormal, borderline, and normal. This study found more subjects with abnormal levels of TC, LDL, and HDL in the smoking group compared to the non-smoking group. Chi square (x^2) analysis showed an association between TC, LDL and HDL level with smoking status.

This study showed differences in lipid profile between smoking and non smoking groups. We found serum TC and LDL significantly higher in smokers as compared to non-smokers and the serum HDL levels were significantly lower in smokers as compared to non-smokers group although the mean levels of TC, LDL and HDL of both groups were within normal limits. The results were similar to Waqar's and Prastyanto et al. although the analysis used were different, this study used Manova multivariate analysis to determine lipid profile as the outcome of body response happening at the same time which was analyzed simultaneously. The study result could not be compared to the study by Afrin et al. which only compared HDL level between the smoking and non smoking group.

Normal mean lipid profile values of smokers group in this study is 29%. It is probably due to 10–20% decrease of TC and LDL levels in adolescence [14]. Other contributing factor on normal lipid profile values in this study is the mean duration of smoking among the subjects was 2.4 years with the longest duration of 6 years and majority of these smokers were light smokers (<10 cigarettes/days), only two moderate smokers (10–20 cigarettes/day) and no heavy smoker (>20 cigarettes/day) was found, which probably would not have an effect yet on the lipid levels. In contrast with previous study in adult smoker, with majority were moderate dan heavy smokers, which has shown dyslipidemia condition in smoking group [15,16].

In this study, abnormal TC level found in smokers was 41%. The abnormal TC level due to the stimulation of adrenal sympathetic system by nicotine level which releases cathecolamine would induce lipolysis in adiposal tissue, thus increasing levels of plasma free fatty acid, promoting cholesterol synthesis and secretion in the liver [16]. Higher TC levels in smokers could also be due to increasing *3-hydroxy-3 methyl-glutaryl CoA reductase* (*HMG-CoA reductase*) activity and increased incorporation of labelled acetat into cholesterol [17]. The increase of TC serum level is one of the factors causing atherosclerosis [8].

This study found significantly abnormal serum LDL level in the smoking group than the non-smoking group. It has been described that nicotine increases the circulatory pool of atherogenic LDL via accelerated transfer of lipids from HDL and impaired clearance of LDL from plasma compartment therefore it increases the deposition of LDL cholesterol in the arterial wall [18]. Increa sed LDL in smokers also due to impaired LDL receptor function due to metabolites in cigarettes. Increased level of LDL is associated with increased risk of coronary heart disease [19].

This study also showed more subject with abnormal serum HDL level in the smoking group compared to the non-smoking group. It is due to impairing synthesis and secretion apolipoprotein-AI (Apo-A1), a major component of HDL particles and decrease *lecitin cholesterol acyltransferase* (LCAT) enzymes activity. Maturation of HDL may be affected by reduced LCAT activity, thereby inducing rapid clearance of nascent HDL from circulation [7]. During puberty and early teens, HDL level is decreased, especially in boys. Sex hormone such as testosterone have an important effect in HDL level, testosteron increase is associated with decrease of HDL level through inhibition of Apo-A1 inhibition. In this group, smoking could further lower HDL level, which in turn increase the risk of early atherosclerosis [8].

This study shows differences in lipid profile between smoking and non smoking group, however, the results were in normal range. The development of the differences into dyslipidemia would need further study.

The limitation of this study includes the cross sectional design which only allows one time reading of TC, LDL and HDL thus further prospective research should be conducted to ensure monitoring. This study also did not investigate the triglycerides level because it requires fasting preparation before the examination, which is hard to be done by the subject. Another limitation is that the smoking status is determined only based on questionnaire, which did not accurately represent nicotine level in the body. The questionnaire also did not specify the type of cigarette smoke, which may have different nicotine levels. This study did not include data of physical activity and nutritional intake such as high carbohydrate and high fat diets, which could affect the lipid profile level, thus also a limitation. Finally, this study did not exclude familial hypercholesterolemia, which could present as a study bias.

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5. Conclusion

This study shows that serum TC, LDL, and HDL levels in adolescent smokers are statistically different compared to those in adolescent non-smokers.

Conflict of Interest Statement

No competing interest to declare.

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