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Original Article

Lipid profiles in smoking and non-smoking male adolescents

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Abstract

Background The prevalence of smoking in adolescents tends to increase. Smoking is associated with a higher risk of dyslipidemia.

Objective To compare the lipid profiles of tobacco-smoking and non-tobacco-smoking male adolescents.

Methods We performed a cross-sectional study in three vocational high schools in Yogyakarta from January to April 2011. Data on smoking status, duration of smoking and number cigarettes consumed per day were collected by questionnaires. We randomly selected 50 male smokers and 50 male non-smokers as the study subjects.

Results Mean differences between smokers and non-smokers were 44.5 (95%CI 28.7 to 60.1) mg/dL for triglyceride levels; 8.0 (95% CI 1.0 to 14.9) mg/dL for low density lipoprotein (LDL) cholesterol; 11.8 (1.1 to 22.4) mg/dL for total cholesterol and -5.7 mg/dL (95% CI -8.8 to -2.6) for high density lipoprotein (HDL) cholesterol. Mean differences (95% CI) between smokers who had engaged in smoking for >2 years and those who had smoked for ≤2 years were -18.1 (95% CI -33.9 to -2.3) mg/dL for total cholesterol; -49.4 (95% CI -67.2 to -31.5) mg/dL for triglycerides. Mean differences between those who smoked >5 cigarettes/day and ≤5 cigarettes per day were -18.4 (95% CI -32.8 to -4.1) mg/dL for total cholesterol and -29.1 (95% CI -53.6 to -4.6) mg/dL for triglycerides.

Conclusion Smoking more than 5 cigarettes/day significantly increases total cholesterol, LDL cholesterol, and triglyceride levels, as well as reduces HDL cholesterol levels; while smoking more than 2 years significantly increases total cholesterol and triglyceride levels. **[Paediatr Indones. 2014;54:232-5.]**

Keywords: *smoking, lipid profile, adolescent*

Smoking remains one of the major causes of morbidity and mortality in the world. Cigarettes have indirectly killed about 1 million people worldwide, more than the combined deaths by tuberculosis, HIV/AIDS, and malaria.[1] The smoking epidemic is increasing rapidly in developing countries. According to the *Global Youth Tobacco Survey*, the prevalences of smoking among male adolescents in Argentina in 2003, in Thailand in 2005, and in Indonesia in 2006 were 22.4 %, 21.7%, and 24.1%, respectively; whereas the prevalences of tobacco smoking among female adolescents in the same countries and periods of time were 27.5%, 8.4%, and 4.1%, respectively.[1] According to the *National Socioeconomic Survey* (NSES) in 2001, the prevalence of smoking in Yogyakarta among people aged ≥15 years were 53.7% in males and 0.2% in females. In 2001, the age group of 15-19 years ranked first as the age of onset of smoking, with 58.9%, an increase from 54.6% in 1995.[2]

The adverse effects of smoking on health have been documented since 1950.[2] Nicotine and carbon

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monoxide have been directly related to the development of clinical diseases, abnormal serum lipid levels and atherogenic lipoprotein buildup.[3] In the last 40 years, the process of atherosclerosis in cardiovascular diseases has been observed to begin early in life and grow progressively, with genetic and environmental factors affecting the progression of the disease.[3,4]

Studies in children and adolescents have shown smoking to be associated with elevated lipid profile levels. In this study, we evaluated the differences in lipid profiles between tobacco-smoking and non-tobacco-smoking male adolescents.

Methods

We performed a cross-sectional study from January to April 2011 in three vocational high schools in Yogyakarta, namely PIRI 1, Taman Siswa, and Perkebunan. We included male adolescents, aged 15-18 years who had been smoking more than one cigarette/day for at least one year into the smoking group and those who had never smoked into the non-smoking group. We excluded adolescents who were obese, former smokers, passive smokers, had a history of alcohol consumption or had used lipid-lowering medications.

We estimated the required sample size to be 50 subjects per group, calculated by unpaired categorical analysis with $\alpha = 0.05$, $\beta = 0.20$ and power = 0.8. Out of 1,386 male students in the three schools, 346 students met the inclusion criteria, consisting of 232 smokers and 114 non-smokers. Of 232 smokers, 146 were excluded (4 obese, 4 alcohol-drinking and 138 passive smokers). Of 114 non-smokers, 41 adolescents were excluded (6 obese, 35 passive smokers). Therefore, we recruited 86 adolescent smokers and 73 non-smokers. Simple random sampling was performed to obtain 50 smokers and 50 non-smokers for our study.

Main outcomes were levels of serum total cholesterol, triglycerides, LDL cholesterol, and HDL cholesterol. Data on smoking status, duration and number cigarretes/day were collected using questionnaires (*Standard Question on Use of Tobacco Among Adolescents*).

Lipid profile was measured at the *CITO Laboratory*. Venous blood samples were taken in the morning after fasting for 10-12 hours, then stored in red-stoppered tubes to be examined on the same day by enzymatic methods using the *Beckman Coulter CX5 Automatic Chemistry Analyzer*. This study was approved by the Commission on Medical Research Ethics, Universitas Gadjah Mada Medical School. All subjects' parents provided written informed consent.

We analyzed the mean differences using Student's T-test. P values of < 0.05 were considered to be statistically significant. All analyses were perfomed using *SPSS 15.0* for Windows (Chicago, IL, USA).

Results

One hundred subjects participated in this study, consisting of 50 smokers and 50 non-smokers. Characteristics of the subjects were similar between two groups as shown in **Table 1**.

The adolescent smokers had significant higher of total cholesterol, LDL cholesterol, and triglycerides levels, as well as lower HDL cholesterol level compared to the non-smokers (**Table 2**).

Table 1. Basic characteristics of subjects

Characteristics	Smokers (n = 50)	Non-smokers (n = 50)
Mean age (SD), years	16.4 (0.9)	16.7 (1.0)
Mean weight (SD), kg	54.7 (7.2)	54.3 (7.6)
Mean height (SD), cm	166.1 (4.2)	165.2 (5.8)
Mean BMI (SD), kg/m^2	19.6 (2.3)	19.8 (2.1)

Table 2. Differences in lipid profiles between smokers and non-smokers

Lipid profiles	Smokers (n = 50)	Non-smokers (n = 50)	Mean difference (95%CI)	P value
Mean total cholesterol (SD), mg/dL	146.3 (26.6)	134.6 (27.0)	11.8 (1.1 to 22.4)	0.03
Mean HDL cholesterol (SD), mg/dL	39.6 (7.2)	45.3 (8.4)	-5.7 (-8.8 to -2.6)	<0.001
Mean LDL cholesterol (SD), mg/dL	80.1(23.3)	72.1(08.4)	8.0 (1.0 to 14.9)	0.03
Mean triglycerides (SD) , mg/dL	130.9 (44.9)	86.5 (33.3)	44.5 (28.7 to 60.1)	<0.001

Adolescents who had smoked for more than 2 years had significant higher of total cholesterol and LDL cholesterol levels compared to they who had smoked for less than 2 years (**Tabel 3**). Regarding the number of cigarettes/day smoked by adolescents, they who smoked more than 5 cigarettes/day had worse lipid profiles compared to the adolescents who smoked less amount of cigarettes per day (**Tabel 4**).

Table 3. Lipid profiles according to duration of smoking

Lipid profiles	Duration of smoking		Mean difference (95% CI)	P value
	≤ 2years (n=15)	>2 years (n=35)		
Mean total cholesterol (SD), mg/dL	133.7 (20.2)	151.8 (27.4)	-18.1 (-33.9 to -2.3)	0.03
Mean HDL (SD), mg/dL	39.5 (7.4)	39.7 (7.2)	-0.2 (-4.7 to 4.4)	0.95
Mean LDL (SD), mg/dL	73.8 (17.3)	82.8 (25.3)	-8.9 (-23.4 to 5.4)	0.22
Mean triglycerides (SD), mg/dL	96.3 (17.5)	145.7 (45.2)	-49.4 (-67.2 to -31.5)	<0.001

Table 4. Lipid profiles according to cigarette consumption/day

Lipid profiles	≤5 cigarettes/day (n = 24)	>5 cigarettes/day (n = 26)	Mean difference (95% CI)	P value
Mean total cholesterol (SD), mg/dL	136.8 (19.3)	155 (29.7)	-18.4 (-32.8 to -4.1)	0.03
Mean HDL (SD), mg/dL	42.0 (7.7)	37.5 (6.1)	4.5 (0.6 to 8.5)	0.01
Mean LDL (SD), mg/dL	70.3 (17.9)	89.1 (24.4)	-18.7 (-31.0 to -6.5)	0.04
Mean triglyceride (SD), mg/dL	115.8 (30.3)	144.9 (51.9)	-29.1 (-53.6 to -4.6)	0.02

Discussion

We found that adolescents who smoked for more than 2 years had significantly higher total cholesterol and triglyceride levels compared to adolescents who smoked for 2 years or less. However, LDL and HDL cholesterol levels did not differ significantly between these two groups. We also compared the lipid profiles of adolescents who smoked > 5 cigarettes per day to those who smoked 5 of fewer cigarettes per day. Those who smoked > 5 cigarettes/day had significantly higher total cholesterol, LDL cholesterol, and triglyceride levels and significantly lower HDL cholesterol than those who smoked ≤ 5 cigarettes/day, similar to results from other studies.[6-10] Previous studies have shown that smoking more cigarettes and for a longer duration increases nicotine levels which can stimulate cathecolamin hormones from adrenal glands and hormones such as cortisol and growth hormone leading to increased lipolysis. This effect can increase free fatty acid concentration in plasma that stimulates the secretion of hepatic very low density lipoprotein (VLDL) and triglycerides.[5] In addition to the above mechanisms, smoking also increases insulin resistance, leading to hyperinsulinemia. Levels of LDL, VLDL and triglycerides increase in people with hyperinsulinemia due to decreased activity of lipoprotein lipase.[6,7] Plasma lipase is an important regulator of plasma lipoprotein concentration. Hepatic lipase, activated in smokers, converts VLDL into LDL.[7,8] McCall *et al.* stated that smoking also plays a role in inhibiting the enzyme lecithin-cholesterol acyl transferase, which is responsible for maintaining adequate levels of HDL in the circulation.[9]

A Brazilian study in 2007 involving 452 adolescents (246 females and 206 males) aged 15-18 years showed that the levels of total cholesterol and LDL cholesterol were higher and HDL cholesterol was lower in adolescent smokers compared to adolescent non-smokers.[10] In addition, Waqar from India showed that smoking adolescent boys aged 14-19 years had significantly higher LDL cholesterol and triglyceride levels and significantly lower HDL cholesterol compared to adolescent non-smokers.[11]

A meta-analysis study conducted in children aged 8-19 year old also showed different results. Levels of HDL cholesterol were lower, whereas triglycerides, LDL cholesterol, and VLDL levels were significantly higher except for total cholesterol in adolescent smokers compared to non-smokers.[12]

Several studies conducted in adult smoking populations documented that race and gender

influenced the association between smoking and dyslipidemia. African smokers had lower cholesterol levels than Asian smokers. This reflects the role of genetic factors.[14] Similarly, female smokers had a higher risk of dyslipidemia compared to male smokers, presumably due to the interaction between nicotine and estrogen, causing a drop in estrogen levels that affects lipid metabolism, leading to dyslipidemia.[7,13,14]

Our study has several limitations. First, other factors affecting dyslipidemia could not be excluded such as diet, genetics, hormones, and physical activity. Second, due to the cross-sectional study design we could not infer causality between smoking and dyslipidemia. Further study is needed to investigate the association between smoking and dyslipidemia in adolescents with a prospective cohort study. Third, assessment of smoking and non-smoking status, duration of smoking, and cigarette consumption per day using questionnaires may result in possible errors or recall bias.

In conclusion, in adolescent males, smoking more than 5 cigarettes/day and for more than two years are significantly associated with increased levels of total cholesterol, triglycerides and LDL, as well as decreased HDL cholesterol level.

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