FIBER AND FAT INTAKE, SMOKING HABITS, BODY MASS INDEX AND CENTRAL OBESITY AND ITS ASSOCIATION WITH LIPID PROFILE OF MAN IN GATOT SUBROTO HOSPITAL HEART POLYCLINIC

Kevinti Febrina[1], Adhila Fayasari[1*]
[1]Nutrition Study Program, Faculty of Public Health, Universitas Binawan, Indonesia
*E-mail: fayasari@gmail.com

ABSTRACT

An increase in blood lipid profile escalates risk of coronary heart disease (CHD). CHD is the leading cause of death in the world. This study was intended to analyze the relationship between fat intake, fiber intake, smoking habits, Body Mass Index (BMI) and central obesity and lipid profile among patients in cardiology clinic. This study used cross sectional design with 60 subjects aged >29 years who were outpatients in the Cardiaology Polyclinic at Gatot Soebroto Hospital. Central obesity was measured using metline by measurung waist circumference; BMI was converted from measurements of body weight and height; characteristics and smoking habits were taken using a questionnaire; and food intake was measured using Semi Quantitative FFQ. Lipid profile data was collected through secondary data. Bivariate data analysis on categorical data used chi-square, Fisher-exact test or spearman ordinal correlation and stratification analysis using ANOVA or kruskall-wallis. There were 78.3% subjects who had abnormal LDL, 76.7% had abnormal HDL, 80% had abnormal triglycerides and 78.3% had abnormal cholesterol. There were signifiant relationship between fat intake, smoking habits, BMI and central obesity to the lipid profile (LDL, HDL, triglycerides and cholesterol) (p <0.05). Fiber intake was not significantly related to lipid profile. However, based on stratification analysis,the more fiber intake the lower levels of LDL profiles, triglycerides and cholesterol, and the higher levels of HDL.

Keywords: fat intake, fiber intake, smoking habits, obesity, lipid profile

BACKGROUND

Coronary Heart Disease (CHD) is a leading cause of death globally. A total of 17.9 million people died due to CHD in 2016 which represented 31% of global deaths (WHO, 2017). Deaths from cardiovascular disease in the Asia Pacific region are mostly due to coronary heart disease and myocardial infarction (AHA, 2012). In Indonesia, coronary heart disease suffered by approximately 2.6 million people or 1.5% of total population. The highest prevalence found in West Java (Ministry of Health of the Republic of Indonesia, 2013). The best indicators of CHD risk include fat profile. The results of the National Health Survey showed high total cholesterol prevalence of 35.9% population had high level of total cholesterol, respectively 76.2% and 22.9% had abnormal LDL and triglycerides, and 22.9% had low level of LDL (Ministry of Health of the Republic of Indonesia, 2013).

Risk factors for the emergence of CHD include low physical activity, obesity, excess food intake, cigarette consumption and alcohol drinking habits (WHO, 2017). Excess food intake, that is, an unspecified and uncontrolled intake based on the amount of intake and frequency of food, can burden the heart's work. A diet high in fat (saturated fat or trans fat) can result in blockage and narrowing of the coronary arteries. Saturated fat (fried fat) from fried foods is harmful to the body because it stimulates the liver to produce cholesterol which will precipitate and inhibits the flow of oxygen in the bloodstream so that it disrupts the metabolism of heart muscle cells and is at risk of CHD events (Hardinsyah and Supariasa, 2017).

Cigarette consumption can inhibit the production High Density Lipoprotein (HDL) so that the blood clots easily, and is at high risk for clogged arteries (Nilawati et al., 2008). Moreover, High LDL levels are found more in

smokers. With the increase in LDL, the production of HDL also decreases because the acroline in cigarettes damages HDL and inhibits the process of transporting fat to liver tissue (Sanhia, 2015).

Body Mass Index (BMI) can be a marker of fat profile disorder. The results of the study by Ecol (2008) state that both men and women in all age groups who have overweight nutritional status will be at risk of increasing total cholesterol and LDL levels (Ecol, 2008).

Central obesity results from metabolic complications and is closely related to low LDL cholesterol concentrations (Li Xu et al., 2012). The accumulation of visceral fat contributes to the low level of adiponectin. Adiponection acts as anti-diabetic, anti-hypertensive and anti-inflammatory. The lower level of adiponectin increased risk of metabolic and cardiovascular diseases (Matsuzawa, 2010). Thus, the researchers intended to analyze the association of fat and fiber intake factors, smoking habits, Body Mass Index and central obesity on the profile of blood fat in men in the Coronary Heart Disease Polyclinic in RSPAD Jakarta.

METHODOLOGY

This study was a cross sectional design in outpatients at the Gatot Soebroto Central Police Hospital. The samples in this study were selected based on inclusion criteria, consisted of male outpatients, aged >29 years old, diagnosed with CHD, and had lipid profile data. Exclusion criteria are patients who have comorbidities. The sampling technique using purposive sampling method.

Data collection was carried out using a structured questionnaire consisting of respondent identity data, anthropometric data, lipid profile and smoking habits. The smoking questionnaire consisted of open-ended questions drawn from the study of Jain & Ducatman (2018) and then modified after tested on patients in the Gatot Soebroto Army Hospital. Ethical approval was obtained from the University of Indonesia with number KET737 / UN2.F1 / ETIK / PPM.00.02 / 2019.

Meal intake data was measured using a Semi-Quantitative Frequency (SQ-FFQ) questionnaire containing 88 items of food consumed including the frequency of consumption (daily, weekly, monthly) within past month. Food intake data obtained through SQ-FFQ then was converted into fat and fiber intake per day using nutrisurvey software. The result of fat intake compared to the RDA was categorized as good intake (≥80% -110%) or excessive intake (≥110%) (WNPG, 2004). Fiber intake was classified based on mean of the fiber intake (16.7 g).

Measured anthropometric datas included weight and height. Weight was measured using a Camry digital scale with an accuracy of 0.1 kg, whereas height was measured with a microtoise (GEA) with an accuracy of 0.1 cm. Body weight and height were converted to Body Mass Index and categorized as normal (18.5-25 kg/m2) and overweight (>25.1 kg/m2). Samples with Body Mass Index <18.5 was excluded in this study. Central obesity was measured by metline (brand: Butterfly) with the accuracy of 0.1 cm, then categorized as normal (<90 cm), and central obesity (≥ 90 cm) (WHO, 2008).

Lipid profile was taken from secondary data in RSPAD Cardiac Polyclinic in the last 3 days. Fat profile datas were categorized as abnormal with LDL cut-off (> 100 mg/dL); triglycerides (>150 mg/dL)); total cholesterol (> 200 mg/dL), except for HDL which defined as abnormal if <40 mg/dL) (Mahan and Escott Stump, 2013)

For the data analysis, we employed univariate analysis (distribution and frequency) for age, education, occupation, family history and medication variables. Furthermore, bivariate analysis was carried out to analyze the relationship of independent variables with categorical dependent using chi-square test (α <0.05), or if expected count <5, using the Fischer exact test or Spearman ordinal correlation if the categorical independent variable is more than 3. The startification analysis was done using ANOVA if the data were normally distributed or Kruskall-Wallis if the data were not normally distributed.

RESULT AND DISCUSSION

Respondents in this study were mostly 50-64 years old (70.0%), the rest were 30-49 years old (26.7%) and 65-80 years old (3.3%). The educational status is dominated by the level of

Table 1. Sociodemography Characteristic

Variable	n	%
Age (y.o)		
30-49	16	26.7
50-64	42	70.0
65-80	2	3.3
Education[1]		
Low	28	46.7
High	32	53.3
Employment status		
Employed	60	100
Unemployed	0	0
Family History		
Exist	32	53.3
Do not exist	28	46.7
Consumed drugs		
Amlodipin	14	23.3
Valsartam	7	11.7
Novorapid	9	15.0
Lovastatin	16	26.7
Atorvastatin	14	23.3
Smoking status		
Active smoker	42	70.0
Former smoker	13	21.7
Non-smoker	5	8.3

Notes : 1) high education defined as >9 years of formal education;

Table 2. The Distribution of Anthropometry, Nutrient Intake, and Lipid Profile

Variable	n	%
BMI		
Normal (>18,5-25)	26	43.3
Overweight (>25,1 kg/m^2)	34	56.7
Central Obesity		
No (<90cm)	17	28.3
Yes (≥90cm)	43	71.7
Fat Intake		
Good (80-109%)	41	68.3
Excessive (≥110%)	19	31.7
Fiber intake		
Less (<mean)	30	50.0
Good (≥mean)	30	50.0
LDL		
Normal	13	21.7
Abnormal	47	78.3
HDL		
Normal	14	23.3
Abnormal	46	76.7
Trigliserida		
Normal	12	20.0
Abnormal	48	80.0
Kolesterol Total		
Normal	13	21.7
Abnormal	47	78.3

higher education as many as 32 people (53.3%) who overall work (100.0%).

Respondents who have a family history of high blood lipid profiles were 32 people (53.3%). Most common drug consumed by the subjects were lovostatin (26.7%). Majority of subjects were active smokers (70.0%).

Over and normal nutritional status had similar proportion (56.7% vs 43.3%), but there is a tendency of respondents in the study to have fat in the abdomen with a greater proportion of central obesity (71.7 %). Consumption of excess fat was found in 41 people (68.3%), while good fiber intake only applied for 30 people (50.0%). Abnormal lipid profile were found ini most of the subjects. In accordance, 76.7%, 78.3%, 80.0%, and 78.% had abdormal LDL, HDL, triglycerides, and total cholesterol (Table 2).

The results of bivariate analysis between food intake, smoking habits, BMI, and central obesity with lipid prifil can be seen in Table 3. Fat intake, smoking habits, BMI and central obesity were significantly related to LDL (p <0.05).

The percentage of excessive fat intake in the abnormal LDL group was greater. Whereas, a higher proportion of active smokers was commonly found in abnormal LDL group. Overweight/obesity as well as central obesity were also frequent in abnormal LDL group.

Other fat profiles such as HDL, triglycerides and total cholesterol were influenced by fat intake, smoking habits, BMI and central obesity. However, fiber intake was not significantly related to blood lipid profile.

Overall fat intake has a significant contribution of increasing blood lipid profile (LDL, HDL, triglycerides and total cholesterol). According to Rahma (2017) and Putri (2016), excess fat intake increased the risk of developing CHD. A study by Rahma (2017) showed subjects with excessive fat intake had 13.5 times greater odds of experiencing CHD (p<0.05).

Excessive fat intake can increase blood lipid so that it brings adverse effects on health (Fathila et al., 2015). Fat accumulation can easily trigger plaque formation around the heart's blood vessels, making it easier to block and inhibit oxygen carried to the heart.

Analysis of the relationship between BMI and lipid profiles showed that respondents with more BMI (obesity) tended to have abnormal

Table 3. Relationship between Food Intake, Smoking Habit, BMI, and Central Obesity with LDL, HDL, Triglycerides and Total Cholesterol

Variable	LDL			HDL			Trigliserida			Cholesterol Total		
	Normal n (%)	Abnormal n (%)	p value	Normal n (%)	Abnormal n (%)	p value	Normal n (%)	Abnormal n (%)	p value	Normal n (%)	Abnormal n (%)	p value
Fat intake												
Good	12 (63.2)	7 (36.8)	0.000[b]*	12 (63.2)	7 (36.8)	0.000[a]*	11 (57.9)	8 (42.1)	0.000[b]*	11 (57.9)	8 (42.1)	0.000[a]*
Excessive	1 (2.4)	40 (97.6)		2 (4.9)	39 (95.1)		1 (2.4)	40 (97.6)		2 (4.9)	39 (95.1)	
Fiber intake												
Less	8 (22.2)	28 (77.8)	0.898[a]	9 (25.0)	27 (75.0)	0.709[a]	8 (22.2)	28 (77.8)	0.598[a]	9 (25.0)	27 (75.0)	0.443[a]
Good	5 (20.8)	19 (79.2)		5 (20.8)	19 (79.2)		4 (16.7)	20 (83.3)		4 (16.7)	20 (83.3)	
Smoking status												
Active smoker	1 (2.4)	41 (97.6)	0.000[c]*	3 (7.1)	39 (92.9)	0.000[c]*	1 (2.4)	41 (97.6)	0.000[c]*	2 (4.8)	40 (95.2)	0.000[c]*
Former smoker	8 (61.5)	5 (38.5)		8 (61.5)	5 (38.5)		7 (53.8)	6 (46.2)		7 (53.8)	6 (46.2)	
Non-smoker	4 (80.0)	1 (20.0)		3 (60.0)	2 (40.0)		4 (80.0)	1 (20.0)		4 (80.0)	1 (20.0)	
BMI												
Normal (>18,5-25)	12 (46.2)	14 (53.8)	0.000[b]*	11 (42.3)	15 (57.7)	0.002[a]*	11 (42.3)	15 (57.7)	0.000[b]*	11 (42.3)	15 (57.7)	0.001[a]*
Overweight (>25,1 kg/m²)	1 (2.9)	33 (97.1)		3 (8.8)	31 (91.2)		1 (2.9)	33 (97.1)		2 (5.9)	32 (94.1)	
Central Obesity												
No (<90cm)	12 (70.6)	5 (29.4)	0.000[b]*	10 (58.8)	7 (41.2)	0.000[a]*	12 (70.6)	5 (29.4)	0.000[b]*	12 (70.6)	5 (29.4)	0.000[b]*
Yes (≥90cm)	1 (2.3)	42 (97.7)		4 (9.3)	39 (90.7)		0 (0.0)	43(100)		1 (2.3)	42 (97.7)	

Keterangan: *p<0.05(signifikan); [a]Pearson chi-square; [b]Fischer Exact test; [c]Spearman Ordinal Correlation

or risky lipid profiles. Waist circumference was associated with BMI (R=0.78; p<0.01), and there is an increase in the volume of visceral tissue in the abdomen (Gierach, et al., 2014). Indicators of BMI and waist circumference (central obesity) have a strong correlation with non-communicable diseases such as hypertension and diabetes mellitus (Susilawati et al., 2015; Deng et al., 2013). In the study of Hotama (2014). It was mentioned that higher BMI generally has a high body fat composition so that it is closely related to an increase in blood lipid levels

The results of data collection were summarized that respondents who have central obesity as a whole have an abnormal blood lipid profile. This causes an increase in total fat in the body that can

occur in visceral fat. Visceral fat is found in the abdominal cavity which can be known from the indicators of central obesity. The increase in the size of central obesity is in line with the increase in abnormal lipid profile levels (triglycerides and LDL) (Umegaki et al., 2008; Kamso, 2007). This result was in line with Sumarni (2016) which showed significant positive correlation between total cholesterol and central obesity with weak correlation strength ($p = 0.005$; $r = 0.342$) and a significant positive correlation between central obesity and triglycerides ($p = 0.002$; $r = 0.377$).

Fiber is a form of carbohydrate that cannot be digested because there is no fiber digestive enzyme in humans. Fiber consists of water-soluble and water-soluble fibers. Water-soluble fiber is

Table 4. Difference of LDL, HDL, Triglycerides and Total Cholesterol Level Based on Fiber Intake Quintil and Smoking Habit Stratification.

	Total	Active Smoker	Former Smoker	Non-Smoker	p-value
LDL					
Fiber Intake					
Q1 (< 15.8)	129.8 ± 47.5	153.9 ± 44.9	89.0 ± 26.7	94.3 ± 10.6	<0.05[a]
Q2 (15.8-16.7)	134.3 ± 37.6	138.0 ± 36.3	94.0 ± 0.0	-	<0.05 [a]
Q3 (16.8-17.7)	116.0 ± 33.9	129.3 ± 26.0	94.5 ± 31.8	69.0 ± 0.0	<0.001 [a]
Q4 (>17.7)	138.3 ± 43.5	152.2 ± 40.3	110.4 ± 32.0	-	<0.05 [a]
p value	<0.05 [a]				
HDL					
Fiber Intake					
Q1 (< 15.8)	40.7 ± 7.5	37.2 ± 6.4	45.3 ± 5.0	47.0 ± 6.7	<0.05 [a]
Q2 (15.8-16.7)	38.3 ± 3.5	37.4 ± 1.6	48.0 ± 0.0	-	<0.05 [a]
Q3 (16.8-17.7)	40.4 ± 6.9	37.0 ± 1.9	47.5 ± 8.7	46.0 ± 0.0	<0.001 [a]
Q4 (>17.7)	37.6 ± 4.5	35.8 ± 3.0	41.2 ± 4.6	-	<0.001 [a]
p value	0.125 [a]				
Triglycerides					
Fiber intake					
Q1 (< 15.8)	170.9 ± 50.9	192.5 ± 52.3	147.3 ± 19.9	129.5 ± 27.7	<0.05 [a]
Q2 (15.8-16.7)	184/0 ± 33.8	188.2 ± 31.3	138.0 ± 0.0	-	<0.05 [a]
Q3 (16.8-17.7)	174.1 ± 30.8	182.6 ± 25.2	163.3 ± 34.1	133.0 ± 0.0	<0.05 [a]
Q4 (>17.7)	192.8 ± 42.1	209.1± 39.1	160.2 ± 20.1	-	<0.001 [a]
p value	0.089 [a]				
Total Cholesterol					
Fiber intake					
Q1 (< 15.8)	223.3 ± 42.2	242.6 ± 42.8	190.7 ± 15.9	194.5 ± 16.9	<0.05 [a]
Q2 (15.8-16.7)	236.8 ± 28.0	240.3 ± 25.8	198.0 ± 0.0	-	<0.05 [a]
Q3 (16.8-17.7)	229.5 ± 30.1	241.5 ± 20.6	207.3 ± 33.8	198.0 ± 0.0	<0.05 [a]
Q4 (>17.7)	241.2 ± 38.0	258.2 ± 31.1	209.4 ± 24.2	-	<0.001 [a]
p value	0.158 [a]				

Notes: [a]Kruskall wallis

contained in fruits, vegetables, oats and barley which functions to reduce cholesterol through several mechanisms, while water-soluble fiber is contained in lignin, cellulose and hemicellulose found in gamdum, nuts, and seeds which functions to increase emptying the stomach and improve the digestive system (Soliman, 2019)

Analysis with chi-square test showed that there was no significant relationship between fiber intake and fat profile (LDL, HDL, triglycerides, and total cholesterol) (p> 0.05). This was presumably because the subject's fiber intake was still relatively low. The average fiber intake of 16.7 g, was still lower than the Indonesian Recommended Nutrient Intake which suggested a minimum fiber intake of 25-30 g/day or 38 g/day for men and 25 g/day for women. The low fiber intake cannot distinguish between proportions of subjects who had over and normal lipid profiles (Soliman, 2019).

The finding of this study was in line with the study by Kustiyah et al (2013), where also found no significant difference in fiber intake between subjects with normal and abnormal blood lipid profiles. This could be happened because fiber intake had indirect effect on blood lipid metabolism through bile acid metabolism, and estradiol pathway (Jenkis et al, 2000; Kay et al, 1980). These result was also consistent with a meta-analysis by Brown et al. (1999) which stated that the provision of fiber, especially water-soluble fibers only gives a small effect on reducing cholesterol.

There was a significant difference in LDL profiles based on fiber intake quintiles (p <0.05). The higher LDL level were found in higher quintiles of fiber intake. However, the lowest LDL level were found in subjects with fiber intake in quintiles 3 (16.8-17.7). Moreover, Stratification analysis based on smoking habit indicated that there was significant differences in lipid profile among subjects with various quintiles of fiber intake. LDL level of active smokers were greater compared to former and non-smokers. Among active smokers group, the higher fiber intake would followed by the decline of LDL, except for subjects with quintile 4 fiber intake. However, there were no significant differences in HDL, triglycerides and total cholesterol between various fiber intake quintiles.

The result of this study was dissimilar with study by Zhou et al (2015) which found that adjustment of several confounding factors including BMI, waist circumference, energy intake, and smoking habits affected the trend of relationship between HDL and fiber intake. The increase doses of fiber intake would enhance lipid level in men. When fiber intake was increased up to more than 30 g/day, a significant improvement on HDL and triglyceride/HDL ratio were found in male subjects (Zhou et al., 2015). In this study, the insignificant correlation between fiber intake and lipid profile could arise due to researchers did not adjust possible confounding variables such as energy intake and other habitual factors such as coffee consumption and physical activity.

This study confirmed that active smokers have abnormally higher lipid profile levels than smokers and non-smokers. Nicotine contained in cigarette can increase the production of adrenaline which results in decreased levels of HDL (High Density Lipoprotein) (Anies, 2006). Other possible mechanism on how smoking can change serum fat levels is that the absorption of nicotine causes secretion of catecholamines, cortisol, and growth hormones, which activating adenyl cyclase in adipose tissue. The process produces triglyceride lipolysis and releases free fatty acids, resulting in an increase in TG and VLDL liver synthesis (Jain and Ducatman, 2018). The decrease in HDL is also caused by an increase in free fatty acids and increased LDL production due to nicotine (Sanhia, 2015).

Researches by Trivedi et al. (2013) and Carlappa et al (2014) in 50 smokers and 50 nonsmokers showed a significant increase in total cholesterol, triglyceride, LDL and a significant decrease in HDL levels in smokers compared to nonsmokers. The long period of smoking and the number of cigarettes significantly influence the high risk of hypercholesterolemia.

There was a significant relationship between smoking and total cholesterol (p = 0,000) found in this study. Active smokers tended to have higher total cholesterol level compared to former and non-smokers. Furthermore, there was also meaningful relationship between BMI, central obesity and total cholesterol (p = 0.001). Subjects with higher BMI

and central obesity were more suspectible to have abnormal total cholesterol.

Table 5 shows that there were differences in LDL, HDL, triglyceride and cholesterol profiles between quintile fat intake (p <0.05). Further stratification analysis represented that only subjects with quintiles 1 fat intake (<63.9 g) had different level of all lipid biomarker based on smoking status. In addition, subjects with quintiles 4 fat intake also showed divergent level of HDL, triglycerides, and total cholesterol based on smoking status (>87.4 g).

Active smokers defined as respondents who were still smoking, while former smokers were respondents who have smoked but have been stopped. Blood lipid levels are higher in active smokers, then followed by former smokers and non-smokers. This was consistent with the results of a study in China that showed active smokers tended to have low triglyceride levels (Yan-Ling, 2012).

LDL and total cholesterol level of non smoker subjects with quintile 1 fat intake 1 was higher compared to former smokers. Unjustified dose of smoking in the study could posibbly affected this results. Meanwhile, non-smokers could be exposed to cigarette smoke from other people, which also did not assessed in this study. Some studies showed passive smokers had a tendency to experience elevated serum LDL levels, as do active smokers. Nicotine levels in cigarette sidestream can reach 4-6 times more than nicotine levels in mainstream cigarettes (U.S. Department of Health and Human Services, 2009; Andrews & Tingen, 2006; Susanna et al, 2003)

CONCLUSION

There was a relationship between fat intake, smoking habits, BMI and central obesity with the lipid profile (LDL, HDL, triglycerides and total cholesterol). Fiber intake was not significantly related to lipid profile. Based on stratification analysis, as fiber intake increase, the lower the LDL, triglyceride and cholesterol profiles, and the higher the HDL levels.

The limitation of this study limitations the use of secondary data on blood lipid profiles, so there is a possibility of bias from the obtained values even though this study has been limited to using data in the last 3 days. Further research should address drug consumption compliance, fiber types, saturated and unsaturated fat intake and nicotine content in cigarettes.

ACKNOWLEDGEMENT

REFERENCES

Andrews J. O., & Tingen M. S. (2006) The effect of smoking, smoking cessaction, and passive smoke exposure on common laboratory values in clinical settings: a review of the evidence. *Crit Care Nurs Clin N Am, 18:* 63-9

American Heart Association/American Stroke Association (AHA/ASA). (2012). Heart disease and stroke statistics-2012 Update. Retrieved from http://circ.ahajournals.org/content/125/1/e2.full?sid= b8024315-9878-42e9-a935-05edb3ec1427

Anies. (2006). Waspada ancaman penyakit tidak menular. Jakarta : PT Alex Media Komputindo

Brown, L., Rosner, B., Willet, W. W, & Sacks, F. M. (1999). Cholesterol-lowering effects of dietary fiber: a meta-analysis. *The American Journal of Clinical Nutrition, 69*(1). Retrieved from https://academic.oup.com/ajcn/article/69/1/30/4694117

Carlappa, K. B., Sathisha, T. G., & Veena, H. (2014). Sequels of Smoking on Blood Lipid Levels in a Rural Population of South India. *Research and Reviews: Journal of Medical and Health Sciences* 3(2)

Deng W. W, Wang J, Liu M. M, Wang D, zhao Y, Liu Y. Q, Wang H, & Dong G. H (2013) Body mass index compared with abdominal obesity indicators in relation to prehypertension and hypertension in adults: the CHPSNE study. *Am J Hypertension 26* (1):58-67 doi: 10.1093/ajh/hps001

Departemen Kesehatan Republik Indonesia. (2013). Riset Kesehatan Dasar DKI Jakarta Tahun 2013. Jakarta: Badan Penelitian dan Pengembangan Kesehatan Departemen Kesehatan RI.

Ecol, J. (2008). *A Study of correlation between lipid profile and body mass index (bmi) in patients*

with diabetes mellitus. Retrieved from http://www.krepublishers.com/02-Journals/

Fathila, L., Edward, Z., & Rasyid, R. (2015). Gambaran profil lipid pada pasien infark miokard akut di RSUP M. Djamil Padang periode 1 Januari 2011–31 Desember 2012. *Jurnal Kesehatan Andalas 4(2)* doi.org/10.25077/jka.v4i2.286

Gierach, M., Gierach, J., Ewertowska, M., Ardnt, A., & Junik, R. (2014) Correlation between body mass index and waist circumference in patients with metabolic syndrome. ISRN Endocrinology Retrieved from https://www.hindawi.com/journals/isrn/2014/514589/

Hardinsyah & Supariasa. (2017). *Ilmu gizi teori dan aplikasi*. Jakarta: Penerbit Buku Kedokteran EGC.

Hotama, A. (2014). Hubungan asupan energi dan zat gizi serta gaya hidup dengan profil lipid orang dewasa dyslipidemia [thesis]. Bogor: Institut Pertanian Bogor.

Jain & Ducatman (2018). Roles of Gender and Obesity in Defining Correlations Between Perfluoroalkyl Substances and lipid/lipoproteins. Sci Total Environ 653:74-81 doi: 10.1016/j.scitotenv.2018.10.362

Jenkins, D. J, Kendall, C. W, & Vuksan V. (2000). Viscous fibers, health claims, and strategies to reduce cardiovascular disease risk. *Am J Clin Nutr.71*(2):401–402. doi: 10.1093/ajcn/71.2.401.

Kamso, S (2007). Dislipidemia dan obesitas sentral pada lanjut usia di Kota Padang. *J. Kesehat. Masy. Nas. 2* (2), doi: 10.21109/kesmas.v2i2.274

Kay, R. M. (1980). Effects of dietary fibre on serum lipid levels and fecal bile acid excretion. *Can Med Assoc J. 123* (12):1213–1217.

Li Xu, Katashima, M., Yasumasu, T., & Li, K.J, (2012). Visceral fat area, waist circumference and metabolic risk factors in abdominally obese chinese adults. *Biomedical and Enviromental Science. 25*(2):141-148

Mahan, L. K., & Escott-Stump, S. (2013). Krause's Food & Nutrition Therapy. USA: Saunders/Elvisier

Matsuzawa, Y. (2010). Establishment of a concept of visceral fat syndrome and discovery of adiponectin. *Proc Jpn Acad Ser B Phys Biol Sci, 86*(2): 131–141.

Nilawati, Sri. (2008). *Care your self* Kolesterol. Jakarta: Penebar Plus

Putri. (2016). Hubungan asupan serat dan lemak total dengan kadar kolesterol total pada anggota polisi Polres Rembang [skripsi yang tidak dipublikasikan]. Surakarta: Universitas Muhammadiyah Surakarta

Rahma, H. H., & Wirjatmadi, R. B. (2017). Hubungan asupan zat gizi makro dan profil lipid dengan kejadian penyakit jantung koroner pada pasien lansia di Rumah Sakit Islam Jemursari Surabaya. *Media Gizi Indonesia*, *12*(2). doi: 10.20473/mgi.v12i2.129-133

Sanhia, Aji M., Pangemanan, Damajanty H.C., & Engka, Joice N.A. (2015). *Gambaran kadar kolesterol low density lipoprotein (LDL) pada masyarakat perokok di Pesisir Pantai*. Manado: Universitas Sam Ratulangi Manado.

Soliman, G. A. (2019) Dietary fiber, atherosclerosis, and cardiovascular disease. *Nutrients* 11(5):1155 doi: 10.3390/nu11051155.

Sumarni. (2016). Hubungan antara obesitas sentral dengan profil lipid pada dewasa obes. *Jurnal Ilmiah Kedokteran: Media Tadulako Vol 3 (3)*

Susilawati, M. D., Muljati, S., & Bantas, K. (2015). Perbandingan imt dan indikator obesitas sentral terhadap kejadian diabetes melitus tipe 2 (dmt2) (Analisis data sekunder baseline studi kohor PTM di kelurahan Kebon Kalapa Bogor tahun 2011). *Bul Penelit.Kesehat 43*(1):17-22

Susanna D., Hartono, B., & Fauzan H. (2003) Penentuan kadar nikotin dalam asap rokok. *Jurnal Ekologi Kesehatan 2*(3) :272-274.

Trivedi, R S., & Anand, A K. (2013) Effect of smoking on lipid profile. *Gujarat Journal of Otorhinolaryngology and Head and Neck Surgery 10(2):* 13-15.

Umegaki, H., Haimoto, H., Ishikawa, J. & Kario, K. (2008). Visceral fat contribution of insulin resistance in elderly people. *J. Am. Geriatr. Soc. 56,* 1373–1375.

U.S. Department of Health and Human Services. (2009). *The health consequences of involuntary exposure to tobacco smoke:* A Report of Surgeon General. Atlanta: Centers for Diseases Control and Prevention.

Kustiyah, L., Widhianti, M. U., & Dewi, M. (2013). Hubungan asupan serat dengan status gizi dan profil lipid darah pada orang dewasa dislipidemia. *Jurnal Gizi dan Pangan 8*(3): 195-200 doi.org/10.25182/jgp.2013.8.3.195-200

[WHO] World Health Organization (2017). Cardiovascular Factsheet. Diakses dari https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds) pada tanggal 24 Agustus 2019.

[WHO] World Health Organization. (2008) *Report of a WHO Expert Consultation: Waist Circumference and Waist-Hip Ratio,* Geneva: WHO Publisher, pp.24-27

[WNPG] Widyakarya Pangan dan Gizi VIII. (2004). *Ketahanan pangan dan gizi di era otonomi daerah dan globalisasi.* Jakarta, 17-19 Mei 2004.

Yan-Ling, Z, Dong-Qing Z, Chang-Quan, H, & Bi-Rong D. (2012). Cigarette smoking and its association with serum lipid/lipoprotein among Chinese nonagenarians/centenarians. *Lipids Health Dis, 11*(94) doi: 10.1186/1476-511X-11-94.

Zhou, Q., Wu, J., Tang, J., Wang, J. J, Lu, C. H., & Wang, P. X. (2015). Beneficial effect of higher dietary fiber intake on plasma hdl-c and tc/hdl-c ratio among Chinese rural-to-urban migrant workers. *Int J Environ Res Public Health. 12*(5): 4726-4738.