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IT'S COCOA EXTRACT BENEFIT TO MALE DYSLIPIDEMIA ATHEROGENIC CENTRAL OBESITY?

Nurpudji Astuti Taslim [1], Saiyed Jamaluddin Assaqaf[2], Maria Ingrid Budiman[3], Lusi Makaba[4], Devintha Virani[5], Dewi Lestari [6]

[1, 2, 3, 4] Nutrition Department Faculty of Medicine, Hasanuddin University, Makassar, Indonesia

[5]Nutrition Department Faculty of Public Health, Hasanuddin University, Makassar, Indonesia

[6]School of Economic and Business, Hasanuddin University, Makassar, Indonesia

[1, 6] Center Study for Food Nutrition and Health, Research and Community Hasanuddin University

Corresponding author:Email; pudji_taslim@yahoo.com

Abstract

Background and objective: Obesity a new epidemic throughout the world and impact to economic and health. This study aimed to examine the effect of cocoa supplement on atherogenic dyslipidemia in central obesity male, based on lipid profile, levels of TNF- α, oxidized-LDL and apolipoprotein B.

Methods and design: Double Blind Clinical Trial by recruiting 34 male subjects, at Hasanuddin University hospital Clinic, aged between >25-55 years, waist circumference of >90 were treated for 8 weeks. Intervention Group (n=17) received 4 gram of cocoa extract cap/day, and control group (n=17) received placebo. Both groups had a 15% energy restriction and fat < 25% without any change in activities.

Result: The study showed after 8 weeks, there was no changes on total cholesterol and triglycerides levels in both groups (p>0.05), LDL-C decreased significantly in both groups. There were no changes on TNF- α, ox-LDL and apo-B levels in both groups (p>0,05). HDL-C increased significantly in cocoa group (p=0.04) in atherogenic cocoa group (p=0.04)

Conclusion: cocoa extract supplementation within a hypocaloric and low fat diet increased HDL-C, decreased triglyceride in atherogenic dyslipidemia. No significant changes showed in TNF- α, ox-LDL and apo- B levels in both groups

Key words: central obesity, atherogenic dyslipidemia, cocoa, functional food.

Introduction

Obesity is a condition that affects quality of life. Obesity can be influenced by food intake, lifestyle, social factors, metabolism, neuroendocrine changes, and genetics[1]. Baseline Health Research in 2013 showed national prevalence of central obesity were 26.6%[2]. A 10-year study in Germany showed 16.2% of 24,500 people was in risk to dyslipidemia with prevalence by 24.0%[3]. Research at Universitas Hasanuddin Makassar found the prevalence of central obesity was 30% of 300 new students (from 4000 students) and about 10% with dyslipidemia[4].

Central obesity is marked by accumulation of abdominal fat. Abdominal fat is present in two main compartments, subcutaneous and visceral. The excess of visceral fat is more related to metabolic risk factors and will cause various disorders of lipoprotein metabolism which can lead to atherogenic dyslipidemia characterized by high serum lipid triglycerides, increased Small Dense Low-Density Lipoprotein (sdLDL), decreased High Density Lipoprotein (HDL) and increases of Cytokine production. Adipose tissue is a dynamic endocrine organ that secretes adipokine which contributes to systemic and vascular inflammation. Visceral fat is more active in producing TNF-α compared to subcutaneous adipose tissue. The decrease in fat mass is correlate with a

decrease of proinflammatory cytokines serum concentration. Increase of TNF-α can trigger inflammation which in turn will trigger insulin resistance and endothelial dysfunction, and eventually lead to atherosclerosis[5].

The increase of VLDL and LDL particles describe the increase in apolipoprotein B observed in atherogenic dyslipidemia. Obesity is related to insulin resistance, in this condition LDL is normal or slightly increased, but there is changes in the composition of LDL particles (oxLDL) that caused by hyper triglycerides. The condition of hypercholesterolemia is usually followed by high levels of LDL, which carry around 65-75% of cholesterol from the liver to peripheral tissues, especially oxLDL. The effect of oxLDL can trigger inflammation of the arterial wall through the release of macrophages. LDL metabolism begins with the binding of LDL particles to the specific receptor apolipoprotein B-100 / E, which is located on the cell surface[6,7].

Table 1. Baseline Characteristics of the Study Subjects for Main Variables (n = 34)

Variables	Groups		*P* Value
	Cocoa (n = 17) Mean ± SD	Placebo (n = 17) Mean ± SD	
Ages (year)	42.2 ± 4.7	43.7 ± 4.9	0.756*
BMI (kg/m^2)	28.76 ± 3.33	30.3 ± 3.46	0.102**
Abdominal circumference (cm)	101.1 ± 6.52	101.75 ± 7.54	0.758*
Fasting blood sugar (mg/dl)	97.69 ± 10.41	101.61 ± 12.34	0.352**
Total Cholesterol (mg/dl)	225.93 ± 52.66	231.18 ± 60.14	0.788*
HDL (mg/dl)	40.07 ± 7.60	40.68 ± 8.65	0.828*
LDL (mg/dl)	138.86 ± 30.48	153.36 ± 47.43	0.297*
riglycerides (mg/dl)	282.96 ± 291.88	236.98 ± 210.52	0.718**
TNF-α (ng/L)	168.43 ± 228.40	134.61 ± 92.63	0.692**
oxLDL (ng/ml)	1091.71 ± 2372.79	463.67 ± 445.56	0.480**
Apolipoprotein B (mg/dl)	186,12 ± 201,97	177.06 ± 100.58	0.380**

** Independent t test* *** U Mann-Whitney test*

Black chocolate is a food product with flavonoid content which is one of antioxidants sources, especially the flavan-3ol sub-class (flavanol) and oligomers from flavanol that is epicatechin and catechins and polymerics known as pro anthocyanidin (procyanidin), that can reduce the risk of atherosclerosis. Cocoa polyphenols have a high concentration of flavanol, with beneficial antioxidant effects. In addition, flavonoids are known can suppress inflammation by inhibiting cyclooxygenase-2, an enzyme that regulates during the inflammation process, and some types of tumor formation. Recent evidence also shows that some flavanols can inhibit atherogenesis by interacting with beta-platelet growth factors. Further beneficial effects include a reduction of blood pressure in hypertensive subjects, increased vasodilation of endothelial function, and inhibition of platelet activation and function. In addition, dark chocolate seems can reduce the concentration of C-reactive protein to modulate dyslipidemia anemia, reduce total plasma cholesterol, LDL cholesterol and triglyceride[8,9].

Consumption of 40 grams of cocoa powder for 4 weeks can significantly increase HDL cholesterol[7]. The most potent flavonoid content in cocoa is theobromine, which can suppress TNF-α and IL-6 [10].

Previous research by Monagas et al (2009) showed that flavonoids and theobromine in cocoa had effects on changes of inflammatory mediators, together as antioxidants[19], antiplatelet, and had a positive effect on vascular role in fighting atherogenic dyslipidemia[11]. This study is a continuation of a study by Suprapti et al. (2011), about nonfermented cocoa from Bantaeng District that tested in rats by looking on the effect of changes in blood fat levels compared with simvastatin suspension where the results of animal cholesterol reduction with cocoa were comparable to simvastatin suspension[12].

As well the limitations of previous studies were carried out only in healthy people without obesity or suffering from dyslipidemia in general, not atherogenic dyslipidemia.

In this study, besides assessing lipid profiles we also measured levels of TNF-α, oxidized-LDL and apolipoprotein B by the ELISA method. This is important so that later the benefits of cocoa with flavonoids and alkaloids can be recommended as a supplement for central obese patients with atherogenic dyslipidemia. To our

knowledge, until now there has been no research in Indonesia about the effect of cocoa supplementation on central obese patients with special conditions atherogenic dyslipidemia by looking at the signs of TNF-α, oxidized-LDL and apolipoprotein B. We consider this novel research.

Thus, this study aims to assess the effect of cocoa extract on atherogenic dyslipidemia on central obese male subjects based on changes in lipid profile, levels of TNF-α, oxidized-LDL and apolipoprotein B.

Materials And Methods

Location, research design, and study population

This research was conducted at the Clinical Nutrition Department of Universitas Hasanuddin Hospital in Makassar and networked for 5 months starting from October 2016 - February 2017. The type of this research is a Double Blind Randomized Clinical Trial study. Variable research consists of: independent variables (cocoa), dependent variables (total cholesterol, HDL, LDL, TNF-α, oxidized-LDL and apolipoprotein B), control variables (food intake, activity physical, genetic, gender, race, age). The population of this study were men with central obesity. The sample of this study was male, age> 25-55 years, abdominal circumference ≥ 90cm, which was taken based on consecutive sampling method during the study period, then given capsules of the same color and weight. For subject allocation in the intervention and control groups, block randomization was conducted.

Data collection

All subjects name, age, gender, address, occupation, income, sport or physical activities, telephone / cellphone number and willingness to take part in the study were recorded. Anthropometric data were collected before and after the intervention. In addition, it was also recorded data on food intake obtained using 2 x 24 hours Food Recall method at the beginning, middle, and after the intervention. Laboratory data were collected before and after the intervention. The measurements taken are anthropometric measurements that include Body Height (BH), Body Weight (BW), and Abdominal Circumference (AC). Measurement of Bioelectrical Impedance Analysis (BIA) which includes % fat,% water, muscle mass, physical rating, BMR, metabolic age, bone mass, and visceral fat. Laboratory tests conducted were: fasting blood sugar, total cholesterol, triglycerides, HDL, LDL, sLDL TNF-α. Oxidized LDL, and Apolipoprotein B.

Data analysis

Data collected was processed using statistical analysis using SPSS 20.0.

Population and Samples

202 prospective subjects were screened, among them there were 87 people who fulfilled the inclusion criteria, but only 40 people were willing to take the follow-up examination and signed the informed consent. Of the 40 people, 4 people were excluded after the results of fasting blood sugar examination ≥126 mg / dl were obtained. Furthermore, 36 people were given intervention in the form of giving cocoa capsules or placebo capsules at random. However, there were 2 people who could not continue this research for reasons of busy work and assignments to other cities, so subjects who attended this study to completion was 34 people.

The subjects of this study consisted of 34 central obese men aged 26 years to 54 years who divided each of 17 people into the cocoa capsule group (A) and 17 people into the placebo capsule group (B). All subjects generally experience dyslipidemia.

Results

The results showed that the baseline characteristics of the study subjects in the cocoa group and placebo group were not significantly different (p> 0.05) for the main variables such as age, BMI, Stomach Circumference, fasting blood sugar, total cholesterol, HDL, LDL, triglycerides, TNF-α, oxidized-LDL and apolipoprotein B (Table 1).

The results of HDL and Total Cholesterol Ratios for HDL in the cocoa and placebo groups showed a significant increase in HDL in the cocoa group (p = 0.044) compared to the placebo group (p = 0.193), so did the increase in HDL in the cocoa group compared to placebo (p = 0.012). The same thing can be seen in the Total Cholesterol Ratio of HDL with a significant decrease in the cocoa group (p = 0.002) compared to the placebo group (p = 0.210). The reduction in total cholesterol ratio to HDL was greater in the cocoa group than placebo (p = 0.001) (Table 2).

The results of TNF-α, oxidized-LDL and apolipoprotein B cocoa and placebo groups showed no significant changes in levels of TNF-α, oxidized-LDL and apolipoprotein B before and after capsule administration in the cocoa group and placebo group (p> 0.05) (Tables 3 and 4). Results of HDL and Cholesterol Ratio Total HDL in the cocoa group and placebo in atherogenic dyslipidemia showed a significant increase in HDL in the cocoa group (p = 0.011) compared to the placebo group (p = 0.575), as well as greater HDL increase in the cocoa group compared to placebo (p = 0.028). The same is seen in the Total Cholesterol Ratio of HDL where there was a significant decrease in the cocoa group (p = 0.008) compared to the placebo group (p = 0.575), a decrease in the Total Cholesterol Ratio to HDL in the cocoa group was also greater than the placebo group (p = 0.012) (Table 5).

The results of TG and LDL examinations of the cocoa and placebo groups in atherogenic dyslipidemia showed no significant decrease in TG levels before and after given of capsules in both the cocoa and placebo groups (p> 0.05), but the decrease in triglyceride levels was greater in the cocoa group than placebo (p <0.05). LDL examination showed no significant changes in the cocoa or placebo groups before and after given of capsules (p> 0.05) (Table 6).

The results of examination of TNF-α, oxidized-LDL and apolipoprotein B in the cocoa group and placebo in atherogenic dyslipidemia showed no significant changes in levels of TNF-α, oxidized-LDL and apolipoprotein B before and after capsule being given in the cocoa and placebo groups (p> 0, 05) (Table 7 and Table 8).

Discussion

This study showed that there were no significant changes in total cholesterol and triglycerides before and after capsule administration in both groups that have dyslipidemia (p> 0.05), so there was no additional benefit of cocoa supplementation for that parameters. Whereas significant improvement with cocoa supplementation was obtained for HDL (p = 0.012) compared to placebo and Total Cholesterol ratio to HDL (p = 0.001) compared to placebo. This means that supplementation of cocoa extract is beneficial to increase HDL levels and reduce the Total Cholesterol ratio to HDL for prevention of cardiovascular disease. For LDL, there was a significant decrease in both the cocoa group (p = 0.003) and placebo (p = 0.004) before and after administration of capsules, but there was no significant difference of the decline between them (p = 0.380) so that there was no additional benefit supplementation of cocoa extract against LDL levels. This similar benefit on Newly TB patient by giving cacao drink to Nutrtional Status, gamma Intervention, itamin D and calcium [17,18].

Some of the previous studies were in line with the results of this study and some were different. It was reported that cocoa supplementation can increase HDL and reduce LDL[9]. A meta-analysis reported that HDL levels increased in the long term with cocoa supplementation accompanied by low fat consumption, while LDL levels and total cholesterol decreased in the short term in subjects less than 50 years old. The meta-analysis also stated that there were no significant changes in triglycerides, total cholesterol and blood sugar in line with the results of this study[13]. Other reports stated that consumption of 850 mg of theobromine in cocoa significantly increased HDL but in the study, this was not controlled for physical activity and diet during intervention[14].

For TNF-α, there was no significant change after getting a cocoa capsule for 8 weeks compared to placebo (p> 0.05), this is similar to the results of the Muniyappa et al[15] study, for 2 weeks with a larger dose of cocoa powder in patients hypertension and research Renzo et al[16], in female obese patients. This result is different from the results of the previous Ibero-Baraibar I et al (2013) study which found a significant change in oxLDL after being given cocoa supplementation for 4 weeks in male and female subjects with BMI around 30 kg / m^2. In this study, there was no significant change in the Apolipoprotein B biomarkers after being given cocoa

capsules (p> 0.05). The results for Apolipoprotein B are the same as the results of previous studies by Khan N et al (2011) which found no significant changes in Apolipoprotein A1 and B, as well as lipoprotein concentrations.

In this study, there were no significant changes of fasting blood sugar and total cholesterol with cocoa supplementation in atherogenic subjects (p> 0.05), this is similar to that obtained in dyslipidemia in general. Whereas for HDL and cholesterol ratio for HDL, there were significant changes in atherogenic subjects as well as in general dyslipidemia (p <0.05).

What is different is that atherogenic subjects found a significant decrease in triglycerides compared to placebo (p<0.05), while in dyslipidemia there was generally no significant change compared to placebo (p>0.05). For LDL, in atherogenic subjects there were no significant changes before and after cocoa supplementation (p> 0.05), whereas in dyslipidemia in general there were significant results (p<0.05). But if each is compared to placebo, the magnitude of the change is not significantly different (p>0.05). For ELISA examination, there were no significant changes in levels of TNF-α, oxidized-LDL and apolipoprotein B in atherogenic subjects given cocoa supplementation (p> 0.05). This is similar to that found in dyslipidemia in general.

The results of this study can be influenced by several things as differences in the baseline characteristics of the subjects in general dyslipidemia and atherogenic dyslipidemia, but generally there were no significant differences in baseline data between the cocoa group and the placebo group as seen in Table 1.

Another important thing is intake. Intake influence in each subjects viewed from Food Recall 2 x 24 hours at the beginning of the study (baseline), mid (week IV) and the end of the study (week VIII). There was no significant difference in the amount of intake between the cocoa group and the placebo group in dyslipidemia and dyslipidemia atherogenic (p> 0, 05) (Table 9 and Table 11). This means that there is no difference in the amount of food intake among the groups studied which can affect the results of this study. While the macronutrient composition was obtained, the percentage of carbohydrates did not differ significantly between the cocoa group and the placebo group, both in general dyslipidemia and dyslipidemia atherogenic (p> 0.05) (Table 10 and Table 12).

Likewise, with the percentage of protein and fat percentage the same results were obtained (p> 0.05). From the Food Recall 2 x 24 hours it also known that there was a significant decrease in the amount of intake in the cocoa group and placebo group compared to the amount of intake at the beginning of the study, both in general dyslipidemia and dyslipidemia (p <0.05). This means that 15% calorie restriction compliance from calorie needs based on the Harris-Benedict formula is relatively the same in the groups studied while showing the importance of calorie restriction in handling central obesity with dyslipidemia atherogenic. As for the macronutrient composition, from Food Recall 2 x 24 hours it was found that there was no significant difference in the composition of carbohydrate, protein, and fat in each group studied when compared to the macronutrient composition at the start of the study (p> 0.05), except the percentage fat which actually increases significantly in dyslipidemia in general (p <0.05), but this result is still in accordance with the target fat is limited to <25% of total calories.

Subject compliance in consuming capsules according to the instructions which is given. In this study, if the subject's compliance to consuming capsules for 8 weeks less than 80% would be considered to drop out. Thus, the influence of different compliance among the groups studied in consuming capsules especially those containing cocoa can be minimized. But also keep in mind, that one of the weaknesses in this study is that compliance is measured only based on history because of limited funds. There is no marker of dietary compliance (eg: phase 2 metabolic syndromes and / or total epicatechin or total flavanols), as obtained in previous studies on cocoa[9].

Effect of changes in subject activities during the study. Although all research subjects were advised not to change activities for 8 weeks underwent research, but because the study time was relatively long, there were 5 subjects who experienced changes in activities due to work / hobbies, Umrah, or because of illness. This certainly can affect the results of this study.

Conclusions And Recommendations

Researchers concluded that cocoa supplementation in central obesity with low diet of calories and fat was limited to increasing HDL levels in dyslipidemia generally. Cocoa supplementation in central obesity with a limited calories and fat diet increased HDL levels in atherogenic dyslipidemia. Cocoa supplementation in central obesity with a low calories and fat diet limited to reduction in triglyceride levels is greater in atherogenic dyslipidemia than placebo. Cocoa supplementation in central obesity with a low calories and limited fat diet does not affect TNF-α levels, oxidized-LDL, and apolipoprotein B both in general dyslipidemia and dyslipidemia atherogenic. The researcher suggested that further research with larger samples be carried out especially for atherogenic subjects with a larger dose of cocoa supplementation. Need to examine several other biomarkers including compliance biomarker to find out more about the benefits of cocoa for cardiovascular disease.

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Table 2. Result of HDL and Total Cholesterol Ratios for HDL in Cocoa and Placebo Groups

Capsules		HDL			*P* value	Total Cholesterol Ratios for HDL			*P* value
		Sb	Ss	Delta		Sb	Ss	Delta	
A	Mean	40.07	43.77	3.70	0.044****	5.73	5.06	0.67	0.002****
	SD	7.60	6.41	6.88		1.48	1.32	0.73	
B	Mean	40.68	38.46	-2.22	0.193****	5.90	5.81	0.09	0.210****
	SD	8.65	5.26	6.07		2.16	1.45	1.31	
P value		0.828*	0.011**	0.012*		0.986**	0.127*	0.001**	

Explanation: A = Cocoa; B = Placebo; HDL = *High Density Lipoprotein*; Sb = Before; Ss = After;
* = *independent t-test*; ** = *Mann-Whitney test*; **** = *Wilcoxon test*.

Table 3. Result of TNF-α and oxLDL in Cocoa and Placebo Groups

Capsules		TNF-α			*P* value	oxLDL			*P* value
		Sb	Ss	Delta		Sb	Ss	Delta	
A	Mean	168.43	126.92	41.52	0.309****	1091.71	932.02	159.69	0.943****
	SD	228.40	148.10	187.91		2372.79	2117.05	2837.13	
B	Mean	134.61	111.86	22.75	0.177****	463.67	429.37	34.29	0.795****
	SD	92.63	121.85	116.85		445.56	322.70	258.84	
P value		0.692**	0.196**	0.593**		0.480**	0.380**	0.877**	

Explanation: A = Cocoa; B = placebo; TNF-α= *Tumour Necrosis Factor Alpha; oxLDL = Oxidized Low Density Lipoprotein*; Sb = before; Ss = after; ** = *Mann-Whitney test*; **** = *Wilcoxon test*

Table 5. Result of HDL and Cholesterol Ratio of Cocoa and Placebo Groups in Atherogenic Dyslipidemia

Groups		HDL			*P* value	Cholesterol Ratio for HDL			*P* value
		Sb	Ss	Delta		Sb	Ss	Delta	
Capsule A Atherogenic	Mean	34.74	41.68	6.93	0.011****	6.32	5.27	1.05	0.008****
	SD	2.91	4.81	5.35		1.73	1.45	0.67	
Capsule B Atherogenic	Mean	33.63	34.44	0.81	0.575****	6.55	6.10	0.45	0.575****
	SD	5.05	2.37	4.99		2.81	1.34	1.82	
P value		0.847**	0.002*	0.028*		0.564**	0.245*	0.012**	

Explanation: A = Cacao; B = Placebo; HDL = *High Density Lipoprotein*; Sb = Before; Ss = After;
* = *independent t-test*; ** = *Mann-Whitney test*; **** = *Wilcoxon test*.
Sb = before; Ss = after; ** = *Mann-Whitney test*; **** = *Wilcoxon test*

Table 6. Result of TG and LDL of Cocoa and Placebo Groups in Atherogenic Dyslipidemia

Groups		TG			*P* value	LDL			*P* value
		Sb	Ss	Delta		Sb	Ss	Delta	
Capsule A	Mean	385.61	264.21	121.40	0.086****	125.16	118.06	7.10	0.189***

Atherogenic	SD	361.37	172.55	214.30		26.46	26.15	14.82	
Capsule B	Mean	291.60	257.89	33.71	0.401****	131.88	120.91	10.96	0.221***
Atherogenic	SD	293.13	163.55	170.98		37.75	34.70	23.05	
P value		0.124**	0.630**	0.043**		0.674*	0.849*	0.386**	

Explanation: A = Cacao; B = Placebo; TG = Trigliserida; LDL = *Low Density Lipoprotein*; Sb = Before; Ss = After

Table 7. Result of TNF-α and oxLDL of Cocoa and Placebo Groups in Atherogenic Dyslipidemia

Groups		TNF-α			*P* value	oxLDL			*P* value
		Sb	Ss	Delta		Sb	Ss	Delta	
Capsule A	Mean	226.97	120.67	106.30	0.515****	1774.23	718.53	1055.70	0.767****
Atherogenic	SD	301.98	98.67	216.95		3173.54	1114.12	2823.25	
Capsule B	Mean	129.78	115.34	14.44	0.263****	389.64	358.69	30.95	0.484****
Atherogenic	SD	68.13	141.28	97.89		229.08	264.38	129.28	
P value		0.630**	0.211**	0.700**		0.500**	0.501**	0.336**	

Explanation: A = Cacao; B = Placebo; TNF-α= *Tumour Necrosis Factor Alpha*; oxLDL = Oxidized *Low Density Lipoprotein*; Sb = Before; Ss = After; * = *independent t-test*; ** = *Mann-Whitney test*; **** = *Wilcoxon test*.

Table 8. Result of Apolipoprotein B in Cocoa and Placebo Groups in Dyslipidemia Atherogenic

Capsules		Apo B			*P* value
		Sb	Ss	Delta	
Capsule A Atherogenic	Mean	272.44	221.56	50.89	0.515****
	SD	249.28	130.01	155.01	
Capsule B Atherogenic	Mean	179.25	184.25	-5.00	0.779****
	SD	51.33	115.98	84.04	
P value		0.847**	0.544*	0.630**	

Explanation: A = Cocoa; B = placebo; Apo B = Apoliprotein B; Sb = before; Ss = after; ** = *Mann-Whitney test*; **** = *Wilcoxon test*

Tabel 9. Result of Food Recall 2 x 24 hours in the beginning, middle, and the end of research in all dyslipidemia subjects

Capsules		FR 2 x 24 jam (kcal)				*P value*
		Beginning	IV weeks	VIII weeks	Delta Beginning – VIII weeks	
A	Mean	2021.82	1901.58	1780.42	241.40	0.004[c]
	SD	359.24	339.48	470.53	387.87	
B	Mean	2012.20	1880.74	1775.91	236.29	0.002[c]
	SD	609.54	439.47	428.33	393.68	
P value		0.956*	0.878*	0.877**	0.970*	

Explanation: A = cocoa; B = placebo; FR = *Food Recall*; * = *Independent t-test*; ** = *Mann-Whitney test;* [C] = *Friedman test*

Tabel 10. Result of Diet Composition in the beginning, middle, and the end of research in all Dyslipidemia Subjects

Capsules		KH (%)			*P* value	Protein (%)			*P* value	Fat (%)			*P* value
		Beginning	IV weeks	VIII weeks		Beginning	IV weeks	VIII weeks		Beginning	IV weeks	VIII weeks	
A	Mean	69.24	66.79	63.64	0.161[c]	15.36	15.66	15.15	0.675[D]	15.40	17.55	21.21	0.047[c]
	SD	8.35	8.70	10.58		2.58	2.36	2.71		6.37	7.04	10.02	
B	Mean	70.56	69.89	69.26	0.790[c]	15.40	15.91	15.09	0.389[D]	14.31	14.20	15.65	0.204[c]
	SD	6.87	5.17	7.39		2.50	2.17	2.35		5.99	5.07	7.16	
P value		0.616*	0.217*	0.088**		0.968*	0.747*	0.938*		0.609*	0.122*	0.095**	

Explanation: A = cocoa; B = placebo; KH: Carbohidrat; * = *Independent t-test*; ** = *Mann-Whitney test;* [C] = *Friedman test*; [D] = *Repeated ANOVA test*

Tabel 11. Result of Food Recall 2 x 24 hours in the beginning, middle, and the end of research in dyslipidemia atherogenic subjects

Capsules		FR 2 x 24 (kcal)				*P value*
		Beginning	IV weeks	VIII weeks	Delta Beginning – VIII weeks	
Capsule A Atherogenic	Mean	1918.47	1764.53	1574.70	343.76	0.013[c]
	SD	382.02	340.36	507.58	406.92	
Capsule B Atherogenic	Mean	2254.34	2054.21	1843.22	411.13	0.011[c]
	SD	522.14	331.31	376.56	488.01	
P value		0.148*	0.096*	0.102**	0.761*	

Explanation: A = cocoa; B = placebo; FR = *Food Recall*; * = *Independent t-test*; ** = *Mann-Whitney test;* [C] = *Friedman test*

Tabel 12. Result of Diet Composition in the beginning, middle, and the end of research in Dyslipidemia Atherogenic Subjects

Capsules		KH (%)			*P* value	Protein (%)			*P* value	Fat (%)			*P* value
		Beginning	IV weeks	VIII weeks		Beginning	IV weeks	VIII weeks		Beginning	IV weeks	VIII weeks	
Capsule A Atherogenic	Mean	70.29	69.79	68.31	0.543[D]	14.71	15.04	14.57	0.700[D]	15.00	15.17	17.14	0.457[D]
	SD	9.68	7.42	6.19		2.76	2.16	2.37		7.46	5.75	5.56	
Capsule B Atherogenic	Mean	70.15	68.68	68.10	0.119[D]	14.98	15.55	14.68	0.635[D]	14.88	15.78	17.23	0.145[D]
	SD	5.84	3.23	5.22		2.42	1.31	2.32		5.23	3.61	6.50	
P value		0.972*	0.701*	0.942*		0.838*	0.574*	0.926*		0.969*	0.801*	0.979*	

Explanation: A = cocoa; B = placebo; KH: Carbohidrat; * = *Independent t-test*; ** = *Mann-Whitney test;* [C] = *Friedman test*; [D] = *Repeated ANOVA test*