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The effect of flexitarian diets on high-densit- lipoprotein (HDL) serum in obese female students

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ABSTRACT

Background: Obesity in female students affects metabolic profiles and reproduction. Consuming a plant-based diet can inhibit the synthesis of fatty acids and cholesterol, thereby reducing the risk of obesity. The lipid profile plays a crucial role in metabolism and includes high-density lipoprotein (HDL), which has a protective effect against cardiovascular disorders, obesity, hypertension, and dyslipidemia.
Objective: To analyze the effect of flexitarian diets on changes in serum HDL levels in obese female students.
Methods: A quasi-experimental design with pre-post-test control group was employed. Twenty-two female students aged 19-25 years were selected using consecutive sampling. The subjects were divided into intervention and control groups. The intervention group received a flexitarian diet for 4 weeks consisting of three meals and two snacks totaling 1500 calories per day. Both groups were provided with educational leaflets about obesity and balanced nutrition. Food intake was assessed using a 3x24-hour food recall, while physical activity was measured using the IPAQ-SF questionnaire. Serum HDL levels were analyzed enzymatically before and after the intervention. Statistical analysis was conducted using SPSS 25, including the Shapiro-Wilk test for normality, paired t-tests, independent t-tests, and the Mann-Whitney test.
Results: There was a significant decrease in serum HDL levels in the intervention group (2.00 ± 2.83 mg/dL), while serum HDL levels in the control group increased (3.27 ± 8.25 mg/dL), although this increase was not statistically significant. There was no significant difference between the two groups in terms of the mean change in HDL levels ($p > 0.05$).
Conclusion: A flexitarian diet for 4 weeks can lead to a significant reduction in serum HDL levels.

Keywords: *Flexitarian Diets; Female Students; Serum HDL; Obesity*

BACKGROUND

Obesity is a topic of a global problem until now it continues to increase every year reaching 1,9 billion and even more than 650 million people in the world according to WHO.[1] The prevalence of overweight with indicators of BMI 25 to <27 kg/m2 was 11,2% (Riskesdas 2013) increasing to 13,6% (Riskesdas 2018).2 While the prevalence of obesity BMI 27 kg/m 2 is 14,8% (Riskesdas 2013), an increase of 21,8% (Riskesdas 2018).[2] It is estimated that 38% of the world's population will be overweight and 20% will be obese in 2030.[3] The prevalence of obesity based on the 2013 Riskesdas for women was 42.1% higher than 11.3% for men. Whereas in Southeast Asia, the prevalence of obesity in women is 8-52%, in men 8-30%.[4] Obesity profile in Central Java is 6,32%, Semarang City is 2,66% with a higher percentage of women than men, and belongs to the productive age group.[5]

Obesity, especially in women, has doubled, this is influenced by physical activity patterns, energy imbalances, and the accumulation of fat distribution in women is higher than in men.[6,7] In addition, there is an increase in hormones and the distribution of peripheral fat to the abdomen. Women of childbearing age (WUS) aged 18-25 years are at risk for obesity.[8] Influenced by the consumption of fast food high in energy and fat, low physical activity, psychological, age, socioeconomic status, imbalanced diet, environment, and genetics of a person.[9] The choice of food intake high in carbohydrates, protein, fat, low in fiber, triggers an increase in triglyceride levels and a decrease in High-Density Lipoprotein (HDL) levels so that it affects a person's lipid metabolism.[10] Obese women have a 1,89 times risk of experiencing reproductive disorders.[8] These disorders include menstruation, pregnancy disorders because of the potential for preeclampsia.[11,12] Long-term impact, obesity in productive women can cause reproductive disorders such as subfertility.[13] College students are a group of women of childbearing age who are at risk of obesity due to the influence of lifestyle.[13]

Obese who have low HDL levels have a risk of 1,12 times compared to non-obese.[14] Several components of the obesity disorder can be at risk of developing metabolic syndrome, with at least three markers of several risks, such as type 2 diabetes mellitus, cardiovascular disease, dyslipidemia, glucose intolerance,

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and hypertension.[15] Metabolic syndrome has become an epidemic problem where it affects 12-37% of the Asian population and 12-26% of the European population.[16] The incidence rate in Indonesia is 39% at the age of 45-65 years, especially in women with a prevalence of metabolic syndrome 46% and 28% in men.16 Women are at risk for abdominal obesity, low HDL levels, and hypertension.[6,8,16]

HDL is a cholesterol transporting lipoprotein in lipid metabolism, rich in antioxidants and anti-inflammatory against the incidence of obesity.[17] The protective effect of HDL can reduce the risk of cardiovascular disease, obesity, hypertension, and dyslipidemia.17 Low HDL levels occur when levels are 50 mg/dL for women, ≤40 mg/dL for men.[18] HDL plays a role in the incidence of dyslipidemia where the lipid profile contains triglyceride levels, Very Low-Density Lipoprotein (VLDL), Intermediate Density Lipoprotein (IDL), Low-Density Lipoprotein (LDL) is high and HDL is low.[19] The higher the classification of a person's obesity, the lower the HDL cholesterol level. For every 1 kg/m^2 increase, HDL decreased by 0,8 mg/dL.[20]

Based on the problems above, there is a need for interventions aimed at reducing these risks. One effort that can be done is to regulate food intake through diet. The diet trend that is easy to apply is the flexitarian or semi-vegetarian diet. This diet includes the type of vegetarian that is flexible because it can still consume animal protein.[21] The flexitarian diet was created by Dawn Jackson Blatner from the United States in 2003, effectively losing 15% of body weight, reducing the risk of diabetes and metabolic syndrome.[22] Research a plant-based diet for 4 weeks can significantly reduce HDL levels by 4.3 mg/dL.[24] The Research with similar explain there was a decrease in HDL levels by 3.6 mg/dL.[25]

Other research explain that the subjects who were given a plant-based diet for 7 months, triglycerides decreased from 171 mg/dL to 103 mg/dL, and HDL levels could increase significantly from 48,3 mg/dL to 54.6 mg/dL.[26] Based on research referring to the American Heart Association (AHA), there is a lifestyle change in the form of providing a diet with functional foods and reducing energy (-500 kcal), on days 45 to 75 there is a significant increase in HDL levels by 8.6%.[20]

The flexitarian diet is effective because it reduces the consumption of red meat, which is potentially carcinogenic and high in calories.[27] The benefits of a plant-based diet on weight loss have reduced the world's mortality rate by up to 23%.28 High fiber content and low in saturated fat can lower total cholesterol and increase HDL.[29] The content of phenolic acids, unsaturated fatty acids, and fiber can inhibit the synthesis of fatty acids and cholesterol by increasing bile salts thereby inhibiting HMG CoA reductase activity, reducing the breakdown of ApoA-1, and increasing HDL levels.[30] Fiber intake of 18 g/day – 30 g/day can increase HDL cholesterol by 10,1% and triglyceride and LDL cholesterol levels decrease by 14,4% in men and 11,1% in women.[31] Research related to the provision of a flexitarian or semi-vegetarian diet is still not widely carried out in Indonesia. This has sparked interest among researchers and serves as a reference for studies aiming to analyze the effect of implementing a flexitarian diet on changes in HDL levels in obese female students.

MATERIALS AND METHODS

Types of Research

This research falls within the scope of community nutrition and employs a quasi-experimental design in the form of a pre-post test control group setup. The study was conducted in the City of Semarang and the CITO Setiabudi Lab in Semarang from July to September 2021. Ethical clearance for this research was obtained from the Medical/Health Research Bioethics Commission, Faculty of Medicine, Sultan Agung Islamic University, Semarang (Approval No. 328/IX/2021/Commission on Bioethics). Given the ongoing Covid-19 pandemic, strict health protocols were implemented throughout the research process. Researchers ensured adherence to these protocols by administering Covid-19 vaccines and rapid antigen tests before conducting screenings, collecting lab data with assistance from CITO laboratory officers, and maintaining proper spatial arrangements, including regular disinfection spraying, with a minimum distance of 1 meter between individuals.

Sampling and Techniques

A total of 22 subjects were enrolled in this study, determined using the formula for hypothesis testing on the mean of two independent populations. Considering a requirement of 10 subjects per group plus an estimated 10% dropout rate, 11 subjects were allocated to each group. The study comprised two groups: the flexitarian diet intervention group and the control group. Subject data were collected using a consecutive sampling method.

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Inclusion criteria consisted of female students aged 19-25 years with a BMI of 25 kg/m^2, HDL levels of 50 mg/dL, residing in Semarang during the study period, absence of physical or specific diseases (such as diabetes mellitus, kidney disorders, cardiovascular disease, or infections), no adherence to a specific diet in the past three months (e.g., diabetes diet, DASH diet, low-salt diet, vegan diet, etc.), non-smoking status, not breastfeeding or pregnant, not taking specific medications for at least the last three months (e.g., cholesterol-lowering drugs such as statins, niacin, gemfibrozil, etc.), and willingness to participate in the research by providing informed consent. Exclusion criteria included resignation, illness, or death during the research process, non-adherence to the intervention (failure to meet <80% of diet days, or unwillingness to provide blood samples).

The subjects were then randomly assigned to either the treatment or control group. The treatment group received the intervention in the form of a flexitarian diet, while the control group did not undergo dietary intervention. However, both groups received nutritional education on obesity management and balanced nutrition. Education was provided through Zoom Meetings, and leaflets related to obesity management with balanced nutrition were distributed.

Data Collection and Measurement

The independent variable in this study is a flexitarian diet, while the dependent variable is HDL levels. The confounding variable is physical activity, and there are intermediate variables in the form of energy intake, carbohydrates, protein, fat, and fiber. Subject data taken were anthropometry, nutrient intake (energy, carbohydrates, protein, fat, fiber, cholesterol, vitamin C, PUFA, MUFA, and SFA), physical activity, and HDL levels measured before and after the intervention. Data on subject characteristics, food intake, and physical activity were collected through interviews using a questionnaire. Weighing using a scale of Tanita brand BC-730 Body Compact Composition Monitor with an accuracy of 0.1 kg and a capacity of 150 kg. Height was measured by microtoise brand GEA number 26SM with an accuracy of 0.1 cm and a capacity of 200 cm. Subjects were categorized as obese if their body mass index (BMI) was 25 kg/m 2.32 HDL levels were measured by laboratory staff at CITO Setiabudi Semarang with the enzymatic method taken through a vein and the subject had previously fasted for 8-10 hours. Subjects who are obese on average are followed by low HDL levels of 50 mg/dL and if normal >50 mg/dL.[33]

The flexitarian diet is given for 4 weeks in stages in the form of flexitarian beginner-advanced – expert. The 1st week was given a beginner stage flexitarian diet in the form of 2 days without meat/poultry consumption, a minimum of 737 grams of meat consumption/week. Weeks 2 and 3 of the advanced flexitarian diet in the form of 3 days without meat/poultry consumption, at least 510 grams of meat consumption/week. Then in the 4th week of the expert stage flexitarian diet in the form of 5 days without meat/poultry consumption, a minimum of 255 grams of meat consumption/week.[22] Subjects calculated the average need for energy, carbohydrates, protein, and fat using the Mifflin-St. Jeor calculation and then reduced by 500 kcal. So that the number of calories given is ±1500 kcal/day with details of 300 kcal for breakfast, 400 kcal for lunch, 500 kcal for dinner, and an interlude of 2 x 150 kcal. The composition of the flexitarian diet consists of carbohydrates as much as 50-57% (187.5 – 213.8 grams), protein 13-16% (48.75-60 grams), fat 25-35% (41.6-58.3 grams), fiber 25-35 grams, cholesterol <200 mg per day.[34]

The diet regimen consisted of three main meals and two snacks per day, provided for a duration of 4 weeks. Food distribution was conducted via researcher home visits from Monday to Friday, while on Saturday and Sunday, subjects were responsible for their own meal preparation, monitored by the researcher. Compliance with the flexitarian diet was monitored daily through WhatsApp and Google Forms, which included questions about diet adherence and required subjects to upload photos of any leftovers. Subjects were provided with a flexitarian booklet guide in both soft and printed formats to serve as a reference for consuming a plant-based diet, facilitating comprehension and application. Additionally, subjects received motivational support for adhering to the diet and could seek consultation via a WhatsApp Group regarding independent meal provision or lapses in adherence.

Data on nutrient intake was obtained from interviews with food and beverage consumption during the past month through the SQ-FFQ to determine the subject's food consumption habits before the intervention.35 Meanwhile, intake during the intervention of the two groups was recorded using the 3x24 hour food recall method (2 weekdays and 1 weekend) on Wednesday, Friday, and Sunday for 4 weeks.35 SQ-FFQ food intake interviews and 3x24 hour food recalls were conducted online via WhatsApp and used food book photo references. Physical activity data were recorded using the International Physical Activity Questionnaire Short Form (IPAQ-SF) questionnaire via a google form. This questionnaire contains details of all activities related

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to the type, frequency, and duration of physical activity within the last 7 days for 4 weeks, analyzed in MET-minutes/week units. Physical activity scores were then categorized as mild (<600 MET-minutes/week), moderate (600-2999 MET-minutes/week), and severe (≥3000 MET-minutes/week).[36]

Data Analysis

The data were processed and analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0 for Windows. Normality of the data was assessed using the Shapiro-Wilk test due to the small sample size (≤50 subjects). HDL levels before and after the intervention were compared using the paired t-test. Differences in HDL level changes (ΔHDL) between the treatment and control groups were assessed using the independent t-test. Disparities in energy, carbohydrate, fat, fiber, PUFA, MUFA, and SFA intake between the treatment and control groups were evaluated with the independent t-test, while discrepancies in protein, cholesterol, and vitamin C intake were examined using the Mann-Whitney test. Physical activity differences between the two groups were analyzed with the independent t-test. A p-value <0.05 was considered indicative of a significant effect.

RESULTS

Characteristic of Research Subjects

A total of 22 research subjects were divided into two groups, namely the treatment and control groups. The characteristics of the initial subjects in the study, including age, weight, height, nutritional status, physical activity, energy intake, protein, fat, carbohydrates, fiber, cholesterol, vitamin C, and HDL levels, are presented in table 1.

Table 1. Characteristic of Research Subjects

Characteristic	Intervention Mean ± SD Median (Min-Max)1	Control Mean ± SD Median (Min-Max)1	p
Age (years)	22,6 ± 1,2	22,3 ± 1,4	0,525a
Weight (kg)	73,8 (65 – 116,5)*	78,9 (54,7 – 101,8)*	0,768b
Height (cm)	155,7 ± 3,4	153,9 ± 6,6	0,446a
IMT (kg/m2)	33,3 ± 7,4	32,0 ± 4,2	0,615a
Physical Activity (MET-min/week)	1201 ± 731,0	2216 ± 1433,7	0,054a
Nutrients Intake			
Energy (kkal)	2559 ± 460,6	2779 ± 599,7	0,347a
Protein (gram)	89,3 ± 24,9	78,4 ± 26,6	0,336a
Fat (gram)	111,9 ± 34,5	122,3 ± 38,8	0,517a
Carbohydrates (gram)	289,1 (241,7 – 414,1)*	346,3 (186,8 – 532,4)*	0,818b
Fiber (gram)	10,2 (7,5 – 26,6)*	15,1 (4,7 – 27,7)*	0,533b
Cholesterol (mg)	271,7 (145,8 – 1198,2)*	278,9 (127,4 – 801,4)*	0,718b
Vitamin C (mg)	37,7 (6,9 – 517,1)*	85,3 (10,5 – 673,4)*	0,412b
PUFA (gram)	26,1 ± 13,6	18,0 ± 9,6	0,752a
MUFA (gram)	18,8 ± 6,2	20,3 ± 6,7	0,549a
SFA (gram)	39,8 ± 15,3	44,5 ± 13,5	0,454a
HDL pre-intervention (mg/dL)	42,4 ± 4,1	41,8 ± 6,8	0,822a

[a]Independent T-Test [b]Mann-Whitney Test

[1]Data with abnormal distribution is presented in Median (Min-Max)

HDL Levels Before and After Intervention

The differences in HDL levels before and after the 4-week intervention are presented as follows: a significant difference was observed in the treatment group (p = 0.041), while no significant difference was found in the control group.

Table 2. HDL Levels Before and After Intervention

Group	HDL Pre (mg/dL) Mean ± SD	HDL Post (mg/dL) Mean ± SD	p
Intervention	42,4 ± 4,1	41,8 ± 6,8	0,041a
Control	40,4 ± 5,5	45,1 ± 5,1	0,218a

[a]Uji Paired T-test

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Differences in Physical Activity, Nutrient Intake During the Intervention and Changes in HDL Levels

The table also indicates whether significant differences were observed in physical activity and intake (energy, protein, fat, carbohydrate, fiber, cholesterol, vitamin C, PUFA, MUFA, SFA), as well as changes in HDL levels, between the treatment and control groups.

Table 3. Differences in Physical Activity, Nutrient Intake During the Intervention and Changes in HDL Levels

Characteristic	Intervention Mean ± SD Median (Min-Max)*	Control Mean ± SD Median (Min-Max)*	p
Physical Activity (MET-min/week)	789 ± 406,1	1445 ± 503,6	0,003a
Intake			
Energy (kkal)	1271 ± 88,7	1853 ± 712,2	0,022a
Protein (gram)	53,0 (44,0 – 60,14)*	57,1 (35,2 – 100,8)*	0,250b
Fat (gram)	40,6 ± 5,6	76,7 ± 41,2	0,016a
Carbohydrates (gram)	190,7 ± 12,2	231,5 ± 84,7	0,143a
Serat (gram)	13,5 ± 1,6	9,8 ± 5,8	0,067a
Kolesterol (mg)	73,4 (39,6 – 170,9)*	255,9 (151,1 – 543,9)*	<0,001b
Vitamin C (mg)	96,5 (75,1 – 126,5)*	45,4 (8,97 – 237,6)*	0,001b
PUFA (gram)	9,5 ± 1,5	14,8 ± 8,6	0,071a
MUFA (gram)	5,9 ± 1,5	14,4 ± 7,3	0,003a
SFA (gram)	10,7 ± 25,2	16,0 ± 13,6	0,006a
Δ HDL (mg/dL)	-2,0 ± 2,8	3,27 ± 8,2	0,067a

[a]Independent T-test [b]Mann-Whitney Test

*Data with abnormal distribution is presented in Median (Min-Max)

Table 4. Percentage of Changes in HDL Levels in the Treatment and Control Groups

HDL Levels	Intervention	Control
Increase	18,2%	45,5%
Decrease	81,8%	54,5%

Table 4 shows that when looking at the increase or decrease in HDL levels of subjects after being given the flexitarian diet intervention, it is indeed found that more subjects in the intervention group experienced an decrease in HDL levels (81,8%) compared to subjects in the control group (54,5%).

DISCUSSION

The results of this study showed a decrease in HDL levels of 2 mg/dL in the treatment group before and after the intervention, which was statistically significant, indicated by the p-value = 0.041. However, in the control group, there was an increase in HDL levels of 3.27 mg/dL which was not significant. These findings indicate that giving a plant-based diet in the form of a flexitarian diet can reduce a person's HDL levels.

In line with other studies explain that consumption of a plant-based diet without consumption of animal products for 4 weeks and did not change the pattern of physical activity, was able to significantly reduce HDL levels by 5,2 mg/dL.[24] There is a study that states that a vegetarian diet for 4 weeks can significantly reduce HDL levels by 3,6 mg/dL in the treatment group and 3.4 mg/dL in the control group.[37] Research with similar explain that a plant-based diet for 8 weeks can significantly reduce HDL levels by 7.3 mg/dL.[38] Based on the theory, this plant-based diet is appropriate that a low-fat plant-based diet can reduce HDL levels, but the cardiovascular risk improves even though HDL levels decrease. So there is a tendency for low serum triglycerides and LDL. The HDL parameter alone cannot predict cardiovascular risk, it needs to be completed using the TG/HDL ratio. HDL's anti-inflammatory properties and its ability to protect LDL from oxidation play an important role in Reverse Cholesterol Transport (RCT).[23]

Still, in line with plant-based diet research, giving a plant-based lifestyle intervention for 30 days to 4654 subjects given a high-fiber and low-fat diet with an emphasis on consumption of whole grains in the form of grains, nuts, fruits, vegetables, consuming 2-2,5 L of water. Daily, limiting sugar, salt, and fat as well as doing physical activity with an intensity of 30 minutes every day and managing stress, can reduce HDL levels by 4.77 mg/dL or 8.7% significantly.[23] The study, is similar to the principle of a “balanced nutrition” diet. However, in this study, this principle has not been fully implemented, from the flexitarian diet intervention given there are still food residues such as vegetables, vegetable side dishes, and fruits. In addition, the level physical activity is still low so it has not reached the guidelines of the principle of "balanced nutrition".

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The subject spends most of the time in sedentary life style activities, such as sitting, lying down, sleeping. This decrease in HDL levels is correlated with the principle of plant-based diet intervention, where there is a decrease in apolipoprotein-A production, thereby reducing HDL concentrations. A vegetarian diet low in cholesterol, total fat, and saturated fatty acids can reduce the absorption and conversion of blood cholesterol. In addition, in terms of the phytochemical content in vegetables, fruits, nuts, seeds can improve health through several mechanisms. Phytosterols can reduce cholesterol absorption in the intestine, phenolics can inhibit LDL oxidation, flavonoids can inhibit cholesterol micelles, and sulfides reduce lipid profiles, namely triglycerides, non-HDL, and LDL by inhibiting cholesterol biosynthesis.[39]

Guidelines for a plant-based diet using references from the American Heart Association (AHA) were also carried out proved that a diet with functional foods and a reduction in energy of 500 kcal can significantly increase HDL levels by 8.6% in women.[20] This study had similar results in increasing HDL levels in the control group, which experienced an increase of 3,27 mg/dL possibly because the choice of food was not limited to plant materials but consumption of animal protein, as well as a higher difference in physical activity in the control group.

This study found that energy intake influences the lipid profile. The energy between the treatment and control groups had a significant difference. Energy intake in the treatment group was not as planned which should be 1500 kcal, but it was sufficient as much as 84.73%. Research with similar explain that giving a low energy moderate carbohydrate diet (MCD diet) for 4 weeks can increase HDL levels by 7.31 mg/dL.40 When compared before the intervention, both groups experienced a decrease in energy. So the lower the energy intake, the higher the HDL level, and vice versa. However, this study does not follow the theory. It was explained that the high accumulation of energy will be stored in the form of adipose tissue and fat, then metabolized, increasing LDL levels causing a decrease in HDL levels.[41]

There was no significant difference in protein intake during the intervention. Protein given to the treatment group of 14.2% was sufficient for planning for 13-16% of energy. Protein sourced from vegetable ingredients is the hallmark of a plant-based diet. The protein consumed has an influence on the lipid profile, one of which is HDL levels. The composition of foods containing protein, especially animal protein in the form of red meat and its processed products, increases the risk of cardiovascular disorders because of the high content of saturated fatty acids.[42] Then, it was different with the fat intake in the two groups when the intervention had a significant difference. The fat intake of this flexitarian diet is 24.36% or lower than the plan of giving 25-35% of the total energy. Fat is a component that has a major influence on cholesterol metabolism.[43] Reducing fat intake in the food consumed is associated with lower HDL levels. Based on the theory states that the intake of foods with high fat will increase cholesterol in the blood.

The application of a plant-based diet tends to contain monounsaturated fatty acids or monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids or polyunsaturated fatty acids (PUFA), as well as low saturated fat or saturated fatty acids (SFA). Unsaturated fatty acids are beneficial in lowering LDL levels and increasing HDL levels. If it is seen from the PUFA, MUFA, and SFA intake data in the treatment group, it is much lower than the control group. The intake of MUFA and SFA of the two groups had a significant difference. This is related to the HDL levels in the treatment group which decreased, while the control group experienced an increase. Recommendations from PERKENI in 2019, intake of SFA <7% calorie requirement, PUFA <10%, and MUFA as much as 12-15%[44] From this reference, the intake of SFA, PUFA, and MUFA for both groups was still lacking. There is a theory related to high consumption of SFA can increase LDL levels so that it has strong potential as atherosclerosis and carcinogenic effects.[40] The randomized control trial, comparing the administration of animal-based protein and plant-based protein diets for 12 weeks, it turned out that PUFA and fiber levels were higher in the plant-based diet group but there was no change in HDL.45 Vegetable fats and proteins rich in PUFA and MUFA can control the process of dyslipidemia. In addition, the explanation of plant-based diets with unsaturated fat content tends to have high phytonutrients, the mechanism for reducing HDL levels has been described previously. Then, the consumption of foods rich in PUFA and MUFA can reduce LDL production so that HDL levels can be increased. This phenomenon explains that low SFA intake accompanied by high PUFA and MUFA will inhibit HMG-CoA reductase activity which can control cholesterol synthesis to the liver.[30]

Carbohydrate intake during the intervention had a significant difference in both the treatment group and the control group. Carbohydrate intake in the treatment group was much lower than in the control group. However, it fulfills 50,8% so it is by the plan of giving carbohydrates 50-57% of energy needs. Previous research in the form of a low-carbohydrate diet was associated with an increase in HDL levels of 1,73 mg/dL.46

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Supported by the research giving a low fat low carbohydrate diet for 12 months can significantly increase HDL levels and reduce triglycerides. Correlating with the theory of decreasing fat intake, there is an increase in carbohydrate metabolism that triggers hypertriglyceridemia.[47] This is incompatible with carbohydrate metabolism and free fatty acids are oxidized to Acetyl-CoA to produce energy. If carbohydrate intake increases, the formation of Acetyl-CoA will also increase and then be stored in the liver and adipose tissue.

Fiber intake during the intervention had no significant difference between the two groups. Fiber consumption in the treatment group met 54% of the recommendations given, so it was still less than 25-35 grams/day. Based on research explain that fiber consumption from initially <18 g/day then increased to >30 g/day, HDL levels will increase by 10,1%.31 There is still a low consumption of vegetables with leftovers in the form of vegetables. Therefore, this study is contrary to the theory that a plant-based diet can increase HDL cholesterol levels and reduce LDL levels through the inhibition of the oxidation process. The content of fatty acids in vegetable proteins such as Polyunsaturated Fatty Acids (PUFA) and Monounsaturated Fatty Acids (MUFA) or unsaturated fatty acids support an increase in HDL levels in the body.[48] Consumption of high fiber can trigger an increase in the excretion of cholesterol and bile salts through the feces, which then enter into enterohepatic metabolism causing the bile salt content to decrease.[49] The bile salts enter the liver pathway and there is low absorption of cholesterol so that total cholesterol can decrease. In the second cycle, fiber can convert bile salts from cholic acid to chenodeoxycholic acid, thereby inhibiting the activity of HMG CoA reductase in cholesterol synthesis. The third cycle, namely through the conversion of bacterial fiber, namely propionate, is in which the inhibition of fatty acids and cholesterol in the blood decreases with an increase in HDL levels.[30,31]

Cholesterol intake in the treatment group had met <200 mg to reduce the incidence of cardiovascular disease and had a significant difference. This is related to the theory that consumption of cholesterol and saturated fat can increase LDL levels by decreasing the synthesis of VLDL and HDL receptors, inhibiting bile acid excretion, and increasing the formation of smaller VLDL. Therefore, the metabolism that occurs in extrahepatic tissues is slower and high cholesterol content can cause atherosclerosis. The mechanism of HDL reduction is due to a large amount of saturated fatty acids in cholesterol and causes a decrease in levels of apolipoprotein A-1 (the precursor for HDL formation) thereby inhibiting the synthesis of HDL levels in the blood.[9,49]

When viewed from the intake of vitamin C it has met even more (128.6%). Based on the theory, the antioxidant content and several phytonutrients contained in a plant-based diet or vegetable sources can increase the activity of HDL levels and fight against ApoA-1.[50,51] In addition, HDL levels will increase with the consumption of foods high in antioxidants. HDL has a metabolic pathway to transport lipid peroxidase and lysophospholipids to the liver and prevent the accumulation of LDL. In addition, the presence of antioxidants and polyphenolic compounds can support the activity of paraoxonase-1 (PON-1) as an enzyme to prevent HDL oxidation, thereby strengthening HDL as an antioxidant and anti-inflammatory.[52] However, in this study, antioxidants in high consumption of vitamin C were not optimally able to increase HDL levels. High ascorbic acid may not only reflect vitamin C content, but there are other correlated nutrients such as potassium, folate, calcium, magnesium, and isoflavones that have cardiovascular benefits. Most likely the explanation for the decrease in HDL levels is due to the Reverse Cholesterol Transport (RCT) process.

There was a significant difference between the physical activity of the two groups between the treatment group and the control group ($p<0.05$). The majority control group had light to moderate levels of physical activity. In the study giving physical activity can increase HDL levels by 0.27-5.41 mg/dL with certain variations of the exercise, duration, and intensity.[53] When viewed in terms of the subject's physical activity before and after the intervention, 63.6% experienced a decrease. Physical activity is correlated with HDL levels, increased physical activity can help the formation of ATP, so the body will respond to the formation of HDL to facilitate excess cholesterol in peripheral tissues and then transfer to the liver for further metabolism.[54] Regular physical activity can help increase the activity of the lipoprotein lipase enzyme and decrease the hepatic lipase enzyme. Lipoprotein lipase will hydrolyze triglycerides and VLDL, thereby increasing the conversion of VLDL and IDL. IDL is then converted to LDL and the rest is taken up by the liver and peripheral tissues via LDL receptors. This is the reason cholesterol levels, LDL decreases, while HDL increases.[9]

Based on the above discussion, it is necessary to improve the right intake to increase HDL levels, in the form of consumption of unsaturated fats, namely PUFA and MUFA which are found in several foodstuffs such as fish, coconut oil, avocado, vegetable protein, and low in saturated fat.[43] This type of vegetarian diet needs to pay attention to the intake of vitamins A, D, B12, calcium, riboflavin, omega 3, protein, iron, and

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zinc.[2]2 Giving a low-fat, low-carbohydrate diet can also significantly increase HDL levels.[47,55] Recommended diet pattern with high intake of fiber and fruit, low-fat dairy, nuts and seeds, reduced consumption of meat, sugar-sweetened foods, and snacks.[22,56] Polyphenol compounds in vegetable protein also act as anti-atherogenic. Polyphenols will increase the size and stability of HDL accompanied by a decrease in triglyceride levels.[52] Therefore, a flexitarian diet can be recommended for a period longer than 4 weeks, to see the effectiveness of changes in HDL levels.

This study aimed to analyze the effect of implementing a flexitarian diet on HDL levels among young adults, particularly obese female students. Given the awareness of the risks and dangers of obesity in this demographic, researchers were motivated to regulate food intake through interventions such as the flexitarian diet, which is not widely practiced in Indonesia. This study serves as a valuable reference for future research initiatives.

Limitations of this study include the relatively short duration of the flexitarian diet and the limited sample size, which was constrained by the scope of the research grant. Nevertheless, efforts were made to ensure uniformity among subjects in terms of characteristics, provide optimal diet and motivation, and implement control measures through regular monitoring of intake and physical activity.

CONCLUSIONS

Providing a flexitarian diet for 4 weeks resulted in a significant reduction of 2 mg/dL in HDL levels within the treatment group. However, no significant difference was observed in the mean change in HDL levels. Energy and fat intake in the flexitarian diet treatment group were lower and significantly different from those in the control group. Although carbohydrate and protein intake were lower, fiber intake was higher in the treatment group, although not significantly so. When considering confounding variables such as physical activity, the control group exhibited greater physical activity compared to the treatment group, with significant differences noted.

SUGGESTION

Further research is needed on plant-based diets for longer durations and with larger sample sizes to fully ascertain the effects of plant-based diets, particularly flexitarian diets, on changes in HDL levels. Additionally, during the administration of a flexitarian diet, it may be more effective to implement control measures for food intake and balance it with regular and structured physical exercise.

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REFERENCES

1. World Health Organization (WHO). Overweight and obesity. Published 2015. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight
2. Kementerian Kesehatan RI. Riset kesehatan dasar 2018. Jakarta : Badan Penelitian dan Pengembangan Kesehatan Kementrian Kesehatan RI.
3. Hruby A, Hu F. B. The epidemiology of obesity: a big picture. Pharmacoeconomics. 2015;33(7):673-689. doi:10.1007/s40273-014-0243-x
4. Rachmi C. N, Li M, Alison Baur L. Overweight and obesity in Indonesia: prevalence and risk factors—a literature review. Public Health. 2017;147:20-29. doi:10.1016/j.puhe.2017.02.002
5. Dinas Kesehatan Provinsi Jawa Tengah. Profil kesehatan Provinsi Jawa Tengah.
6. Dieny F. F, Widyastuti N, Fitranti D. Y. Sindrom metabolik pada remaja obes: prevalensi dan hubungannya dengan kualitas diet. Jurnal Gizi Klinik Indonesia. 2015;12(1):1-11. doi:10.22146/ijcn.22830
7. Meldrum D. R. Introduction: obesity and reproduction. American Society for Reproductive Medicine. 2017;107(4):831-832. doi:10.1016/j.fertnstert.2017.02.110
8. Azkia F. I, Miko Wahyono T. Y. Hubungan pola konsumsi makanan berisiko dengan obesitas sentral

e-ISSN : 2338-3119, p-ISSN: 1858-4942

pada wanita usia 25-65 tahun di Bogor tahun 2011-2012. Jurnal Epidemiologi Kesehatan Indonesia. 2019;2(1). doi:10.7454/epidkes.v2i1.1675
9. Mahan L. K, Raymond J. L. Krause's Food & the Nutrition Care Process. 14th ed. Elsevier Inc.; 2017.
10. Kurdanti W, Suryani I, Syamsiatun NH, et al. Faktor - faktor yang mempengaruhi kejadian obesitas pada remaja. Jurnal Gizi Klinik Indonesia. 2015;11(4):179. doi:10.22146/ijcn.22900
11. Dieny F. F, Widyastuti N, Fitranti D. Y, Nissa C, Tsani A. F. A, Jauharany F. F. Defisiensi besi pada wanita usia subur pranikah obesitas. Media Gizi Mikro Indonesia. 2019;10(2):101-110. https://doi.org/10.22435/mgmi.v10i2.599
12. Fernando M. L. Gambaran citra tubuh pada wanita dewasa awal. Jurnal Ilmiah Psikologi Terapan. 2019;07(01):101-118. doi:10.22219/jipt.v7i1.6369
13. Widiawati S, Novayanti M, Ismiyati. Diet pada wanita usia subur (WUS) obesitas. In: Pameran Poster Ilmiah Pencegahan Penyakit Tidak Menular. Poltekkes Kemenkes Banten; 2019:37-41.
14. Mamat, Sudikno. Faktor-faktor yang berhubungan dengan kadar kolesterol HDL (Analisis data of the Indonesian family life survey 2007/2008). Jurnal Gizi Indonesia. 2010;33(2):143-149.
15. Mahbuba S, Mohsin F, Rahat F, Nahar J, Begum T. Descriptive epidemiology of metabolic syndrome among obese adolescent population. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. Published online 2018:6-11. doi:10.1016/j.dsx.2017.12.026
16. Sigit F. S, Tahapary D. L, Trompet S, Sartono E, Dijk K. W. Van. The prevalence of metabolic syndrome and its association with body fat distribution in middle-aged individuals from Indonesia and the Netherlands : a cross-sectional analysis of two population-based studies. Diabetology & Metabolic Syndrome. 2020;12(2):1-11. doi:10.1186/s13098-019-0503-1
17. Rose S, Dieny FF, Nuryanto, Tsani AFA. The correlation between waist-to-height ratio (WHtR) and second to fourth digit ratio (2D:4D) with an increase in metabolic syndrome scores in obese adolescent girls. Electronic Journal of General Medicine. 2020;17(3):1-9. doi:10.29333/ejgm/7872
18. Woudberg N. J, Goedecke J. H, Blackhurst D, et al. Association between ethnicity and obesity with high-density lipoprotein (HDL) function and subclass distribution. Lipids in Health and Disease. 2016;15(1):1-11. doi:10.1186/s12944-016-0257-9
19. Erwinanto E, Santoso A, Putranto J. N. E, et al. Panduan Tata Laksana Dislipidemia Tahun 2017. Perhimpunan Dokter Spesialis Kardiovaskular Indonesia; 2017. http://jki.or.id
20. Guevara-Cruz M, Flores-López AG, Aguilar-López M, et al. Improvement of lipoprotein profile and metabolic endotoxemia by a lifestyle intervention that modifies the gut microbiota in subjects with metabolic syndrome. Journal of the American Heart Association. 2019;8(17):e012401. doi:10.1161/JAHA.119.012401
21. Derbyshire E. J. Flexitarian diets and health: a review of the evidence-based literature. Frontiers in Nutrition. 2017;3(55):1-8. doi:10.3389/fnut.2016.00055
22. Blatner D. J. The Flexitarian Diet : The Mostly Vegetarian Way to Lose Weight, Be Healthier, Prevent Disease and Add Years to Your Life. McGraw-Hill; 2009. doi:10.1036/0071549579
23. Kent L, Morton D, Rankin P, et al. The effect of a low-fat, plant-based lifestyle intervention (CHIP) on serum HDL levels and the implications for metabolic syndrome status - a cohort study. Nutrition and Metabolism. 2013;10(1):1-6. doi:10.1186/1743-7075-10-58
24. Najjar R. S, Moore C. E, Montgomery B. D. A defined, plant-based diet utilized in an outpatient cardiovascular clinic effectively treats hypercholesterolemia and hypertension and reduces medications. Clinical Cardiology. 2018;41(3):307-313. doi:10.1002/clc.22863
25. Horikawa C, Kodama S, Yachi Y, et al. Skipping breakfast and prevalence of overweight and obesity in Asian and Pacific regions: A meta-analysis. Preventive Medicine. 2011;53(4-5):260-267. doi:10.1016/j.ypmed.2011.08.030
26. Dunaief DM, Fuhrman J, Dunaief JL, Ying G. Glycemic and cardiovascular parameters improved in type 2 diabetes with the high nutrient density (HND) diet. Open Journal of Preventive Medicine. 2012;02(03):364-371. doi:10.4236/ojpm.2012.23053
27. Goldberg A. M. Farm animal welfare and human health. Current Environmental Health Reports. 2016;3(3):313-321. doi:10.1007/s40572-016-0097-9
28. Willett W, Rockström J, Loken B, et al. Food in the anthropocene: the EAT–Lancet Commission on healthy diets from sustainable food systems. The Lancet. 2019;393(10170):447-492. doi:10.1016/S0140-6736(18)31788-4

e-ISSN : 2338-3119, p-ISSN: 1858-4942

29. Forestell C. A. Flexitarian diet and weight control: healthy or risky eating behavior? Frontiers in Nutrition. 2018;5(July):1-6. doi:10.3389/fnut.2018.00059
30. Gropper SS, Smith JL, Carr TP. Advanced Nutrition and Human Metabolism. 7th ed. Wadsworth; 2017. doi:10.1016/0307-4412(92)90040-s
31. Zhou Q, Wu J, Tang J, Wang J. J, Lu C. H, Wang P. X. Beneficial effect of higher dietary fiber intake on plasma HDL-C and TC/HDL-C ratio among Chinese rural-to-urban migrant workers. International Journal of Environmental Research and Public Health. 2015;12(5):4726-4738. doi:10.3390/ijerph120504726
32. World Health Organization (WHO). The Asia - Pacific Perspective : Redefining Obesity and Its Treatment.; 2000. doi:ISBN 0957708211
33. Grundy SM, Stone NJ, Bailey AL, et al. 2018 guideline on the management of blood cholesterol : a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2019;73(24):e285-e350. doi:10.1016/j.jacc.2018.11.003
34. Magkos F, Tetens I, Bügel S. G, et al. A Perspective on the transition to plant-based diets: a diet change may attenuate climate change, but can it also attenuate obesity and chronic disease risk. Advances in Nutrition. 2020;11(1):1-9. doi:10.1093/advances/nmz090
35. Tang Y, Liu Y, Xu L, et al. Validity and reproducibility of a revised semi-quantitative food frequency questionnaire (SQFFQ) for women of age-group 12-44 years in Chengdu. Journal of Health, Population and Nutrition. 2015;33(1):50-59. doi:10.3329/jhpn.v33i1.3194
36. Fogelholm M, Malmberg JJ SJ. International physical activity questionnaire. Critical Reviews in Physical and Rehabilitation Medicine. 2006;38(4):753-760. doi:10.1249/01.mss.0000194075.16960.20
37. Yokoyama Y, Levin SM, Barnard ND. Association between plant-based diets and plasma lipids: A systematic review and meta-analysis. Nutrition Reviews. 2017;75(9):683-698. doi:10.1093/nutrit/nux030
38. Campbell EK, Fidahusain M, Campbell TM. Evaluation of an eight-week whole-food plant-based lifestyle modification program. Nutrients. 2019;11(2068):1-12. doi:10.3390/nu11092068
39. Wang F, Zheng J, Yang B, Jiang J, Fu Y, Li D. Effects of vegetarian diets on blood lipids: A systematic review and meta-analysis of randomized controlled trials. Journal of the American Heart Association. 2015;4(10). doi:10.1161/JAHA.115.002408
40. Michalczyk MM, Maszczyk A, Stastny P. The effects of low-energy moderate-carbohydrate (MCD) and mixed (MixD) diets on serum lipid profiles and body composition in middle-aged men : a randomized controlled parallel-group clinical trial. International Journal of Environmental Research and Public Health. 2020;17(1332):1-15. doi:10.3390/ijerph17041332
41. Yuliantini E, Sari A, Nur E. Hubungan asupan energi, lemak dan serat dengan rasio kadar kolesterol total-HDL. Penelitian Gizi dan Makanan (The Journal of Nutrition and Food Research). 2015;38(2):139-147. doi:10.22435/pgm.v38i2.5543.139-147
42. Hicks T. M, Knowles S. O, Farouk M. M. Global provisioning of red meat for flexitarian diets. Frontiers in Nutrition. 2018;5(50). doi:10.3389/fnut.2018.00050
43. Guyenet SJ SM. Regulation of food intake, energy balance, and body fat mass : implications for the pathogenesis and treatment of obesity. Journal Clinical Endocrinology Metabolism. 2018;97(5):745-755. https://doi.org/10.1210/jc.2011-2525
44. Soelistijo SA, Lindarto D, Decroli E, et al. Pedoman pengelolaan dan pencegahan diabetes melitus tipe 2 dewasa di Indonesia 2019\. Perkumpulan Endokrinologi Indonesia. Published online 2019:1-117. https://pbperkeni.or.id/wp-content/uploads/2020/07/Pedoman-Pengelolaan-DM-Tipe-2-Dewasa-di-Indonesia-eBook-PDF-1.pdf
45. Päivärinta E, Itkonen S. T, Pellinen T, Lehtovirta M, Erkkola M, Pajari A. M. Replacing animal-based proteins with plant-based proteins changes the composition of a whole nordic diet—a randomized clinical trial in healthy Finnish adults. Nutrients. 2020;12(4):1-16. doi:10.3390/nu12040943
46. Santos F, Esteves S, Pereira A, Yancy Jr W NJ. Systematic review and meta-analysis of clinical trials of the effects of low carbohydrate diets on cardiovascular risk factor. Obesity Review. 2012;13:1048-1066. doi:10.1111/j.1467-789X.2012.01021
47. Chawla S, Silva FT, Medeiros SA, Mekary RA, Radenkovic D. The effect of low-fat and low-carbohydrate diets on weight loss and lipid levels: A systematic review and meta-analysis. Nutrients. 2020;12(12):1-21. doi:10.3390/nu12123774

e-ISSN : 2338-3119, p-ISSN: 1858-4942

48. Rachmawati S, Sulchan M. Asupan lemak dan kadar high density lipoprotein (HDL) sebagai faktor risiko peningkatan kadar C-reactive protein (CRP) pada remaja obesitas dengan sindrom metabolik. Journal of Nutrition College. 2014;3(3):337-345. doi:10.14710/jnc.v3i3.6586
49. Trajkovska K. T, Topuzovska S. High-density lipoprotein metabolism and reverse cholesterol transport: Strategies for raising HDL cholesterol. Anatolian Journal of Cardiology. 2017;18(2):149-154. doi:10.14744/AnatolJCardiol.2017.7608
50. Barnard ND, Goldman DM, Loomis JF, et al. Plant-based diets for cardiovascular safety and performance in endurance sports. Nutrients. 2019;11(1):1-10. doi:10.3390/nu11010130
51. Thaochalee C, Sarikaputi A, Bumrungpech A, Chalermchai T, Nararatwanchai T. Anti-inflammatory effects of plant-based thai diets in overweight and obese individuals. Songklanakarin Journal of Science and Technology. 2021;43(3):840-846. doi:10.14456/sjst-psu.2021.111
52. Rondanelli M, Giacosa A, Morazzoni P, et al. MediterrAsian diet products that could raise HDL-cholesterol: A systematic review. BioMed Research International. 2016;2016. doi:10.1155/2016/2025687
53. Palazón-Bru A, Hernández-Lozano D, Gil-Guillén VF. Which physical exercise interventions increase HDL-cholesterol levels? a systematic review of meta-analyses of randomized controlled trials. Sports Medicine. 2021;51(2):243-253. doi:10.1007/s40279-020-01364-y
54. De Munter J, Van Valkengoed IG, Stronks K, Agyemang C. Total physical activity might not be a good measure in the relationship with HDL cholesterol and triglycerides in a multi-ethnic population: a cross-sectional study. Published online 2011. doi:10.1186/1476-511X-10-223
55. Mansoor N, Vinknes KJ, Veierød MB, Retterstøl K. Effects of low-carbohydrate diets v. low-fat diets on body weight and cardiovascular risk factors: a meta-analysis of randomized controlled trials. Published online 2015. doi:10.1017/S0007114515004699
56. Trautwein EA, McKay S. The role of specific components of a plant-based diet in management of dyslipidemia and the impact on cardiovascular risk. Nutrients. 2020;12(9):1-21. doi:10.3390/nu12092671

e-ISSN : 2338-3119, p-ISSN: 1858-4942