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Potential of avocado oil (*Persea americana*) in improving triglyceride and High Density Lipoprotein (HDL) levels in rats (*Rattus Novergicus*) model of dyslipidaemia due to high fat diet

Eka Puspita Sari1, Brian Wasita2, Muthmainah3

1Department of Nutrition Science, Postgraduate Faculty, Sebelas Maret University of Surakarta, Indonesia
2Department of Anatomic Pathology, Faculty of Medicine, Sebelas Maret University of Surakarta, Indonesia
3Department of Histology, Faculty of Medicine, Sebelas Maret University of Surakarta, Indonesia

**Correspondence*: ekapsari25@gmail.com

ABSTRAK

Latar Belakang: *Dislipidemia adalah keabnormalan kadar profil lipid yang berisiko pernyakit kardiovaskular. Tingginya kandungan "Asam Lemak Tak Jenuh Tunggal" (MUFA) dan "Asam Lemak Tak Jenuh Ganda" (PUFA) pada minyak alpukat dapat memperbaiki profil lipid pada dislipidemia.*
Tujuan: *Studi ini bertujuan untuk mengetahui potensi minyak alpukat dalam memperbaiki kadar trigliserida dan HDL.*
Metode: *Penelitian ini menggunakan desain pre-post test kontrol grup dengan tikus wistar jantan berumur 8 minggu disertai berat badan 150-200 g. Tikus dibagi menjadi enam kelompok yaitu kelompok N, K-, K+, P1, P2, dan P3. Hanya kelompok N yang tidak diberikan intervensi berupa induksi HC maupun minyak alpukat selama penelitian, sedangkan kelompok lainnya diberikan induksi HC selama 28 hari, setelah induksi kemudian pada kelompok K+ diberikan simvastatin dan kelompok perlakuan (P1, P2, dan P3) diberikan minyak alpukat selama 28 hari. Data dianalisis secara statistik menggunakan program SPSS versi.25 dan menunjukan hasil yang signifikan jika p<0,05.*
Hasil: *Rerata perubahan kadar (mg/dL) untuk TG -29,67 (P1), -48,99 (P2), -56,78 (P3) dan untuk HDL 41,75 (P1), 46,35 (P2). Terdapat perbedaan yang signifikan pada kadar TG dan HDL antara P1, P2, P3 jika dibandingkan dengan K- (p<0,05).*
Kesimpulan: *Minyak alpukat dapat memperbaiki kadar TG dan HDL serta dapat menjadi pertimbangan dalam konsumsi sehari-hari*

KATA KUNCI*: avocado oil; Dyslipidaemia; trigliserida; HDL*

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ABSTRACT

Background: *Elevated levels of Dyslipidaemia pose a significant threat to cardiovascular health. Fortunately, the abundance of polyunsaturated and monounsaturated fatty acids (PUFA and MUFA) present in avocado oil offers a promising avenue for individuals with Dyslipidaemia to improve their lipid profile levels.*

Objectives: *The objective of this research is to ascertain the potential of avocado oil in improving triglyceride and HDL levels.*

Methods: *A pre-post test control group design was employed, utilizing male Wistar rats aged 8 weeks with a body weight ranging from 150-200 g. The mice were segregated into six groups: N, K-, K+, P1, P2, and P3. Throughout the trial, solely group N abstained from ingesting avocado oil or HC. Conversely, all other groups were administered HC and subsequently, K+ was prescribed simvastatin while P1, P2 and P3 received avocado oil for a duration of 28 days. The data was subjected to statistical analysis using SPSS vr.25. The statistical significance threshold was set at $p<0.05$.*

Results: *Mean changes in levels (mg/dL) for TG -29.67 (P1), -48.99 (P2), -56.78 (P3) and for HDL 41.75 (P1), 46.35 (P2). There was a significant difference in TG and HDL levels between P1, P2, P3 when compared with K- ($p<0.05$).*

Conclusions: *Avocado oil exhibits efficacy in lowering TG levels and elevating HDL levels, rendering it a viable option for daily consumption.*

KEYWORD*:* *avocado oil; dyslipidaemia; triglyceride (TG); High Density Lipoprotein (HDL)*

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INTRODUCTION

Dislipidaemia is an abnormality in the lipid profile that is indicated by abnormal changes in lipid profile levels (3). As per the data amassed by the National Centre for Health Statistics (NCHS), a staggering 11.4% of the global adult population exhibited elevated levels of total cholesterol, while 17.2% suffered from reduced high-density lipoprotein (HDL) between 2015 and 2018.In 2018, the incidence of Dyslipidaemia (Total Cholesterol/TC ≥ 190 mg/dL) in Southeast Asia was recorded at 30.3%, whereas Indonesia showed a prevalence rate of approximately 36% for Dyslipidaemia (TC ≥ 200 mg/dL) among adult populations.

Dyslipidaemia is a predisposing factor for “Atherosclerotic Cardiovascular Disease” (ASCVD), marked by the deposition of plaque within the vasculature (1). Dyslipidaemia-induced heart disease has been shown to significantly contribute to mortality rates. Shockingly, the year 2016 saw a staggering 17.9 million deaths attributed to this ailment alone, accounting for a whopping 31% of all global fatalities (2). These individuals demonstrated an elevated proportion of LDL at or above the threshold value of 190 mg/dL, a diminished proportion of HDL below the level of 40 mg/dL, and an increased proportion of TG that exceeded the cutoff point of 50mg/dL.(1-3).

Managing Dyslipidaemia is crucial in averting complications within the cardiovascular system. The primary objective of Dyslipidaemia control entails regulating LDL levels, which represents the main atherogenic cholesterol, triglyceride levels, and maintaining body weight below normal thresholds in accordance Body Mass Index. A dietary regimen capable of regulating LDL levels entails decreasing the consumption of saturated and trans fatty acids while simultaneously increasing intake of “Monounsaturated Fatty Acids”(MUFA) and “Polyunsaturated Fatty Acids”(PUFA) (6). The recommended total fat consumption is around 20-35% of total energy requirements/day. For PUFA it is around 6-11% and MUFA 0-25% by encouraging the consumption of olive oil as recommended by the

Spanish Federation of Food, Nutrition and Dietetics. MUFA and PUFA reduce TC, LDC, TG and increase HDL-C (30). Additionally, it should regulate carbohydrate intake to be less than 60% of total calories (6,7). In addition to lifestyle management, Dyslipidaemia control can be achieved through pharmacological intervention. Study by Putir (2018) mentioned that statin drugs are the most commonly prescribed drugs to control Dyslipidaemia (8). However, it is important to note that the utilization of statin-class medications may result in adverse effects over an extended duration, including myalgia, myositis/myopathy, and rhabdomyolysis (8).

In light of the adverse effects associated with pharmacological interventions, attempts have been made to regulate Dyslipidaemia through employment of natural substances that are anticipated to elicit lesser side less effects. Amongst such alternative options is avocado oil, which has exhibited potential in managing Dyslipidaemia. Avocado production is abundant in Indonesia. The avocado is a highly valued holticultural commodity fruit. Plant's economic significance is demonstrated by the annual increase in yield recorded by the Indonesian Central Bureau of Statistics (9).

Avocado oil boasts the benefit of possessing a high concentration of MUFA, which endows it with exceptional stability against oxidation during storage and when subjected to high temperatures (10,11). Consequently, it is an excellent alternative cooking oil that offers superior health benefits compared to other oils. Moreover, avocado oil contains of PUFA that facilitating the reduction of lipid profile levels (11). Avocado oil produced through cold pressing methods comprises of monounsaturated fatty acid (MUFA) within the range of 65.29-71.31% and polyunsaturated fatty acid (PUFA) ranging from 11.30-16.41%. The primary fatty acid present in this oil is oleic acid, accounting for about 59.46-67.69% (12). However, the availability of avocado oil in the market is not as much as other oils because the distributor of avocado oil production in Indonesia is still minimal, besides that avocado oil consumers are still very rare when compared to other countries where the use of vegetable oils is very common, which can be seen from the MUFA consumption rate in Indonesia is still very low around 5.2% compared to the United States 12.5%, Australia 11.8%, UK 117%, South Africa 9.5%, China 8.1% (13).

Based on research Nasef & Ahmed, (2019) (14) Administration of 25% avocado powder was effective (P ≤0.05) in reducing total cholesterol, triglycerides, LDL, VLDL and atherogenic index and reducing serum "aspartate aminotransferase"(AST), "alkaline phosphatase"(ALP) and "Alanine transaminase"(ALT) levels compared to the positive control group but administration of avocado fruit powder did not show histopathological changes. The administration of freeze-dried avocado pulp and avocado paste resulted in a significant reduction of 76.47% and 46.06%, respectively, in serum TG levels. Additionally, the consumption of freeze-dried avocado pulp led to a decrease in serum TC levels by 16.39%(15). Prior studies on urinary metabolism have demonstrated that virgin avocado oil has the capacity to ameliorate metabolic dysfunction induced by hypercholesterolemia. Over a period of 28 days, the administration of VAO at doses of 450 and 900 mg/kgBW resulted in reduced levels of "total cholesterol" (TC), "triglycerides" (TG), and "low-density lipoprotein" (LDL) compared to the control group that was subjected to a high-fat diet. Conversely, there was an increase in "high-density lipoprotein" (HDL). Additionally, avocado oil administration led to significant differences ($p < 0.05$) in "alanine transaminase" (ALT), "aspartate transaminase" (AST), and "alkaline phosphatase"(ALP) levels (16,17).

Several researchers scoping review study by sari.,et all (2024) (29) have conducted studies on the relationship between avocado oil and Dyslipidaemia. However, no research has been carried out using Indonesian-grown avocado oil produced through cold-press methods in rat models of Dyslipidaemia. Based on this and the description above, the researcher wants to identify the effect of avocado oil in improving TG levels and HDL levels in HC (high Cholesterol) dyslipidaemia model rats. The benefits of writing this article are to provide information related to the benefits of avocado oil in reducing TG levels and increasing HDL levels in Dyslipidaemia conditions and can be used as further reference material to

be applied in everyday life by using avocado oil in the cooking process.

MATERIALS AND METHODS

The objective of this research was to assess the efficacy of avocado oil on triglyceride and high-density lipoprotein levels. The research true experimental employed a pre-posttest design with a control group, and was carried out at the Centre for Food and Nutrition Studies (PSPG) Laboratory of Gadjah Mada University in Yogyakarta from May through July 2023. The Centre for Food and Nutrition Studies (PSPG) Yogyakarta also measured TG and HDL levels. “The Research Ethics Committee of the Faculty of Medicine, Sebelas Maret University”with letter No. 86/UN27.06.11/KEP/EC/2023, has approved and granted ethical permission for all procedures performed on the research subjects.

Research Materials

The Virgin Avocado Oil (VAO) was acquired from the esteemed PT Tamba Sinjiwani Bali Official Store and is certified by BPOM MD number 111222043142. This premium avocado oil is meticulously crafted using the cold-pressed method and has undergone rigorous quality testing in accordance with ISO and SNI standards, which has earned it a distribution permit granted by BPOM. The use of the cold pressed method is carried out at low temperatures so that the resulting oil is richer in nutrients and high quality. Researchers are not part of PT Tamba Sinjiwani and do not receive any compensation. The ingredients for making High Fat Diet (HC) consist of: Cholic acid (Lot #MKBR9198V) and cholesterol (Lot #SLBR3491V) from SIGMA Life Science company and Comfeed AD II standard feed from JAPFA company. Materials for checking TG and HDL levels were GPO-PAP reagent (Lot16896) and CHOD-PAP reagent (Lot19421) from Berkat Mulia company.

Animal and Diet

Thirty six (36) male Wistar rats (Rattus novergicus) aged 8 weeks old, weighed between 150-200 grams, and showed sound health with no physical abnormalities were used in this study. The exclusion criteria included any rats that did not develop Dyslipidaemia after the induction period or those that died during the research.

The preparation of the high Cholesterol (HC) was formulated using the method of Tan et al., (2018) (17). The HC feed comprised of a blend of 95% conventional feed components, 4% cholesterol and 1% cholic acid. Following the mixing process, all the feed constituents were combined to form pellets using an extender and subsequently underwent desiccation in a cabinet dryer at approximately ±40 0C. The HC was consumed ad libitum at a rate of 20 g/day for twenty-eight uninterrupted days.

The research total sample 36 rats and was divided into 6 groups, namely: N (normal group not given HC and simvastatin or avocado oil treatment), K- (negative control group given HC without simvastatin or avocado oil treatment), K+ (positive group given HC and simvastatin 0.9 mg/kgBW), P1 (treatment group 1 given HC and low dose avocado oil 0.48 ml/200gBW), P2 (treatment group 2 given HC and medium dose avocado oil 0.96 ml/200gBW), and P3 (treatment group 3 given HC and high dose avocado oil 1.44 ml/200gBW). The dose calculation in rats is a conversion from the dietary guidelines for Dyslipidaemia patient (15% MUFA of all total energy requirements).

Avocado oil administration dosage

Avocado oil was orally administered via a oral gavage on a daily basis for 28 consecutive days based on research Tan et al., (2018) (16). According to the dietary guidelines for individuals with dyslipidaemia, an adult intake of 15% MUFA (monounsaturated fatty acids) (29) is necessary from the total energy requirement of 397 kcal/ 2650 kcal. It has been established that each 100g of avocado oil contains approximately 7.4g of MUFA content. Thus, the dose of avocado oil needed to meet the dietary needs of dyslipidaemia in humans is 397 kcal/7.4 g = 53.6 kcal/g. As for the need for rats, the dose in humans is multiplied by a certain conversion rate. The conversion rate of a human dose with a body weight of 70 kg to a rat weighing 200 g is 0.018. So the dose of avocado oil given to rats is 0.96 kcal/g. 0,96/66,6 x 100 = 1,44 ml. 66.6 from MUFA levels (7.4%) multiplied by 9 (converted to energy, where 1 gram of fat = 9 Kcal). 1,44 ml the calculated dose is used

as a high dose. 1.44 ml is divided into 3 to get a low dose of 0.48 ml. The medium dose is obtained by reducing the high dose with a low dose, is 0.96 ml.

Simvastatin Dosage

Simvastatin hinders the activity of 3-hydroxy-3-methylglutaroyl-coenzymeA (HMG-CoA) enzyme, administration of simvastatin can lower triglycerides, by as much as approximately 50%, and raise high-density lipoprotein (HDL). The greater the baseline level of triglycerides the greater the percent triglyceride reduction will be with statin treatment (18). In the study, based on research Untari & Pramukantoro, 2020 has reported the optimal dose of the simvastatin in animal trial, is 0.9 mg/kgBW rats(19).

TG and HDL Level Check

The analysis of TG and HDL concentrations was conducted on the serum samples obtained from rats via the retroorbital vein. Prior to blood sampling, the rats were administered ketamine at a dosage of 1 mg/kgBW intramuscularly for anaesthesia. Blood was taken through the orbital sinus as much as ± 2 mL, then the blood was centrifuged for 10 minutes at 300 rpm, then the serum was taken using a pipette and mixed with reagents. Triglyceride levels were checked using the GPO-PAP method. 10 µL of plasma was reacted with 1000 µL of triglyceride kit reagent, shaken until pink, and incubated for 10 minutes at room temperature. Standards were made by mixing 10 µL of triglyceride standard and 1000 µL of triglyceride kit reagent. Blank samples were prepared from 1000 µL of reagent kit reagents without additional ingredients. The absorbance of the samples was measured using a 5010V5+ photometer at a wavelength of 546 nm, and the HDL value was determined using the CHOD-PAP method (20).

The measurement of TG and HDL levels in mg/dL occurred both prior to and subsequent to treatment with avocado oil.

Statistical analysis

Statistical analysis was conducted to ascertain differences in TG and HDL levels between groups before and after treatment, using One-Way Anova test followed by Tukey HSD post hoc test. Similarly, paired t-tests were employed to determine variations in TG and HDL levels before and after avocado oil treatment within each group. The statistical significance threshold for all tests was set at $p \leq 0.05$. All data analysis was carried out using SPSS VERSION 25.

RESULTS AND DISCUSSIONS

As per the One-Way Anova statistical examination presented in Table 1, it is evident that the intake of avocado oil doses measuring 0.48 mL/200gBW (P1), 0.96 mL/200 gBW (P2), and 1.44 mL/200gBW (P3) has a an impact ($p<0.05$) on TG levels for a period of twenty-eight days. The difference in the mean value of the pre-test and post-test of the avocado oil group doses of 0.48 mL/200 gBW (P1), 0.96 mL/200 gBW (P2), and 1.44 mL/200 gBW (P3) shows that the greater the dose used, the greater the effect of reducing TG levels.The outcomes of the paired t-test analysis as presented in **Table 1** reveal that there is a significant difference in the mean value of pre-test and post-test within each group, namely N, K-, K+, P1, P2, and P3 with $p<0.05$. Notably, the difference between pre-test and post-test TG levels was observed to be 29.67 mg/dL for group P1, 38.48 mg/dL for group P2, and 56.78 mg/dL for group P3. The P3 cohort exhibited a greater disparity in triglyceride (TG) levels compared to the K+ group, with the latter showing an overall TG level difference of 54.70 mg/dL. A visual representation of the pre-test and post-test TG level variations across all groups is depicted in **Figure 1**.

According to the results of the One-Way Anova statistical analysis presented in **Table 1**, it is evident that the administration of varying doses of avocado oil (P1: 0.48 mL/200gBW, P2: 0.96 mL/200gBW, and P3: 1.44 mL/200gBW) has a significant impact ($p<0.05$) on elevating HDL levels for a duration of 28 days. The difference in the average value of pre-test and post-test avocado oil group doses of 0.48 mL/200 gBW (P1), 0.96 mL/200 gBW (P2), and 1.44 mL/200 gBW (P3) shows that the greater the dose used, the greater the effect of increasing HDL levels. **Table 1** displays the outcomes of the paired t test research, indicating that every N, K-, K+, P1, P2, and P3 group exhibits a noteworthy dissimilarity in their average pre-test and post-test values with

p<0.05. Group P1 saw an elevation of 41.75 mg/dL in their HDL levels between pre- and post-tests; group P2 experienced a rise of 46.35 mg/dL; while group P3 had an impressive increase of 564.34 mg/dL in their HDL levels during this period. The P3 group had a higher difference in TG levels than the K+ group which had a difference in HDL levels of 48.74 mg/dL. group.

Table 1. Characteristics of Subjects

Lipid Profile	Group	Mean±S.D. (mg/dL) Before Treatment	Mean±S.D. (mg/dL) After Treatment	∆ (mg/dL) Lipid Profile	p
	N	69.49 ± 1.52	70.80 ± 1.94	1.30 ± 0.67	<0.001[a]*
	K-	136.04 ± 2.55	138.53 ± 3.5	2.49 ± 1.67	<0.001[a]*
	K+	135.10 ± 2.25	80.40 ± 3.06	-54.70 ± 1.61	<0.001[a]*
TG	P1	133.80 ± 1.82	104.13 ± 3.44	-29.67 ± 2.71	<0.001[a]*
	P2	132.86 ± 2.89	83.86 ± 4.94	-48.99 ± 5.01	<0.001[a]*
	P3	134.51 ± 4.16	77.73 ± 1.55	-56.78 ± 4.72	<0.001[a]*
	P	0.000[b]*	0.000[b]*	0.000[c]*	
	N	81.32 ± 1.55	79.92 ± 1.99	-1.40 ± 0.87	<0.001[a]*
	K-	25.27 ± 1.32	24.18 ± 1.53	-1.09 ± 0.36	<0.001[a]*
HDL	K+	25.16 ± 1.25	73.90 ± 2.34	48.74 ± 2.32	<0.001[a]*
	P1	24.49± 1.25	66.25 ± 2.39	41.75 ± 3.13	<0.001[a]*
	P2	24.27 ± 2.30	70.62 ± 1.89	46.35 ± 2.07	<0.001[a]*
	P3	25.16± 1.10	79.51 ± 2.13	54.34 ± 2.70	<0.001[a]*
	P	0.000[b]*	0.000[b]*	0.000[c]*	

Description: N: Normal group not given HC induction and not given avocado oil; K-: group given HC induction but not given avocado oil; K+: group given HC induction and simvastatin 0.18 mg/200gBW; P1: group given HC. a) statistical test with paired t-test; b) statistical test with One-Way ANOVA

As depicted in **Table 1**, it is evident that all witnessed an elevation in their TG and HDL levels except for the standard and K- groups. This is because in the normal group the rats were not given HC and were not given the intervention of either simvastatin or avocado oil, while in the K- group the rats were given HC but were not given the intervention of either simvastatin or avocado oil. In the other groups, rats received interventions in the form of both simvastatin and avocado oil.

According to the Tukey HSD post hoc test analysis, there exists a statistically significant difference (p<0.05) subsequent to the administration of avocado oil. This signifies that dyslipidemic rats given avocado oil in treatment groups (P1, P2, and P3) for 28 days experienced a reduction in TG levels and an augmentation in HDL levels compared to the K- group. The post hoc data obtained identified that the difference value of the dose group 0.96 mL/200 gBW (P2) and 1.44 mL/200 gBW (P3) compared to the K+ group (simvastatin) was statistically not significantly different with a value of p=0.215 and p=0.896 respectively in the TG group and a value of p=0.468 and p=0.229 respectively in the HDL group. The outcomes of this analysis demonstrate that both KP:P2 and KP:P3 possess the capability to mitigate TG levels and enhance HDL levels, with a marginal difference from simvastatin medications. Notably, P3 exhibits a substantial decrease in TG levels by an average of 56.78 mg/dL and a significant increase in HDL levels by an average of 54.34 mg/dL compared to other treatments.

In this research, induction for 28 days with HC was carried out and successfully created a dyslipidaemia model with reference values of serum lipid profile of dyslipidaemia rats according to Ihedioha et al. (2013) (21) namely: HDL <50 mg/dL, LDL >74 mg/dL, TG >104 mg/dL, total cholesterol >145 mg/dL. The effectiveness of the Dyslipidaemia model is evidenced by the results of a Post-Hoc Tukey HSD test conducted on rats induced with HC prior to treatment. Specifically, statistical significance was observed (p<0.05) when comparing the normal group to all groups

that received HC. Induction of dyslipidaemia in this research used HC formulated by the Chin Xuan Tan method. This research is in line with research conducted by Kodariah & Wahid, (2020) (22) which showed that giving HC as ad libitum feed for 14 days in rats can significantly increase TG levels ($p<0.05$). In this study, induction was carried out for 28 days with HC and succeeded in creating a dyslipidemia model with reference to the serum lipid profile values of dyslipidemic mice according to Ihedioha et al. (2013) (21), namely: HDL <50 mg/dL, LDL >74 mg/dL, TG >104 mg/dL, total cholesterol >145 mg/dL. The effectiveness of the Dyslipidemia model was proven by the results of the Post-Hoc Tukey HSD test carried out on HC-induced mice before treatment was given. Specifically, statistical significance was observed ($p<0.05$) when comparing the normal group with all groups receiving HC. Induction of dyslipidemia in this study used HC formulated using the Chin Xuan Tan method. This research is in line with research conducted by Kodariah & Wahid, (2020) (22) which shows that giving HC as ad libitum feed for 14 days to mice can increase TG levels significantly ($p<0.05$). In this study, it can be concluded that the group given the induction experienced hyperlipidemia and a significant decrease in HDL, and the induction was declared successful.

The impact of HC on triglyceride levels is a direct consequence of its ability to elevate free fatty acids (FFA) in excess, leading to the accumulation of liver triglycerides. Following this, hepatic synthesis occurs and results in the release of triglycerides into the circulatory system as Very Low-Density Lipoprotein (VLDL). In nascent VLDL, the triglyceride core is metabolised in muscle and adipose tissue, releasing fatty acids through its interaction with Lipoprotein Lipase (LPL), which is activated by apoCII. After the VLDL core is reduced, a residual particle called Intermediate Density Lipoprotein (IDL) is formed. IDL takes up cholesterol esters from HDL via CETP. Eventually, IDL and cholesterol from HDL form LDL through its interaction with liver lipases. High-density lipoprotein (HDL) can acquire cholesterol from cells via scavenger receptor class B type 1 (SR-B1) or passive diffusion, and then either transport it directly to the liver through interaction with hepatic SR-B1 or indirectly transfer it to very low-density lipoprotein (VLDL) or low-density lipoprotein (LDL), which is facilitated by cholesteryl ester transfer protein (CETP). Triglycerides are packaged into chylomicrons through their interaction with apolipoprotein B48 (apoB48), the backbone apolipoprotein. ApoC-II are obtained from HDL as chylomicrons circulate in the bloodstream. Decreasing HDL levels and increasing TG levels can occur due to an increase in VLDL due to the presence of cholesterol ester transfer protein (CETP) as a means of transporting cholesterol esters from HDL to apolipoprotein B, apart from that there are VLDL, residual VLDL, IDL, and LDL as a substitute for triglycerides (20-22).

The present research demonstrates that the administration of avocado oil can effectively reduce TG levels, as evidenced by Figure 1. This figure depicts a significant increase in HDL and reduction in TG levels upon treatment with avocado oil. Moreover, the results of the Post Hoc test indicate a notable difference in TG and HDL levels between the treatment group (P1, P2, P3) and K- group.

Avocado oil has the ability to lower lipid profile levels as a result of its MUFA content. This component can alter the composition of VLDL, regulate the activity and expression of enzymes and proteins involved in VLDL endovascular processing and catabolism. On the other hand, PUFA inhibits the synthesis of VLDL-C and apolipoprotein-B100, leading to a decrease in serum TG concentrations by reducing triglyceride synthesis through inhibition of diacylglycerol acyltransferase, fatty acid synthase, and acetyl-CoA carboxylase. Additionally, PUFAs increase fatty acid β-oxidation via peroxisome proliferator-activated receptor (PPAR-α), thereby decreasing substrate utilisation for triglyceride formation (26). Avocado oil contains vitamin E which can neutralize intermediate peroxidase and prevent damage to vital molecules by converting radicals into hydroperoxide. Vitamin E also plays a role in increasing the performance of HDL (High-Density Lipoprotein) and reducing the speed of HDL oxidation (27).

The results of this research are in line with the results of Carvajal-Zarrabal, et al., (2014)(28) in rats induced by 30% sucrose for 4 weeks then given avocado oil can reduce TG levels and

maintain HDL levels. Other results were also found by Tan et al., (2018)(16); Tan et al., (2018)(17) stated that hypercholesterolemia rats induced by HC for 4 weeks and then given avocado oil were able to reduce TG ($p < 0.05$) and increase HDL ($p < 0.05$). In the research Carvajal-Zarrabal, et al., (2014), Tan et al., (2018) determines lipid profile levels using avocado oil whose avocados are Australian avocado hass, while in this research using avocado oil whose avocados use local Indonesian Balinese avocados. The results of this research have an advantage over previous studies where the effective dose of 3 which uses a dose of 1.44 mL / 200gBW can increase HDL higher and the same therapy time for 4 weeks.

The outcomes of this research indicate that administering 0.96 mL/200 gBW (P2) and 1.44 mL/200 gBW (P3) doses of avocado oil can result in a decrease in TG levels and an increase in HDL levels, comparable to the effects seen with simvastatin administration. This conclusion is supported by the post-hoc test results which demonstrate no significant difference between the mean differences in pre- and post-test TG levels for both the P3 group and K+ group, as well as for pre- and post-test HDL levels. A decrease in triglyceride levels and an increase in HDL levels can occur because avocado oil contains ingredients such as MUFA, PUFA, and vitamin E which affect improving triglyceride and HDL levels.

The administration of Simvastatin in this research has been found to effectively diminish triglycerides while elevating high-density lipoprotein levels. This is attributed to the ability of statins to impede cholesterol synthesis within the liver by inhibiting HMG-CoA reductase enzyme activity. The recommended dosage for statin drugs is once daily, preferably at nighttime. Statins have demonstrated significant lipid-modifying effects such as increasing HDL levels by 4-15% and reducing LDL and triglyceride concentrations by 18-55% and 7-30%, respectively. Taking statin drugs has side effects such as myopathy and increased liver enzymes with contraindications to acute/chronic liver disease(5).

The outcomes of this research suggest that dosages of avocado oil at 0.96 mL / 200 gBW (P2) and 1.44 mL / 200 gBW (P3) are capable of elevating HDL levels to near-normal ranges. This is evidenced by the Tukey HSD Post-Hoc test results, which reveal that the P3 cohort exhibits an average HDL level after a 28-day course of treatment with avocado oil that is not significantly different from that observed in the N group.

CONCLUSIONS AND RECOMMENDATIONS

Avocado oil has been proven to significantly reduce TG levels and increase HDL levels in rats with Dyslipidaemia. Among the doses tested, Dose 3 proved most effective in reducing TG levels and elevating HDL levels, surpassing the effects of simvastatin administration in the comparison group.

Avocado oil may serve as a viable option for Dyslipidaemia therapy, with its daily incorporation in cooking leading to lowered TG levels and heightened HDL levels.

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