# 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

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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

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## ABSTRAK

***Latar Belakang:*** *Dislipidemia adalah keabnormalan kadar profil lipid yang beresiko penyakit 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 terhadap peningkatan kadar HDL dan penurunan kadar trigliserida (TG).*

***Metode:*** *Penelitian ini menggunakan desain pre-post test kontrol grup dengan subjek 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 HFD maupun minyak alpukat selama penelitian, sedangkan kelompok lainnya diberikan induksi HFD 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. Hasil signifikan jika p<0,05.*

***Hasil:*** *Setelah 28 hari perlakuan terjadi penurunan kadar TG dan peningkatan kadar HDL pada kelompok P2dan P3, serta terdapat perbedaan signifikan kadar TG, kadar HDL antar kelompok P2, P3 dibandingkan dengan K-. Dosis P3 secara efektif menurunkan kadar TG dan meningkatkan kadar HDL.*

***Kesimpula***n: *Minyak alpukat dapat memperbaiki kadar TG dan HDL serta dapat menjadi pertimbangan dalam konsumsi sehari-hari*

**KATA KUNCI:** *Dislipidemia, Trigliserida, HDL*

## ABSTRACT

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

***Objectives:*** *The objective of this research is to ascertain the potential of avocado oil in elevating HDL and lowering TG 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 HFD. Conversely, all other groups were administered HFD 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****: Following treatment 28 days, there was a notable reduction in TG levels and an elevation in HDL levels observed within the P2 and P3 cohorts, with significantly divergent TG and HDL concentrations being noted between these groups as compared to K-.*

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



***KEYWORDS:*** *Dyslipidemia, 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. 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 dyslipidemia (Total Cholesterol/TC ≥ 190 mg/dL) in Southeast Asia was recorded at 30.3%, whereas Indonesia exhibited a prevalence rate of approximately 36% for dyslipidemia (TC ≥ 200 mg/dL) among adult populations. 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).

Dyslipidemia is a predisposing factor for "*Atherosclerotic Cardiovascular* Disease" (ASCVD), marked by the deposition of plaque within the vasculature (4). Dyslipidemia-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 (5).

Managing dyslipidemia is crucial in averting complications within the cardiovascular system. The primary objective of dyslipidemia control entails regulating LDL levels, which represents the main atherogenic cholesterol, managing triglyceride levels, and maintaining body weight below normal thresholds. 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). Additionally, it should regulate carbohydrate intake to be less than 60% of total calories (6,7). In addition to lifestyle management, dyslipidemia control can be achieved through pharmacological intervention utilizing medication. In the research of Putir (2018) mentioned that statin drugs are the most commonly prescribed drugs to control dyslipidemia (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.*

In light of the adverse effects associated with pharmacological interventions, attempts have been made to regulate dyslipidemia through employment of natural substances that are anticipated to elicit lesser side effects. Amongst such alternative options is avocado oil, which has exhibited potential in managing dyslipidemia.

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. Consequently, it is an excellent alternative cooking oil that offers superior health benefits compared to other oils. Moreover, avocado oil contains lower levels of PUFA than MUFA, thereby facilitating the reduction of lipid profile levels (9,10). 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%, while β-sitosterol serves as the main phytosterol component (11). 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% (12).

Based on research Nasef & Ahmed, (2019) (13) 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% (14). 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/kgBB 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, VAO administration led to significant differences ($p < 0.05$) in "alanine transaminase" (ALT), "aspartate transaminase" (AST), and "alkaline phosphatase" (ALP) levels (15,16).

Several researchers have conducted studies on the relationship between avocado oil and dyslipidemia. However, no research has been carried out using Indonesian-grown avocado oil produced through cold-press methods in rat models of dyslipidemia. 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 high-fat diet-induced 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 dyslipidemia 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 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. The ingredients for making High Fat Diet (HFD) 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 reagent and CHOD-PAP reagent.

***Research Subject***

The participants in this research were male Wistar rats (Rattus novergicus) who were 8 weeks old, weighed between 150-200 grams, and exhibited sound health with no physical abnormalities. The exclusion criteria included any rats that did not develop dyslipidemia after the induction period or those that died during the research. The research sample amounted to 36 rats and was divided into 6 groups, namely: N (normal group not given HFD and simvastatin or avocado oil treatment), K- (negative control group given HFD without simvastatin or avocado oil treatment), K+ (positive group given HFD and simvastatin 0.9 mg/kgBB), P1 (treatment group 1 given HFD and low dose avocado oil 0.48 ml/200gBB), P2 (treatment group 2 given HFD and medium dose avocado oil 0.96 ml/200gBB), and P3 (treatment group 3 given HFD and high dose avocado oil 1.44 ml/200gBB).

***Establishment of Dyslipidemia Rats Model***

The preparation of the high-fat diet (HFD) was formulated using the method of Tan et al., (2018) (16). The HFD 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 ±400 C. The high-fat feed was consumed ad libitum at a rate of 20 g/day for twenty-eight uninterrupted days.

***Avocado Oil Administration Dosage***

Avocado oil was orally administered via a gastric sonde on a daily basis for 28 consecutive days, which equates to four weeks. According to the dietary guidelines for individuals with dyslipidaemia, an intake of 15% MUFA (monounsaturated fatty acids) is necessary from the total energy requirement of 397 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 g/200gBB = 0.96 ml/200gBB rats, the calculated dose is used as a medium dose. The lower dosage is determined by dividing the calculated dose by 2, resulting in 0.48 mL/200gBB rats. Conversely, the higher dosage is obtained by adding the medium dose to the lower one, amounting to 1.44 mL/200gBB rats.

***Simvastatin Dosage***

Simvastatin hinders the activity of 3-hydroxy-3-methylglutaroyl-coenzymeA (HMG-CoA) enzyme, resulting in a decline in cholesterol and LDL levels. The suggested daily intake of simvastatin for mature individuals is 10 mg. With respect to drug conversion from human to rat, it stands at 0.018. Hence, the appropriate dosage for rats would be calculated as follows: 10 mg x 0.018 = 0.18 mg/200 gr = 0.9 mg/kgBB rats (17).

***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/kgBB 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. To evaluate TG levels, GPO reagent was utilized, whereas CHOD-

PAP reagent was used to determine HDL levels. 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, utilizing the 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/200gBB (P1), 0.96 mL/200 gBB (P2), and 1.44 mL/200gBB (P3) has a noteworthy impact ($p<0.05$) on diminishing 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 gBB (P1), 0.96 mL/200 gBB (P2), and 1.44 mL/200 gBB (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 disparity 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/200gBB, P2: 0.96 mL/200gBB, and P3: 1.44 mL/200gBB) 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 gBB (P1), 0.96 mL/200 gBB (P2), and 1.44 mL/200 gBB (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. The value and trend of changes in *pre-test* and *post-test* HDL levels in all groups can be seen in Figure 1.

**Table 1 Avocado oil administration on TG and HDL levels**

| Lipid Profile | Group | Mean±S.D. (mg/dL) Before Treatment | Mean±S.D. (mg/dL) After Treatment | Δ (mg/dL) Lipid Profile | p |
|---|---|---|---|---|---|
| TG | 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*] |
| | 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*] | |
| HDL | 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*] |
| | 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*] | |

Source: Primary Data (2023)

Description: N: Normal group not given HFD induction and not given avocado oil; K-: group given HFD induction but not given avocado oil; K+: group given HFD induction and simvastatin 0.18 mg/200gBB; P1: group given HFD

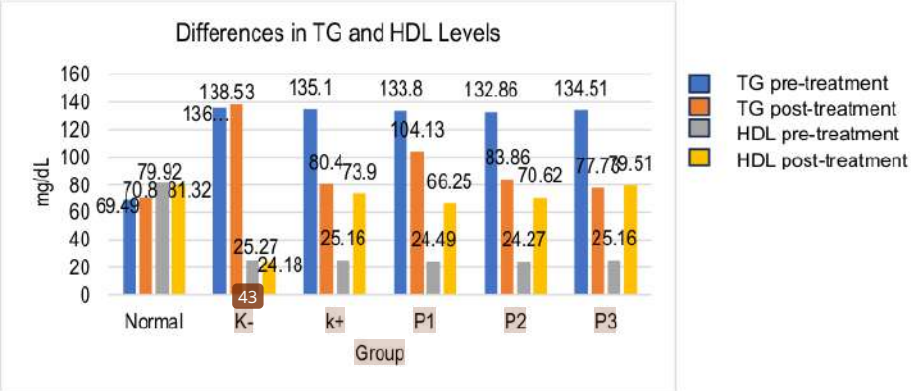


**Figure 1 Mean changes in TG and HDL levels pre test and post test**

As depicted in Figure 1, it is evident that all cohorts 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 HFD and were not given the intervention of either simvastatin or avocado oil, while in the K- group the rats were given HFD 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.

**Table 2 Post-hoc analysis of tukey hsd pre and post test and post hoc games howell delta levels of TG and hDL**

| Comparison between groups | P-value | | | | | |
|---|---|---|---|---|---|---|
| | Pre Test | | Post Test | | Delta | |
| | TG | HDL | TG | HDL | TG | HDL |
| N vs K- | <0,001* | <0,001* | <0,001* | <0,001* | 0,621 | 0,962 |
| N vs K+ | <0,001* | <0,001* | <0,001* | <0,001* | <0,001* | <0,001* |
| N vs P1 | <0,001* | <0,001* | <0,001* | <0,001* | <0,001* | <0,001* |
| N vs P2 | <0,001* | <0,001* | <0,001* | <0,001* | <0,001* | <0,001* |
| N vs P3 | <0,001* | 0,004* | 0,011* | 0,999 | <0,001* | <0,001* |
| K- vs K+ | 0,990 | 1,000 | <0,001* | <0,001* | <0,001* | <0,001* |
| K- vs P1 | 0,698 | 0,945 | <0,001* | <0,001* | <0,001* | <0,001* |
| K- vs P2 | 0,335 | 0,859 | <0,001* | <0,001* | <0,001* | <0,001* |
| K- vs P3 | 0,917 | 1,000 | <0,001* | <0,001* | <0,001* | <0,001* |
| K+ vs P1 | 0,958 | 0,971 | <0,001* | <0,001* | <0,001* | 0,015* |
| K+ vs P2 | 0,697 | 0,908 | 0,462 | 0,96 | 0,215 | 0,468 |
| K+ vs P3 | 0,999 | 1,000 | 0,722 | <0,001* | 0,896 | 0,229 |
| P1 vs P2 | 0,989 | 1,000 | <0,001* | 0,011* | <0,001* | 0,011* |
| P1 vs P3 | 0,997 | 0,971 | <0,001* | <0,001* | <0,001* | <0,001* |
| P2 and P3 | 0,890 | 0,909 | 0,035* | <0,001* | 0,144 | 0,167 |

Source: Primary Data (2023)

Description: K-: Negative control; K+: Positive control; P1: Low dose Avocado oil, P2: Medium dose Avocado oil, P3: High dose Avocado oil; *) Significant difference (p<0.05).

According to the Tukey HSD post hoc test analysis presented in Table 2, there exists a statistically significant disparity (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 gBB (P2) and 1.44 mL/200 gBB (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 HFD 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) (18) namely: HDL <50 mg/dL, LDL >74 mg/dL, TG >104 mg/dL, total cholesterol >145 mg/dL. The effectiveness of the dyslipidemia model is evidenced by the results of a Post-Hoc Tukey HSD test conducted on rats induced with HFD prior to treatment. Specifically, statistical significance was observed ($p<0.05$) when comparing the normal group to all groups that received HFD. Induction of dyslipidaemia in this research used HFD formulated by the Chin Xuan Tan method. This research is in line with research conducted by Kodariah & Wahid, (2020) (19) which showed that giving HFD as ad libitum feed for 14 days in rats can significantly increase TG levels ($p<0.05$).

The impact of HFD 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 and E are obtained from HDL as chylomicrons circulate in the bloodstream. “Cholesterol ester transfer protein (CETP) promotes the transfer of cholesterol esters from HDL to apoB containing lipoproteins, including VLDL, residual VLDL, IDL, and LDL in exchange for triglycerides. As a result, HDL cholesterol decreases, and the cholesterol content of VLDL increases” (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 (23).

The results of this research are in line with the results of Carvajal-Zarrabal, et al., (2014) (24) 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) (15); Tan et al., (2018) (16) stated that hypercholesterolemia rats induced by HFD 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 / 200gBB can increase HDL higher and the same therapy time for 4 weeks.

The outcomes of this research indicate that administering 0.96 mL/200 gBB (P2) and 1.44 mL/200 gBB (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.

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 (3).

The outcomes of this research suggest that dosages of avocado oil at 0.96 mL / 200 gBB (P2) and 1.44 mL / 200 gBB (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

Avocado oil has been shown to significantly decrease TG levels and increase HDL levels. 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.

## ADVICE

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

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