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The effects of consuming coconut milk on SGOT and SGPT levels of rats serum (*Rattus norvegicus* strain wistar) fed with high fat diet (HFD)

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ABSTRAK

Latar Belakang: *Pola makan tinggi lemak merupakan pemicu utama terjadinya obesitas dan penyakit kardiometabolik. Akumulasi lemak yang berlebihan menyebabkan peningkatan Reactive Oxygen Species (ROS) melalui hidrokarbon aromatik polisiklik (PAH), yang bermanifestasi dalam peningkatan kadar Serum Glutamic Oxaloacetic Transaminase (SGOT) dan Serum Glutamic Pyruvic Transaminase (SGPT). Santan mengandung kandungan polifenol yang dapat menghambat mekanisme ROS. Penelitian sebelumnya menyebutkan bahwa pemberian MCFA dan polifenol pada tikus Wistar yang diinduksi hiperkalori tidak memberikan efek hepatoprotektif dan mengindikasikan adanya peningkatan kadar SGOT dan SGPT.*

Tujuan: *Penelitian ini bertujuan untuk membuktikan pengaruh pemberian santan terhadap kadar SGOT dan SGPT pada tikus yang diinduksi diet tinggi lemak (HFD).*

Metode: *Penelitian ini merupakan penelitian true eksperimental dengan rancangan posttest control group design. Sampel terdiri dari 25 ekor tikus wistar (Rattus norvegicus) jantan dibagi menjadi 5 kelompok secara acak. Pengukuran SGOT dan SGPT dilakukan sesuai standarisasi IFCC (International Federation of Clinical and Chemistry and Medical Laboratory) dengan mengumpulkan sampel serum yang dilanjutkan dengan pemeriksaan menggunakan Spektrofotometer Biolyzer 100 untuk menganalisis kadar SGOT dan SGPT setelah 45 hari perlakuan. Analisis data menggunakan uji ANOVA.*

Hasil: *Hasil penelitian menunjukkan bahwa, peningkatan dosis santan makin menurunkan kadar baik SGOT/SGPT. Hasil uji Anova menunjukkan hasil yang signifikan secara statistik. Perbandingan LSD Post-hoc antara kelompok K dan P3 menunjukkan signifikansi terhadap SGPT (0,276) dan SGOT (0,707). Pemberian santan dosis 10 mL/kg berat badan/ hari pada tikus yang diinduksi HFD secara statistik, dapat menghambat peningkatan SGOT dan SGPT).*

Kesimpulan: *Berdasarkan hasil tersebut disimpulkan bahwa pemberian santan mampu mencegah peningkatan kadar SGOT dan SGPT.*

KATA KUNCI*: diet tinggi lemak;santan kelapa;SGOT;SGPT*

ABSTRACT

Background: *High-fat diets contribute to obesity and cardiometabolic diseases by increasing Reactive Oxygen Species (ROS)levels. Coconut milk's polyphenols may counteract ROS effects which manifests in increasing levels of Serum Glutamic Oxaloacetic Transaminase (SGOT) and Serum Glutamic Pyruvic Transaminase (SGPT). Coconut milk contains polyphenols which can inhibit the ROS mechanism. Previous research stated that administration of MCFA and polyphenols to Wistar rats induced by hypercalories did not provide a hepatoprotective effect and indicated an increase in SGOT and SGPT levels.*

Objectives: *This study aimed to prove the influence of coconut milk administration on the levels of SGOT and SGPT in rats induced by High Fat Diet (HFD).*

Methods: *The study was a true experimental research employing a posttest control group design. It involved 25 male Wistar rats (Rattus norvegicus) split into 5 randomly assigned groups. SGOT and SGPT measurements were carried out according to IFCC standards by collecting serum samples followed by examination using the photometric method to analyze SGOT and SGPT levels after 45 days of treatment. Data analysis used the ANOVA test using SPSS Statistics V21.0.*

Results: *The results of research on both SGOT and SGPT levels showed that increasing the dose further reduces both SGOT and SGPT levels. The Anova test results show statistically significant results. Post-hoc LSD comparison between groups K and P3 showed significance for SGPT (0.276) and SGOT (0.707).*

Conclusions: *Based on these results, it can be concluded that high-dose coconut milk administration was able to prevent the increase in SGOT and SGPT levels.*

KEYWORD*: coconut milk; high fat diet; SGOT; SGPT*

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INTRODUCTION

Fast food with high-fat content poses potential harm to the liver. This hepatic damage occurred as excessive fatty acids converted into chylomicrons within the vasculature, elevating various oxidants such as hydroxyoctadecadienoic acids (HODE), hydroxyeicosatetraenoic acids (HETE), thiobarbituric acid-reactive substances (TBARS), and oxidized lipid oxidation products (LDL). This oxidative-antioxidant imbalance led to disrupted hepatic lipid metabolism, resulting in increased levels of serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT). SGOT and SGPT are two enzymes found in the liver that are often measured in a blood test to assess liver function. SGOT is more specific to the liver than SGPT. Elevated levels of SGOT and SGPT can be caused by liver damage, but they can also be caused by other conditions such as muscle damage and heart attacks (1,2,3).

Indonesia, as the world's largest coconut producer, utilized 3.7 million hectares of land for coconut cultivation (4). A commonly consumed coconut derivative in Indonesia is coconut milk, integral to traditional Indonesian cuisine and daily meals. Due to its high saturated fatty acid content, coconut and its derivatives like coconut milk are closely associated with increased body fat (5). Saturated fatty acids found in coconut milk were classified as medium-chain saturated fatty acids or Medium Chain Fatty Acids (MCFA), which do not raise cholesterol levels as they can be rapidly metabolized in the liver (6). Consumption of coconut milk porridge can lower Low-Density Lipoprotein (LDL) and stabilize or increase High-Density Lipoprotein (HDL). Additionally, coconut milk contains galactomannan and polyphenols (7,8,9) which do not affect lipid profiles (8).

The polyphenol content in coconut milk acts as an antioxidant potentially inhibiting reactive oxygen species (ROS) mechanisms through hydroxyl groups in phenolic compounds, maintaining the balance of oxidative-reductive reactions (8). Curcumin is a plant polyphenol in turmeric root and a potent antioxidant. It binds to antioxidant response elements for gene regulation

by nuclear factor erythroid 2-related factor 2, thereby suppressing reactive oxygen species (ROS) and exerting anti-inflammatory, anti-infective and other pharmacological effects. Furthermore, polyphenols modulate cellular signaling cascades and regulate transcription factor activities to induce gene expression related to antioxidant enzymes used in cell metabolism (10), preventing intracellular hepatic protein damage and reducing SGOT and SGPT levels (11). This suggests that polyphenol-containing coconut milk may potentially act as a hepatoprotective agent.

The World Health Organization (WHO) reported that coconut meat contains coconut oil, which can reduce serum levels of SGOT, SGPT, and alkaline phosphatase (ALP) (12). Middle-chain triacylglycerol (MCT) helps prevent liver diseases by easily converting into energy, thus reducing the liver's workload and preventing fat accumulation (13). Manganese content in coconut milk also aids the body in utilizing various vitamins such as choline, thiamine, vitamin C, and vitamin E, thereby activating hepatic metabolism (14).

Previous research presented contradictory results, suggesting that administering MCFA and polyphenols to hypercaloric-induced Wistar rats did not provide hepatoprotective effects, accompanied by increased SGOT and SGPT levels (15). This discrepancy in findings sparked interest in researching the effects of coconut milk administration on SGOT and SGPT levels in rats (Rattus norvegicus strain Wistar) subjected to a high-fat diet (HFD).

MATERIALS AND METHODS

The research adopted a true experimental design with randomization into control and treatment groups, employing a posttest randomized control group design. Male Wistar rats (Rattus Norvegicus Wistar) were chosen as the experimental animals, meeting specific inclusion criteria such as male gender, body weight between 150-250 grams, and age ranging from 2 to 3 months. Criteria for dropout included rats that died during the acclimatization period until termination. Sample size calculation followed Federer's formula, with each group comprising five rats, totaling 25 rats for the study, alongside a sample size correction to anticipate potential dropouts (14). The study was conducted at the Biochemistry Laboratory and Animal Research House, Faculty of Medicine, University of Jember, spanning from January 2023 to December 2023.

In this study, the independent variables included the administration of High Fat Diet (HFD) using duck egg yolk at 2.5 mL/200gBW given every two days, used cooking oil administered at 3 mL/day, and coconut milk at varying doses of 2.5 mL/kgBW/day, 5 mL/kgBW/day, and 10 mL/kgBW/day to Wistar rats. Dependent variables encompassed the levels of SGOT and SGPT in Wistar rats. Controlled variables encompassed the type of experimental animal, gender, body weight, age, maintenance, duration of treatment, technique for making coconut milk and high-fat diet feed, as well as the frequency, volume, and method of high-fat diet administration (14,15,16).

Preparation of the High-Fat Diet involved the use of equipment such as a basin, scale, stirrer, and glass beaker, with administration using a gastric tube and gloves. The composition of HFD consisted of used cooking oil and duck egg yolk (15). The measurement of SGOT and SGPT levels followed the IFCC (International Federation of Clinical Chemistry and Laboratory Medicine) method, utilizing necessary equipment and materials including vacutainer plain tubes, Eppendorf tubes, 3 cc syringes, digital spectrophotometer Biolyzer 100, vortex mixer, cuvette, micropipette, yellow tip, blue tip, SGOT and SGPT reagents, and NADH ≥ 1 mmol/L (19).

Data obtained from the research results were analyzed using Statistical Product and Service Solution (SPSS) Statistics V21.0 software with a significance level or probability value of 0.05 ($p = 0.05$) and a confidence level of 95% ($\alpha = 0.05$). Levene's test was employed to assess the homogeneity of the research data, while the Shapiro-Wilk test was utilized to evaluate normality. If the data distribution was normal and homogeneous ($p > 0.05$), further analysis involved testing the negative control group and the HFD groups (P1-P3) using One Way ANOVA to determine the effect of coconut milk administration on SGOT and SGPT levels in rats induced by HFD. Post hoc LSD test was then conducted to identify significant differences in SGOT and SGPT levels. Ethical clearance for this study was obtained from the Ethics Committee and

Institutional Review Board of the Medical Faculty of Jember University, with the ethical clearance registration number 709/UN.1.10.2/KE/2024.

RESULTS AND DISCUSSIONS

The researcher divided the experimental animals into 5 groups and conducted interventions for 45 days. These 5 groups consisted of Group K, which was only given standard feed, Kn which was only given HFD, P1 which was given by HFD and 2.5 ml/kgBW/day of coconut milk, P2 given HFD and 5 ml/kgBW/day of coconut milk, and P3 given HFD and 10 ml/kgBW/day of coconut milk, with each group containing 5 rats.

Table 1. Average SGOT SGPT levels and standard deviation

Groups*	Average SGOT levels (U/L)	Average SGPT levels (U/L)
K	61.16 ±1.36	122.86 ± 2.44
Kn	69.14 ± 0.99	138.74 ± 1.09
P1	69.4 ± 1.34	132.8 ± 1.64
P2	64 ± 1.58	125.2 ± 2.17
P3	60.86 ±0.77	121.6 ± 1.14

*Group K, which was only given standard feed, Kn which was only given HFD, P1 which was given by HFD and 2.5 ml/kgBW/day of coconut milk, P2 given HFD and 5 ml/kgBW/day of coconut milk, and group P3 given HFD and 10 ml/kgBW/day of coconut milk

On the 46th day, blood samples were taken for examination of SGOT and SGPT levels. Blood samples were taken from the rat's heart and placed into vacutainer tubes. SGOT and SGPT examinations were performed using the kinetic IFCC method with a digital photometer. The results of SGOT and SGPT level measurements are shown in **Table 1**, **Figures 1** and **2**. **Figure 1** show bar diagrams of the mean differences of each group. The mean increase occurred between the control group and the treatment group, while the mean decrease occurred successively in groups Kn, P1, P2, and P3. The research results indicate a significant difference between the mean SGOT levels of the control group at 61.16 ± 1.36 U/L and the Kn group at 69.14 ± 0.99 U/L($p < 0.05$), as well as the SGPT levels of the control group at 123.86 ± 2.44 and the Kn group at 138.74 ± 1.09 U/L ($p<0.05$). These findings are consistent with previous studies which reported the mean SGPT levels in normal rats to be 85.6-110.1 U/L and SGOT levels to be 80.2-115 U/L (16).

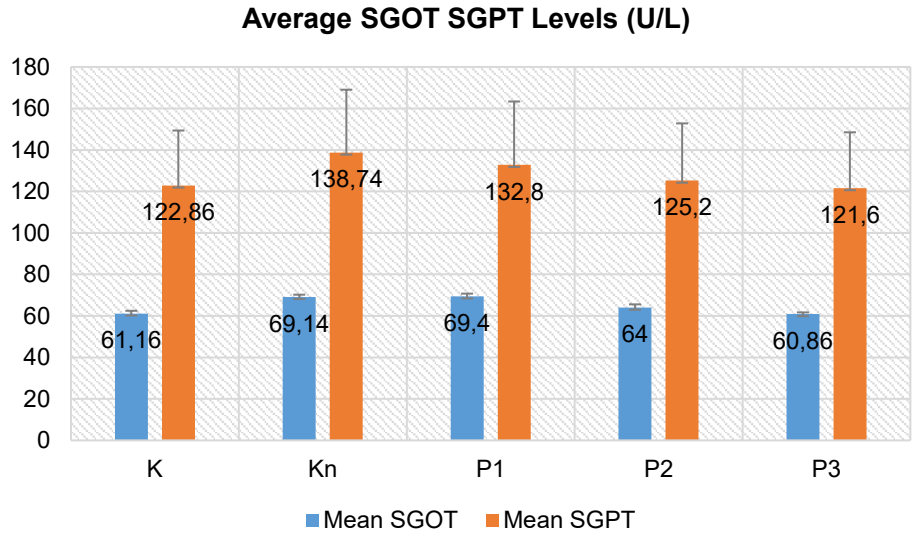

Figure 1. Delta average SGOT levels and SGPT levels

The increase in SGPT levels in the control group given standard feed occurred because physical activity can lead to an increase in SGPT, which is in line with Akbulut's theory stating that any physical activity requires energy derived from food (17). This food is converted into Adenosine Triphosphate (ATP) and used as an energy source. The less the movement activity of the rats, the less food is converted into ATP, resulting in the food intake being stored in the body as cholesterol reserves (17). Excessive cholesterol increase suppresses the production of anti-inflammatory cytokines such as adiponectin and GSH, leading to uncontrolled oxidative stress in the liver (18,19).

Table 2. Statistical data analysis of the effect of coconut milk on SGOT SGPT levels

	df1	F	Sig.
SGOT	4	56.185	0.000
SGPT	4	84.244	

Table 3. *Post hoc LSD* SGPT test

Treatment Groups	K	KN	P1	P2	P3
K		0.000*	0.000*	0.051	0.276
KN	0.000		0.000*	0.000*	0.000*
P1	0.000*	0.000*		0.000*	0.000*
P2	0.051	0.000*	0.000*		0.005*
P3	0.276*	0.000*	0.000*	0.005*	

(*) denotes significant difference (p < 0.05)

One Way ANOVA comparison test was conducted, revealing a significant difference between the control group and the treatment group (p <0.05). After the One Way ANOVA comparison test, post hoc LSD tests were conducted to determine the effect of coconut milk and the significance difference in SGOT and SGPT levels. The post hoc LSD results can be seen in **Tables 2** and **3**. The SGPT levels of the control group given standard feed compared to the negative control group given HFD showed significance of 0.000, meaning that the administration of HFD in the form of duck egg yolks and margarine can increase SGPT levels. The SGPT levels of the negative control group given HFD compared to treatment group 1 given HFD and coconut milk at a dose of 2.5 ml/kgBW/day showed significance of 0.000, indicating that the administration of coconut milk resulted in a decrease in SGPT levels. The SGPT levels of the negative control group given HFD compared to treatment group 2 given HFD and coconut milk at a dose of 5 ml/kgBW/day showed significance of 0.000, meaning that coconut milk administration resulted in a decrease in levels. When comparing SGPT levels between treatment group 1 and treatment group 3, the significance was 0.000, indicating that increasing the dose of coconut milk significantly contributed to the decrease in SGPT levels.

The SGPT levels of the control group given standard feed compared to treatment group 3 given HFD and coconut milk at a dose of 10 ml/kgBW/day showed a decrease in significance of SGPT at 0.276, meaning that high-dose coconut milk administration was able to inhibit the increase in SGPT levels approaching those of the control group. The SGPT levels of treatment group 1 given HFD and coconut milk at a dose of 2.5 ml/kgBW/day compared to treatment group 2 given HFD and coconut milk at a dose of 5 ml/kgBW/day showed significance of 0.000, indicating that increasing the dose of coconut milk had a significant effect on decreasing SGPT levels. When comparing the SGPT levels of treatment group 1 with treatment group 3, the significance was 0.000, indicating that increasing the dose of coconut milk had a significant effect on decreasing SGPT levels.

The post hoc LSD results of SGOT levels **Table 4** showed significance of 0.000 for the control group given standard feed compared to the

negative control group given HFD, meaning that HFD administration in the form of duck egg yolks and margarine could increase SGOT levels. The SGOT levels of the negative control group compared to treatment group 1 showed a significance of 0.744, meaning that the dose of coconut milk at 2.5 ml/kgBW/day was not significant in preventing an increase in SGOT. Comparison of the negative control group given HFD with treatment group 2 given HFD and coconut milk at a dose of 5 ml/kgBW/day showed a decrease in significance with a value of 0.000, indicating that coconut milk administration resulted in a decrease in SGOT levels. Similarly, comparing the negative control group with treatment group P3 showed a significant decrease in SGOT of 0.000, meaning that coconut milk administration resulted in a decrease in SGOT levels. The control group given standard feed compared to treatment group 3 given HFD and coconut milk at a dose of 10 ml/kgBW/day showed significance of SGOT at 0.707, meaning that high-dose coconut milk administration could inhibit the increase in SGOT levels, thus approaching the levels of the control group. Treatment group 1 given HFD and coconut milk at a dose of 2.5 ml/kgBW/day compared to treatment group 2 given HFD and coconut milk at a dose of 5 ml/kgBW/day showed a decrease in significance with a value of 0.000, indicating that increasing the dose of coconut milk had a significant effect on decreasing SGOT levels. When comparing the SGOT levels of treatment group 1 with treatment group 3, the significance was 0.000, indicating that increasing the dose of coconut milk had a significant effect on decreasing SGOT levels.

Table 4. *Post hoc LSD* SGOT test

Treatment Groups	K	KN	P1	P2	P3
K		0.000*	0.000*	0.002*	0.707
KN	0.000*		0.744	0.000*	0.000*
P1	0.000*	0.744		0.000*	0.000*
P2	0.002*	0.000*	0.000*		0.001*
P3	0.707	0.000*	0.000*	0.001*	

(*) denotes significant difference ($p < 0.05$)
*P1 which was given by HFD and 2.5 ml/kgBW/day of coconut milk, P2 given HFD and 5 ml/kgBW/day of coconut milk, and group P3 given HFD and 10 ml/kgBW/day of coconut milk,

The effect of HFD administration has been elucidated in previous studies, showing that administration for 16 weeks has been proven to induce obesity and inflammation in rats, evidenced by increased natural killer (NK) cells and CD8+ cells in the liver, which play a role in NASH (20). HFD administration in rats has also been shown to cause obesity, moderate hyperglycemia,hyperinsulinemia,hypertriglyceridemia, steatosis, beta-cell dysfunction and hypertrophy, as well as insulin resistance in muscles and liver (21). Insulin resistance leads to lipolysis in adipose tissue, increasing fatty acids and glycerol in the liver, thus triggering gluconeogenesis and inducing de novo lipogenesis, resulting in hepatic steatosis (22). After gluconeogenesis occurs, hepatic glucose production increases, leading to systemic insulin resistance (23). Systemic insulin resistance and relatively reduced insulin secretion from pancreatic β cells lead to type 2 diabetes mellitus (24). Insulin resistance is characterized by decreased insulin activity in target tissues due to decreased glucose uptake in fat and muscle tissues, decreased suppression of endogenous glucose production in the liver, decreased suppression of lipolysis in adipose tissue, and decreased glycogen synthesis induced by insulin (22).

When steatosis occurs, the liver signals various other pro-inflammatory activities such as hepatocellular LTβR and canonical NF-κB signaling, facilitating the transition from NASH to hepatocellular carcinoma (HCC) (25). Moreover, hepatic steatosis causes the progression of genetic mutations in Patatin Like Phospholipase Domain Containing 3 (PNPLA3) or specific variations in the PNPLA3 gene. PNPLA3 contributes to lipid storage in the liver (26,27). Steatosis also results in fat accumulation in hepatocytes due to triglyceride deposition caused by lipid metabolism imbalance (26,28). Excessive

fat accumulation leads to increased reactive oxygen species (ROS) through polycyclic aromatic hydrocarbons (PAH), which can disrupt DNA synthesis, resulting in increased oxidative stress burden on various liver enzymes, including SGOT and SGPT (29).

Liver damage can also be caused by massive oxidative stress due to lipid accumulation (18). Excessive expression of cytochrome P40 (CYP2E1), commonly associated with hepatotoxicity, correlates with increased liver damage due to apoptosis (30). The enzyme CYP2E1, working in the liver, forms toxic reactive N-acetyl-p-benzo-quinoneimine (NAPQI). All processes occurring in the liver due to HFD increase cellular activity, leading to lipotoxicity, hepatocyte apoptosis, inflammation activation, and mitochondrial dysfunction contributing to excessive fatty acid accumulation, hepatocellular injury, inflammation, and excessive progressive extracellular matrix accumulation (31). It is known that the Kn group has significantly higher levels of SGOT and SGPT compared to the control group. Meanwhile, the comparison between the control group and the P3 group with a dose of 10 mL/kgBW/day resulted in SGPT significance values of 0.276, SGOT significance values of 0.707. This indicates that the administration of coconut milk at a dose of 10 mL/kgBW/day can reduce SGOT and SGPT levels towards normal values. In other words, coconut milk at a dose of 10 mL/kgBW/day can neutralize the oxidative stress caused by HFD administration.

The difference in the administration of coconut milk doses in groups P1 at 2.5 mL/kgBW/day, P2 at 5 mL/kgBW/day, P3 at 10 mL/kgBW/day resulted in differences in the decrease of SGOT and SGPT levels in rats, with higher doses resulting in greater decreases. This is consistent with research conducted by Elhassaneen (2020) which stated that coconut milk administration at a dose of 500 mL resulted in a higher decrease in SGOT and SGPT levels compared to a dose of 250 mL (14). The highest dose, in group P3 at 10 mL/kgBW/day, resulted in a greater decrease in SGOT and SGPT, with SGOT and SGPT levels approaching those of the control group given standard feed. This could be because the higher the dose of coconut milk given, the more polyphenols, galactomannan, and MCFA content present in the experimental animals' bodies, resulting in a greater decrease in SGOT and SGPT levels. Desty et al. researched that using growol which contains glucomannan can have positive control on glycemic index by the same mechanism galactomannan of coconut decreases SGOT and SGPT (45).

Protection by coconut milk is obtained through its polyphenol, MCFA, and galactomannan content. Polyphenols act as antioxidants, inhibiting XO and transferring electrons and hydrogen to H2O2, thus disrupting free radical formation (6,32). The hepatoprotective mechanism occurs due to cytochrome P450 inhibition, preventing free radical formation. Additionally, polyphenols also play a role in activating various other antioxidants such as SOD and GPX (33). Phenolic compounds contained in coconut milk amount to 816 µg/100 ml, which can be useful as antioxidants (6). Another in vivo study in rats containing 79.3 µg/mg resulted in decreased apoptosis histopathologically due to decreased MAP kinase and cytochrome P-450, as well as decreased lipid peroxidation indicated by low levels of malondialdehyde (MDA) and increased antioxidants such as GSH-Px, SOD, and catalase (34).

The Medium Chain Fatty Acids (MCFA) content in coconut milk also contributes to the decrease in SGOT and SGPT (35–37). MCFAs can be rapidly absorbed by the intestines and directly transported to the liver via the portal vein for metabolism. This is because MCFA absorption into cells does not require transporters and can enter mitochondria without carnitine. The short half-life of MCFAs results in no increase in blood cholesterol levels. MCFAs are more rapidly converted into energy compared to LCFA, preventing triglyceride accumulation in adipose tissue, thus reducing the risk of hepatic steatosis characterized by decreased SGOT and SGPT levels (38–40).

The reduction in cholesterol levels can also be achieved through bile metabolism with the administration of MCFA, which increases the expression of liver X receptor (LXR). The LXR agonists enhance the regulation of ATP-binding cassette sub-family G member 5 (ABCG5) and member 8 (ABCG8), while reducing cholesterol absorption in the small intestine. ABCG5 and

ABCG8 play a role in the formation of sterol transporters in the liver and small intestine to prevent sterol accumulation. ABCG8 also facilitates cholesterol efflux to be transported to the liver and excreted as bile. MCFA increased activity of cytochrome P450 7A1 (CYP7A1) and P450 27A1 (CYP27A1) by reducing the expression of small heterodimer partner (SHP) in the liver, thus leading to increased synthesis and excretion of bile acids (40). The acidic nature of MCFA can also reduce cholesterol in artery walls, thereby preventing lipid peroxidation in LDL and arteriosclerosis (8). It has been reported that treatment with MCFA less than 200 µM does not cause clear toxicity to liver cells (41). Furthermore, MCFA does not induce apoptosis, oxidative stress, and inflammation in steatotic liver cells (42). MCFA acts on the surface receptor GPR84, which plays a role in granulocytes (43). GPR84 regulates myeloid cells during inflammation through the signaling of chemokine CCL2 and chemokine receptor CCR2. When myeloid infiltration occurs in hepatocytes, it leads to fibrosis and steatosis.

Galactomannan in coconut milk directly affects SGPT and SGOT levels. Galactomannan can affect liver health in several ways, including altering SGPT and SGOT levels, through Nrf2 regulation activation, resulting in increased antioxidant levels such as GSH, GPx, SOD, and glutathione-reductase (GRx), thus maintaining the balance of oxidant-antioxidant which manifests in inhibiting ROS-mediated liver damage This is consistent with previous research that a 0.05% galactomannan content can reduce SGOT, SGPT, and alkaline phosphatase (ALP) levels (44). The growol which contains glucomannan can have positive control on glycemic index by maintaining oxidant-antioxidant and inhibit ROS like mechanism galactomannan of coconut decrease SGOT and SGPT (45). Another mechanism involved in reducing SGOT and SGPT levels is increased detoxification due to the galactomannan content containing diosgenin and 4-hydroxyisoleucine, which mediate lipid-lowering effects by reducing serum triglyceride synthesis, increasing HDL levels in circulation, and suppressing HepG2 accumulation in lipid steatosis with HFD administration, reducing SREBP1c synthesis, and stimulating PPARα. SREBP1c and PPARα are two transcription factors that promote triglyceride synthesis (46). Chenxuan et al. 2019 found the effect of Hepatoprotective Guar Gum that contains galactomannan, against Acute Alcohol-Induced Liver Injury and reduced SGOT and SGPT(47).

CONCLUSION AND RECOMMENDATION

This study demonstrates that coconut milk administration can protect the liver from damage caused by HFD consumption. Although HFD administration for 45 days resulted in a significant increase in SGOT and SGPT levels, a dose of coconut milk at 10ml/kgBW was able to inhibit this increase. The study suggests expanding the focus on the effects of coconut milk on SGOT and SGPT levels by considering processed coconut milk in cooking and establishing relevant health indicators for the community. Furthermore, further research should take into account other factors such as dyslipidemia. Liver biopsy examinations in rats should also be included to confirm hepatic steatosis. Additionally, further research is needed to determine the effective dose of coconut milk, including doses higher than 10ml/kgBW. This will provide more comprehensive insights and more accurate recommendations regarding the effects of coconut milk on liver health.

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