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THE EFFECT OF A HIGH-FAT DIET ON LIPID PROFILE LEVELS (LDL CHOLESTEROL, HDL CHOLESTEROL, TRIGLYCERIDES, AND TOTAL CHOLESTEROL) IN MALE RATTUS NOVERGICUS WITH OBESE

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ABSTRACT

Obesity is a complex disease that has a significant impact on public health and its prevalence is increasing worldwide. Obesity causes lipid accumulation in adipose tissue. The purpose of this study was to determine the effect of a high-fat diet on lipid profiles in male white rats. This study used a true experimental study on male white rats with a post-test control-only group design. The group division in this study was divided into two groups, consisting of a negative control group (K1) of 5 rats and a HFD group (K2) of 20 rats. This study was conducted from October 2024 to April 2025 at the Biomedical and Biochemistry Laboratory, Faculty of Medicine, Andalas University. The population in this study were male white rats of the Rattus norvegicus strain wistar obtained from the Biomedical Laboratory, Faculty of Medicine, Andalas University. The sampling technique in this study used purposive sampling. The number of samples used in this study was 25 rats with a minimum reserve of 10%. The results of the study with the average values of LDL cholesterol, HDL cholesterol, Total cholesterol, and TG levels in the K1 group were 45.72, 62.28, 67.66, and 107.60 mg/dL, respectively, while in the K2 group they were 61.62, 43.14, 83.17, and 167.20 mg/dL, respectively. Based on the results of the independent T test, the significance of the two groups was 0.001, 0.001, 0.001, and 0.005, respectively, which means there was a significant or real difference in the average levels of LDL, HDL, total cholesterol, and triglycerides in the K1 and K2 groups.

Keywords: cholesterol;HDL;LDL; lipid profile; obesity; rattus novergicus; triglycerides

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INTRODUCTION

Obesity is a complex disease that impacts public health and is increasing in prevalence worldwide (Perez et al., 2021). According to World Health Organization (WHO) data, by 2024, the global obesity rate will exceed 1.9 billion, comprising 650 million adults, 340 million adolescents, and 39 million children. Indonesia has a very high annual obesity rate, at 3.9 percent, ranking first in Southeast Asia. According to data from the Indonesian Obesity Association, West Sumatra has seen an annual increase of 2% in obesity cases over the past four years.

Obesity causes lipid accumulation in adipose tissue. Lipids are broken down into fatty acids and glycerol during lipolysis, which can be oxidized to provide energy (Bansal and Cassagnol, 2024). Cholesterol synthesis occurs through the breakdown of carbohydrates, proteins, or fats in the form of acetyl-CoA (Aziz et al., 2020; Oktavelia and Kusuma, 2022). This risk can lead to metabolic syndrome, which can lead to dyslipidemia. Dyslipidemia causes impaired lipoprotein metabolism, characterized by increased total cholesterol, increased low-density lipoprotein (LDL) cholesterol, increased triglycerides, and decreased high-density lipoprotein (HDL) cholesterol (Huang et al., 2023; Yuniarifa et al., 2021).

A high-fat diet (HFD) is a diet high in fat that affects digestive system metabolism and can lead to obesity. A HFD can also lead to metabolic syndrome, including heart disease, dyslipidemia, hypertension, type 2 diabetes mellitus, cancer, non-alcoholic fatty liver disease (NAFLD), and others. This metabolic syndrome impacts quality of life, productivity, mental health, and healthcare costs in individuals with these conditions. Based on the above description, researchers have conducted a study on the effect of a high-fat diet on lipid profiles in male white rats (Rattus norvegicus strain wistar).

METHOD

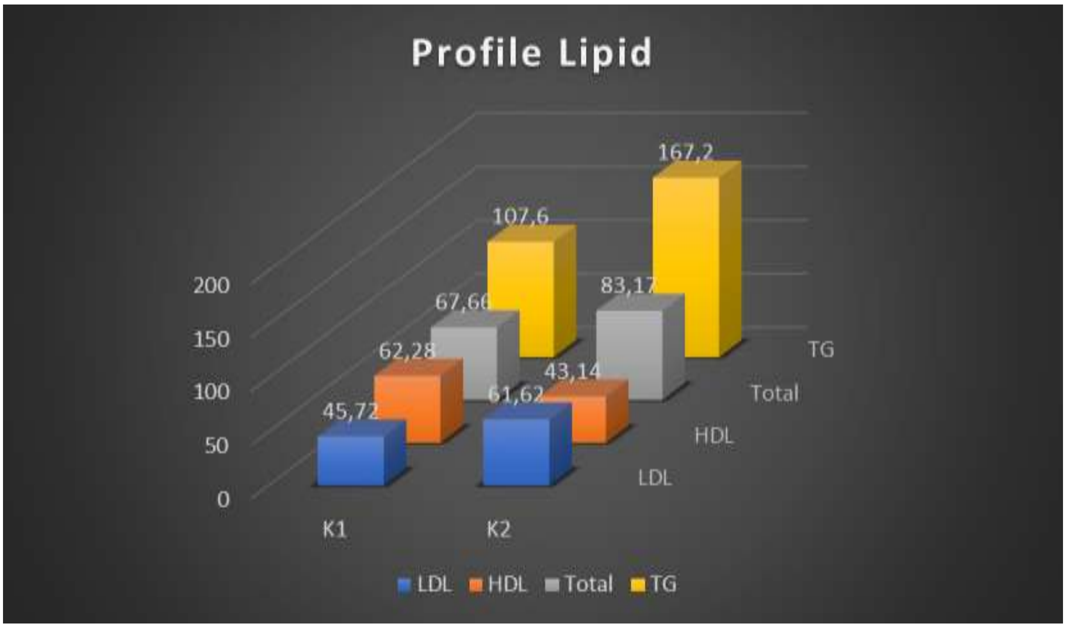
This study used a true experimental study on male white rats (Rattus norvegicus strain Wistar) using a post-test control-only group design. Data were collected after the treatment (experiment) was administered. The study was conducted by comparing the observation results in the experimental and control groups. The study group was divided into two groups: a negative control group (K1) of 5 rats and a HFD group (K2) of 20 rats. This study was conducted from October 2024 to April 2025 in the Biomedical and Biochemistry Laboratories of the Faculty of Medicine, Andalas University. The study population consisted of healthy, normally active male white rats (Rattus norvegicus strain Wistar), aged 2 months and weighing 200 grams, obtained from the Biomedical Laboratory, Faculty of Medicine, Andalas University. The sample size used in this study was calculated using the Federer formula, obtaining a minimum sample size of 25 rats and a minimum reserve of 10%.

The research materials used were standard feed (Biomedical Lab, Faculty of Medicine, Andalas University) and high-fat feed, namely goat oil and egg yolk. Reagent Kits for LDL cholesterol, HDL cholesterol, total cholesterol, and triglycerides. Mice adapted for 7 days, then continued with a high-fat diet for 3 months for K2 mice and a normal diet for K1 mice. Mice were fasted for 8-12 hours before blood sampling. 2 ml of mouse blood was taken and then centrifuged to separate plasma and serum. Lipid profile examinations such as LDL cholesterol, HDL cholesterol, total cholesterol, and triglycerides were carried out using enzymatic methods and spectrophotometry tools according to the procedures listed in the Kit. Hypothesis testing used the Independent t-test to analyze data in the control group and the treatment group for each group. The t-test is a test to compare mean differences between two groups (Kimura et al., 2017; Yuli et al., 2020). Descreption of ethical approval number 520/UN.16.2/KEP.FK/2024.

RESULT

Table 1.
Mean and Standard Deviation was of LDL, HDL, total cholesterol, and triglycerides of Levels

Group	N	Levels (X±SD)		mg/dL	
		LDL	HDL	Total	TG
K1	5	45.72±5.99	62.28±8.57	67.66±1.84	107.60±34.16
K2	20	61.62±9.26	43.14±6.47	83.17±9.34	167.20±39.39
Sig		0.001	0.001	0.001	0.005



Picture 1. Lipid Profile Level

Based on table 1, it can be seen that the average levels of LDL cholesterol, HDL cholesterol, Total cholesterol, and TG in group K1 were 45.72, 62.28, 67.66, and 107.60 mg/dL, respectively, while in group K2 they were 61.62, 43.14, 83.17, and 167.20 mg/dL, respectively. Based on the results of the independent T test, the significance of the two groups was 0.001, 0.001, 0.001, and 0.005, respectively, which means there was a significant or real difference in the average levels of LDL, HDL, total cholesterol, and triglycerides in group K1 and group K2.

DISCUSSION

The mice were divided into two groups, the K1 negative control group with a normal diet and the K2 high-fat diet with goat fat 2 ml/200 grams of mouse body weight and egg yolk 1 ml/200 grams of mouse body weight. The fat given consisted of goat fat and egg yolk by means of a tube for 3 months. Cut's research in 2022, a total of 31 male Wistar rats weighing 150-250 grams aged 4-5 months with a HFD diet for 50 days with a daily fat content of 66.28%, the results showed a difference in triglyceride levels with a P value of 0.030 and HDL in the negative control group with the treatment group with a P value of 0.011. Sukmawati's 2017 research, on 15 Rattus Novergicus mice aged 3-4 months and weighing 150-200 mg were given a high-cholesterol diet by mixing a dose of 200 mg/BW cholesterol, 1 ml/200 BW egg yolk, 10% coconut oil plus distilled water for 7 days, with the results of the study showing an increase in cholesterol to 101.0 mg/dL. Teuku Heriansyah's 2013 research on 24 Rattus Novergicus mice weighing 50-100 grams and aged 2 months with various durations of 8, 16 and 22 weeks of HFD diet found an increase in TG, LDL levels, and a decrease in HDL. Isdadiyanto's research on male Rattus Novergicus mice with a 30 gram/day HFD diet and 2.5 ml/200 gram BW duck egg yolk for 32 days with the results of the study showing an increase in cholesterol, triglycerides, LDL and a decrease in HDL levels.

Kurniawati's research in 2022, the research sample of 30 Wistar rats aged 2-3 months with a HFD diet for 56 days as much as 30 grams / rat / day with a composition of 55% rat bio, 10% margarine, 20% beef fat, and 15% coconut milk powder with the results of the study there was an increase in total cholesterol levels after the HFD diet of 2.7% mg / dL. Dewi Leona et al's research (2021) on male Rattus Novergicus rats weighing 125-150 grams using a categonic diet obtained research results of a decrease in cholesterol, TG and LDL levels and an increase in HDL. Febriyatna Ayu's research on 2-month-old Rattus Novergicus rats weighing 150-200 grams with a HFD diet of 25% margarine, 5% beef brain, 15% coconut cream powder for 9 weeks obtained results of HDL control group 30.27 mg / dL higher than the treatment group 26.32 mg / dL. Gani's 2013 study, involving 16 male Wistar rats, aged 2-3 months and weighing 120-250 grams, fed a high-density lipoprotein (HFD) diet for 14 days, containing 100 grams of goat fat (10%), 50 grams of egg yolk (5%) in 1,000 grams of standard feed, revealed increased levels of total cholesterol, LDL, and triglycerides in the HFD-fed rats compared to control rats.

Lipids are carried in plasma by lipoproteins, which have an external hydrophilic region containing proteins, phospholipids, and non-sterile cholesterol, and an internal hydrophobic core containing triacylglycerols (TAGs) and cholesterol esters. The fat circulating in the body comes from two sources: dietary intake and liver production, which can be stored in fat cells as an energy reserve (Hafidz et al., 2017). Fats found in food are broken down into cholesterol, triglycerides, phospholipids, and free fatty acids during digestion in the intestine (Jiang et al., 2018). These four fats are absorbed from the intestine and enter the bloodstream. Fats are insoluble in water, meaning they are also insoluble in blood plasma. To transport fats into the bloodstream, they bind to specific proteins in the plasma, forming a water-soluble macromolecular complex. This bond between fats (cholesterol, triglycerides, and phospholipids) and proteins is called a lipoprotein (Nurmawati et al., 2021; Yu et al., 2022).

A lipid profile is a snapshot of lipid levels in the blood (Yuliadewi NS and Arimbawa, 2020). Based on their composition, density, and mobility, lipoproteins are divided into chylomicrons, very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) (Siregar and Makmur, 2020; Li et al., 2023). Cholesterol synthesis uses acetyl-CoA, a raw material derived from the breakdown of carbohydrates, proteins, or fats. Cholesterol formation occurs through an enzymatic reaction in the mevalonate pathway, where the enzyme HMG-CoA reductase catalyzes the conversion of HMG-CoA to mevalonate, which is necessary for cholesterol biosynthesis (Aziz et al., 2020; Oktavelia and Kusuma, 2022).

The fatty foods we eat consist of triglycerides and cholesterol (Manalu et al., 2021). Triglycerides and cholesterol are absorbed into the enterocytes of the small intestine (Rahman et al., 2014). Triglycerides are absorbed as free fatty acids, while cholesterol is absorbed as cholesterol (Sumertayasa et al., 2020). Free fatty acids in the small intestine are converted into triglycerides, while cholesterol undergoes esterification into cholesterol sterol ester (Sun et al., 2018). Together with phospholipids and apolipoproteins, these two form large lipoprotein particles called chylomicrons (Villano et al., 2023). These chylomicrons carry them into the bloodstream. Triglycerides in chylomicrons are broken down by the endothelial-derived enzyme lipoprotein lipase, resulting in the formation of free fatty acids and chylomicron remnants (Maigoda et al., 2016; Kim et al., 2017).

Triglycerides and cholesterol synthesized by the liver are transported endogenously in the form of VLDL (Yuniarifa et al., 2021; Laffin et al., 2023). VLDL undergoes hydrolysis in circulation by lipoprotein lipase, which also hydrolyzes chylomicrons into IDL (Lestari and Santika, 2023). IDL particles are then taken up by the liver and further broken down into the final product, LDL (Indriputri and Maulana, 2019). LDL is taken up by LDL receptors in the liver and undergoes catabolism. This LDL is responsible for transporting cholesterol throughout the body. HDL originates from the liver and intestines during chylomicron hydrolysis under the influence of the LCAT enzyme (Tsalissavrina et al., 2006; Xu et al., 2018). These cholesterol esters are transferred from HDL to VLDL and IDL, thus reversing the direction of cholesterol transport from the periphery to the liver. This activity may contribute to its anti-hypertensive properties (Mohammadi-Shemirani et al., 2020).

HDL is released as small, cholesterol-rich particles containing apolipoproteins (apo) A, C, and E, called nascent HDL. Nascent HDL originates from the small intestine and liver, is flattened, and contains apolipoprotein A1 (Khoo and Aziz, 2009; Xu et al., 2018). Nascent HDL migrates to macrophages to take up the cholesterol stored in them. After taking up cholesterol from macrophages, nascent HDL transforms into mature, spherical HDL (Sumertayasa et al., 2020).

Based on the results of the examination and analysis using the Independent T-test hypothesis test, in the K2 group there was an increase in total cholesterol levels in the blood with a difference of 15.51 mg / dL and a significance of 0.001, LDL cholesterol in the blood with a difference of 15.90 and a significance of 0.001, HDL cholesterol in the blood with a difference of 19.14 and a significance of 0.001 and triglycerides in the blood with a difference of 59.60 and a significance of 0.005 This occurs because the feed given contains a lot of goat oil fat (66.6%) and egg yolk (33.3%) with triglyceride, cholesterol, and phospholipid content of 62%, 33%, 5%, respectively. After the physicochemical process in the body, triglycerides are converted into free fatty acids and are then used as energy stored in adipocyte cells as energy reserves. In addition, triglycerides can migrate to the liver to be resynthesized into VLDL, IDL and LDL. Cholesterol in food is absorbed by the small intestine in the form of chylomicrons, then enters the lymphatic system and is transported into the bloodstream, migrating to the liver for synthesis into VLDL and LDL, which are then transported to the tissues. HDL collects excess cholesterol in peripheral tissues and macrophages, while HDL transports cholesterol back to the liver. Cholesterol is used to synthesize bile acids and is excreted in the bile through the feces. The higher the dose and the longer the HFD is administered, the higher the lipid profile in male white rats. Increased cholesterol, triglycerides, and LDL levels, while decreased HDL levels are directly related to high-fat diet consumption.

CONCLUSION

Based on the results of the research conducted, a high-fat diet has an effect on lipid profile levels.

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