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The Effect of Carnitine on Reducing Triglyceride Levels

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Abstract
Carnitine is a crucial compound involved in the transport of long-chain fatty acids across mitochondrial membranes, playing an essential role in converting fat into energy. One of the most accessible ways to increase L-carnitine levels is through the consumption of red meat, which is a more affordable alternative compared to supplements. However, there is limited information on the optimal amount of red meat required to reduce blood triglyceride levels. This study aims to compare the effects of 50 mg and 100 mg of L-carnitine derived from red meat on blood triglyceride levels. The research utilized a quantitative approach with a quasi-experimental design. Participants included individuals aged 30-60 years with high triglyceride levels, who consumed beef did not take anti-cholesterol medications or had a history of diabetes. They also agreed to provide blood samples after an 8-9 hour fast. A simple random sampling method was used, with the first and odd-numbered participants assigned to the 100 mg group, and the second and even-numbered participants assigned to the 50 mg group. A total of 38 respondents were involved, with 19 in each group. The results indicated that the mean rank for the 100 mg carnitine group (17.32) was lower than that of the 50 mg group (21.68), though statistical analysis revealed no significant difference in triglyceride reduction between the two doses. The conclusion is the 50 mg carnitine group exhibited a higher increase in triglyceride levels compared to the 100 mg group. These findings suggest that a 12-day intervention with 100 mg of carnitine may prevent an increase in blood triglycerides, while 50 mg may not have the same effect.

Keywords: Carnitine, Beef, Triglycerides.

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1. INTRODUCTION

Low-density lipoprotein (LDL) cholesterol is one of the leading causes of cardiovascular disease. This occurs when LDL cholesterol binds to oxygen in plasma or the subendothelial space of the artery wall, causing various events that result in increased use of LDL cholesterol on receptors in macrophages (Hasanah et al., 2020; Ikawati et al., 2019). Oxygen binding (oxidation) reaction by LDL cholesterol causes muscle cells to form foam cells. Increased oxidation of LDL cholesterol results in increased conversion of arterial monocytes or macrophages into foam cells which are the main ingredients for the formation of atherosclerotic plaques (Laguna-Fernandez et al., 2018; Wang et al., 2020).

Oxidation of LDL cholesterol occurs in various atherosclerotic lesions, producing various additional compounds that are dangerous, such as disorders of the blood vessel walls, which result in inhibition of the vasodilation process, which occurs in hypertensive patients (Kalma et al., 2021; Sposito et al., 2019). Oxidation of LDL cholesterol occurs in various atherosclerotic lesions, producing various additional compounds that are dangerous, such as disorders of the blood vessel walls, which result in inhibition of the vasodilation process, which occurs in hypertensive patients (Cheng et al., 2017; Taylor et al., 2024). To avoid the adverse effects of LDL cholesterol oxidation, the body forms a compound that can reduce blood levels of triglycerides known as carnitine. Carnitine is a molecule that plays a role in transporting fatty acids into the mitochondria for metabolic processes or energy burning. Carnitine itself is a compound that is produced from the body naturally. It is composed of two essential amino acids, namely lysine and methionine. Its primary function is the transport of fatty acids into energy through beta-oxidation. Carnitine also plays a role in the detoxification process and nervous system function. L-carnitine can help lower triglyceride levels in the blood by increasing the burning of fatty acids as an energy source (Lee et al., 2016).

L-carnitine has a significant role as a transport of fatty acids with a long chain to cross the mitochondrial membrane so that it can be continued with β oxidation and ATP production through subsequent oxidative phosphorylation. Mitochondria are the central storehouses that produce energy within the body's cells, which the body needs for normal function (Zare et al., 2015). Energy is generated when fat is burned inside the mitochondria, and mitochondria need oxygen to burn fat (Gnoni et al., 2020). It also translocates acetyl-CoA into the cytoplasm while acetyl-L-carnitine is transported out of the mitochondria. Thus, L-carnitine may restore the high-energy phosphate pools in the myocardium and other cell types (Cheng et al., 2017; Gnoni et al., 2020; Tackling & Borhade, 2021).

Based on previous research conducted on test animals, it was found that L-carnitine prevented the development of atherosclerosis lesions in hypercholesterolemic rabbits. L-carnitine blocks the development of atherosclerosis lesions due to its antioxidant and lipid-lowering effects (Dunning & Robker, 2012). Another research result is the study of Ulfa, (2015) on male white rats of the Wistar strain; as many as 24 rats divided into four groups, namely Group 1 (standard feed AD II and aquades), Group 2 (standard feed AD II, aquades and L-carnitine 18 mg), Group 3 (standard feed AD II, aquades and L-carnitine 36 mg) and Group 4 (standard feed AD II, aquades and L-carnitine 54 mg). All treatments are for ten days. The mean result of reduction after triglyceride level treatment in Group 1: 89.55 mg/dl, Group 2: 85.13 mg/dl, Group 3: 80.84 mg/dl, and Group 4: 73.40 mg/dl (Ulfa, 2015).

L-carnitine has benefits for preventing or reducing the risk of cardiovascular disease as assessed by plasma lipid and lipoprotein levels. Carnitine consumption can also shift the liver's metabolic bias from esterification and triglyceride synthesis to acetylcarnitine formation (Dambrova & Liepinsh, 2015). It may decrease the synthesis of triglycerides and VLDL cholesterol and possibly increase the β-oxidation of fatty acid mitochondria. Studies supporting this hypothesis show that L-carnitine lowers serum cholesterol, triglycerides, free fatty acids, and HDL cholesterol; however, in another study, it was found that carnitine did not affect triglycerides, total cholesterol, and LDL cholesterol (Jiang et al., 2020; Taylor et al., 2024).

Research Results of Ni Wayan Sukma Antari et al. (2013) Administration of L-carnitine in male rats for 42 days with dosage variations of 100 mg/kg bb, 150 mg/kg bb, and 200 mg/kg bb. The results showed that administering L-carnitine supplements in high doses for a long time can decrease spermatozoa quality, namely morphology, motility, viability, and membrane integrity (Antari & Damayanti, 2020).

L-carnitine is generally produced endogenously by the liver and kidneys but at low levels. It can also be obtained from outside the body, namely through food and supplements. L-carnitine that comes from food is often found in red meat, dairy products, and fish. Meanwhile, many supplements are available in packages with certain dosages and are widely used by athletes (Jiang et al., 2020).

L-carnitine is useful for building muscle and increasing energy. In people with heavy physical activity, it has an impact on reducing L-carnitine levels in the body, so the body needs enough L-carnitine to keep muscles strong and healthy (Sepehrvand & Ezekowitz, 2016). Taking L-carnitine is very safe. The benefits of L-carnitine are that it helps burn fat to get extra energy and an ideal body shape, increases HDL (good cholesterol), and increases body stamina (Lee et al., 2016).

To increase carnitine levels in the body, it is necessary to take actions that can trigger the body to produce carnitine. One of them is consuming foodstuffs that contain carnitine, such as red meat, milk, poultry, fish, etc. Consuming red meat is one of the most likely options for increasing L-carnitine levels in society, compared to using more expensive supplements. However, no reference can explain how much red meat can be consumed to lower triglyceride levels. The study aims to compare the effect of L-carnitine in 50 mg red meat and 100 mg red meat on reducing triglyceride levels in the blood.

2. RESEARCH METHOD

This study is a quantitative study with a quasi-experimental design of 2 treatment groups using the pretest-posttest group design approach. This study measured the effects of L-carnitine in 50 mg red meat and 100 mg carnitine on reducing triglyceride levels in blood. This research was conducted between December 2022 and November 2023.

The population of this study was all residents of RT/RW 02/02 Buaran Jati Village, Sukadiri Subdistrict, Tangerang Regency, who had high triglyceride levels aged 50-65 years. The number of samples was calculated based on the experimental sample formula; the total sample of 38 people was divided into two groups: the carnitine 50 mg intervention group and the carnitine 100 mg group. The sampling method uses a simple random sample. The initials of the respondent's name or identity are entered into a closed box and then drawn randomly. The identity that comes out first and the odd number immediately enters the 100 mg group, and the second and even numbers enter the 50 mg group. A total of 38 participants were given intervention based on groups. The process of grouping and collecting data by group can be seen in Figure 1.

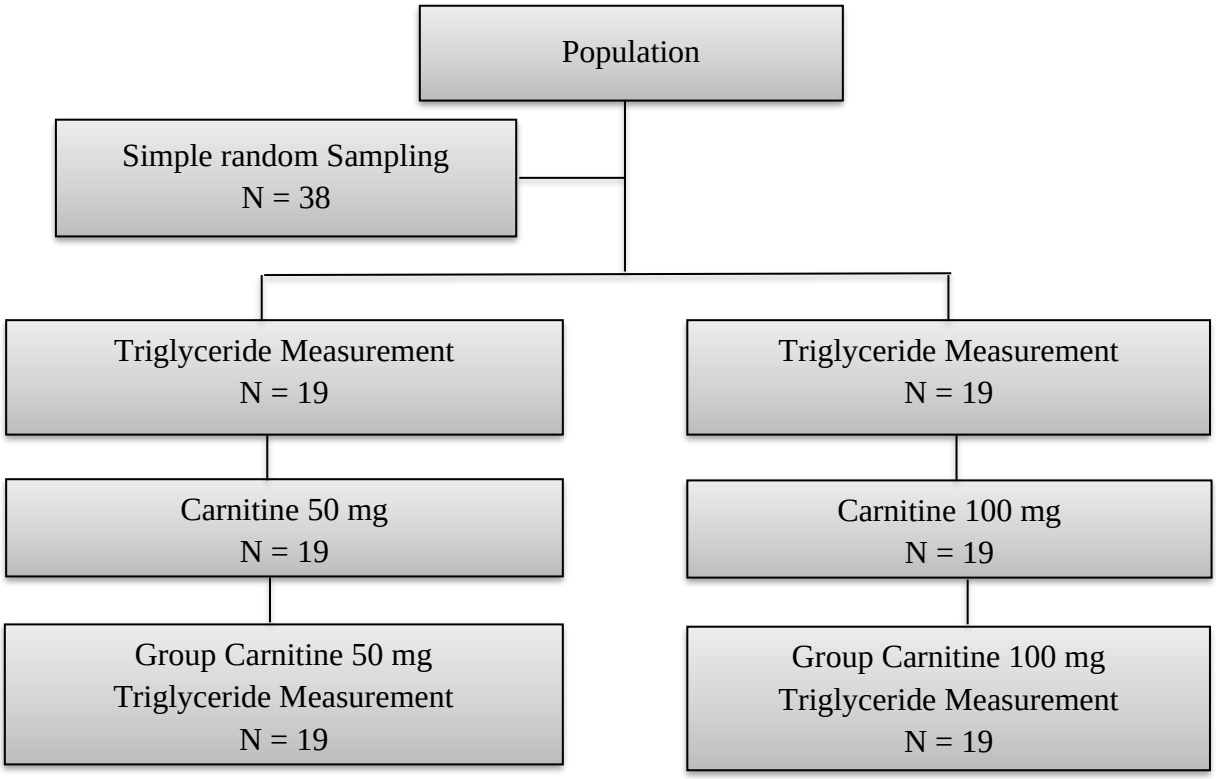

Figure1. Experimental research design

The sample inclusion criteria in this study were having triglyceride levels >150 - 300, being willing to do blood tests before and after being given carnitine, and consuming beef in meatballs as a source of carnitine during the study. Exclusion criteria in this study were taking anticholesterol drugs, having diabetes mellitus, and being willing not to consume animal protein during the study. Carnitine is given in the form of beef-based meatballs. Each meatball contained 25 mg of carnitine. The 100 mg carnitine group received four meatballs, and the 50 mg group received two. The intervention was conducted for 12 days at lunchtime. The measurement tool used in this study was to assess triglyceride levels in the blood and Medical laboratory personnel take blood samples in the arm of the median cubital vein, which was examined in the clinic laboratory. Measurement of triglyceride levels was carried out the day before the intervention was given and the day after the intervention was given.

Data collection was carried out before and after the intervention. Data screening is carried out to ensure that pre and post-triglyceride level data is entered into the computer. The data were encoded for analysis using SPSS version 25.0. As a result, the study was complete, and no respondents came out of the research process.

Statistical tests were conducted in response to the research hypothesis, displaying univariate and bivariate data. Before conducting a statistical test, a data normality test is conducted to determine the distribution of data among respondents. The results of the data normality test using Shapiro-Wilk were found to have abnormal data distribution, so the bivariate analysis used the Wilcoxon test and the Mann U Whitney test.

The researcher conducted a descriptive analysis to describe the characteristics of the respondents and the variables of fear of hypoglycemia. Inferential analysis was carried out to determine the effect of L-carnitine red meat 50 mg and 100 mg interventions. The Wilcoxon sign-rank test was used to analyze the results of the pre-test and post-test of each group because the data of each group was not normally distributed (p-value $< \alpha = 0.05$). In each group, the distribution was normal ($p > \alpha = 0.05$). Therefore, the differences between the treatment and

control groups were analyzed using an independent t-test to compare the pre-test and post-test results of each group.

This research has been conducted an ethical review and approved by the ethics committee of the Banten Ministry of Health Polytechnic with reference number 03/KEPK/EC/XII/2022. Before conducting the research, the researcher provided information to prospective respondents about general matters. Description of the research. In addition, the researcher explained the objectives, benefits, procedures, potential risks, and rewards for participation. Prospective respondents who agree to participate are required to sign a consent form that has been prepared by the researcher.

3. RESULTS AND DISCUSSION

Table 1. Test Normality Group 50 mg

Variable	N	Mean	Standard Deviasi	Sig.
Pre 50	19	197.42	47.4	0.010
Post 50	19	213.68	42.7	0.005
Mean Difference		16,26		

Table 1 shows that Shapiro-Wilk normality test, on variables before administration (pre) carnitine 50 mg, mean triglycerides in the blood 197.42 mg/dl, Standard Deviation 47.4 mg/dl, Significance Value (Sig.) 0.010 and variables after administration (post) carnitine 50 mg, average (Mean) triglycerides in the blood 213.68 mg/dl. Standard Deviation 42.7 mg/dl, Significance value (Sig.) 0.005. In the variable before administration (pre), carnitine 50 mg significance value (Sig) 0.010 < 0.05. This suggests that the data did not follow the normal distribution at a significance level of 0.05. In the variables after administration (post), carnitine 50 mg significance value (Sig) 0.005 < 0.05. This suggests that the data did not follow the normal distribution at a significance level of 0.05.

Table 2. Test Normality Group 100 mg

Variable	N	Mean	Standard Deviasi	Sig.
Pre 100	19	174.16	25.9	0.002
Post 100	19	193.89	35.6	0.468
Mean Difference		19,73		

Table 2 shows that the number of respondents in the variable before administration (pre) Carnitine 100 mg or variable after administration (post) Carnitine 100 mg is 19. Because 19 respondents were less < 50 people, the normality test used Shapiro–Wilk. In the Shapiro-Wilk normality test, on the variable before administration (pre) carnitine 100 mg, the mean (Mean) triglyceride in the blood 174.16 mg/dl, Standard Deviation 25.9 mg/dl, Significance Value (Sig.) 0.002 and the variable after administration (post) carnitine 100 mg, the average (Mean) triglyceride in the blood 193.83 mg/dl. Standard Deviation 35.6 mg/dl, Significance value (Sig.) 0.468. In the variable before administration (pre), carnitine 100 mg significance value (Sig) 0.002 < 0.05. this showed that the data did not follow the normal distribution at a significance level 0.05. In the post-administered (post) carnitine 100 mg variable, the significance value (Sig) of 0.468 > 0.05. this showed that the data followed the normal distribution at a significance level of 0.05 in the normality test if one of the data variables did not follow the normal distribution and the respondent < 50. So, the Wilcoxon dependent test was carried out.

Table 3. The Average Difference in the Value of Pre-Test and Post-Test Triglyceride Level Results After Giving 50 mg Carnitine Beef Meatballs (n = 19)

Intervention	Category Rank	N	Mean Rank	Rank	p-value
Post 50 Mg < Pre 50 Mg	Negative Ranks	5	7.8	39.00	0.043
Post 50 Mg > Pre 50 Mg	Positive Ranks	13	10.15	132.00	
Post 50 Mg = Pre 50 Mg	Ties	1			
	Total	19			

Table 3 shows that After the intervention of 50 mg carnitine. 5 respondents experienced a decrease in triglyceride levels, 13 experienced an increase in triglyceride levels, and one respondent did not experience a change in triglyceride levels. Wilcoxon's statistical test compared two different variables, namely comparing the variable of results after administration (post) of carnitine 50 mg with the variable before administration (pre) of carnitine 50 mg. The statistical test p-value = 0.043<0.05 shows a significant difference between the variables after treatment and before treatment on the average increase in triglycerides.

Table 4. The Average Difference in the Value of Pre-Test and Post-Test Triglyceride Level Results After Being Given 100 mg Carnitine Beef Meatballs (n = 19)

Intervention	Category Rank	N	Mean Rank	Sum Rank	p-value
Post 100mg < pre 100mg	Negative Ranks	3	9.33	28.00	0.007
Post 100mg > pre 100mg	Positive Ranks	16	10.13	162.00	
Post 100mg = pre 100mg	Ties	0			
	Total	19			

Table 4 shows that after the 100 mg carnitine intervention, three respondents had decreased triglyceride levels, 16 had elevated triglyceride levels, and none had the same triglyceride levels during *the pre-test* and *post-test*. Wilcoxon statistical test to compare two different variables, namely the variable after administration (Post) carnitine 100mg with the variable before administration (Pre) carnitine 100 mg. The statistical test p-value 0.007 < 0.05 shows a significant difference in average triglycerides between the variables after treatment and before treatment of the average increase in triglycerides..

Table 5. The difference between the average 50 mg carnitine group and the 100 mg carnitine average group

Intervention	N	Mean Rank	Man Whitney U	Z	p-value
Group 50 mg	19	21.68	139.0	-1.212	0.226
Group 100 mg	19	17.32			

Table 5 shows the results of statistical tests using the Man-Whitney test to determine the median difference in triglyceride levels between the 50 mg carnitine beef meatball feeding group and the 100 mg carnitine beef meatball feeding group. In the Man Whitney p-value test, 0.226 > 0.05. This showed no significant difference between the 50 mg carnitine group and the 100 mg carnitine group on the reduction of triglycerides and the increase in triglyceride levels in the blood. The Mean Rank of the 100 mg carnitine group is 17.32, lower than that of the 50mg carnitine group of 21.68.

DISCUSSION

Based on table 5. In the Man Whitney test, the P-value was 0.226 >0.05. This showed no significant difference between the 50 mg carnitine group and the 100 mg carnitine group on the

decrease and increase in blood triglyceride levels, meaning that although there was a difference between the mean triglyceride (pre-post) of 50 mg carnitine and the mean triglyceride (pre-post) of 100 mg carnitine, there was no significant average increase in triglycerides in 50 mg carnitine and 100 mg carnitine.

The difference in the mean triglycerides (pre-post) from the 100 mg carnitine group to the 50 mg carnitine group (19.73 mg/dl - 16.26 mg/dl) was 3.47 mg/dl. In addition, the Average Rating of the 100 mg carnitine group was 17.32, lower than the Average Rating of 21.68 from the 50 mg carnitine group. This suggests that 100 mg carnitine treatment can suppress the increase in triglycerides on average. This study's results align with a study conducted on 47 patients with Coronary Artery Disease (CAD), 24 of whom were placebo interventions. Another 23 patients who were given L-carnitine as much as 1000 mg/day for 12 weeks showed a significant increase in High-Density Lipoprotein (HDL) and a slight decrease in triglyceride levels (Lee et al., 2016).

Daily administration of l-carnitine and carbohydrates for twelve weeks in humans increases carbohydrate content because the presence of carnitine increases fatty acid metabolism in the body so that fat mass in the body decreases (Dambrova & Liepinsh, 2015; Stephens et al., 2013)**.** Carnitine carries long-chain fatty acid acyl groups through the mitochondria membrane. So fatty acids can be broken down by mitochondria by a beta-oxidation reaction, converting them into acetate to produce energy in the Krebs cycle reaction.

Before being carried by carnitine, fatty acids must first be activated (Ferreira & McKenna, 2017; Shahreza et al., 2023). This process goes through three main stages. The first stage is the formation of acyl-Co-A derived from fatty acids. At this stage, the fatty acids and coenzyme A (Co-A) are attached through thioester bonds. This reaction is catalyzed by the enzyme synthetase acetyl-Co-A fat and completed with the help of the enzyme pyrophosphatase to form the asil-Co-A compound (Vasudevan et al., 2016). The second stage is the transfer of the acyl group from acetyl-Co-A to the carnitine molecule to form an acyl-carnitine compound assisted by the enzyme carnitine acyltransferase I (palmitoyltransferase). Then, the third stage is the transport of these molecules into the mitochondria. This stage occurs with the help of two other enzymes. 1) Acyl carnitine is carried by carnitine-acylcarnitine translocase 2) Acyl-carnitine is converted back to acyl-CoA by acyltransferase II carnitine (palmitoyltransferase) located in the inner membrane of the mitochondria. The released carnitine is returned to the cytosol (Shahreza et al., 2023)

Carnitine is widespread in the body and is mainly in the muscles. Outside the mitochondria, palmitoyltransferase-1 carnitine will convert long-chain Acyl-COA into acylcarnitine, which can enter the mitochondria by penetrating the inner membrane and accessing the β-oxidation system of enzymes (Jiang et al., 2020). Translocase carnitine-asilkarnitin functions as a carrier exchanger in the inner membrane of the mitochondria. Asilkarnitin is transported in and accompanied by transport out of a single carnitine molecule; Acylcarnitine then reacts with COA, catalyzed by carnitine palmitoyltransferase-11 within the inner membrane. Acyl-COA was reformed in the mitochondrial matrix, and carnitine was released (Shahreza et al., 2023).

Suppose a person consumes an excessive amount of protein that the body needs. In this case, amino acids will be determined, nitrogen will be removed, and the remaining carbon fragments will be converted into fats and stored as energy reserves. In this way, protein can lead to obesity (Stephens et al., 2013). Carnitine is a non-essential amino acid derived from the essential amino acids methionine and lysine, which lowers triglyceride levels at specific doses. Proteins containing low levels of carnitine are at risk of increasing body fat (Gnoni et al., 2020). Beef contains very high carnitines compared to other animal proteins (per 85 grams of beef, contains 81 mg of carnitine) compared to chicken meat (per 85 grams of chicken meat, contains only 3 mg of carnitine (Cardenas & Ochoa, 2023).

Carnitine (*3-hydroxy-4-N-trimethylaminobutyrate*) is derived from amino acid derivatives, and micronutrients function as mediators in metabolism. Carnitine mediates the transport of long-chain fatty acids from the cytosol to the mitochondrial matrix for oxidation of β fatty acids (Virmani & Cirulli, 2022). Other functions of carnitine are maintaining membrane integrity, stabilizing the physiological ratio of coenzyme A (CoASH)/acetyl-CoA in mitochondria, and reducing the production of lactate, which is harmful to the body (Dambrova & Liepinsh, 2015). Carnitine is widely found in the human body in the form of asilkarnitin esters, free carnitines, and some carnitines that are bound to various yl groups that are supplied throughout the body with various functions (Fielding et al., 2018).

At rest, the skeletal muscle carnitine pool is distributed as 80-90% free carnitine and 10-20% short-chain acylcarnitine (with some 10 carbon atoms). Carnitine in the human body is about 300 mg/kg in the heart and skeletal muscle, a small part in the liver, kidneys, and plasma. The amount of circulating plasma carnitine is only 0.5% of the total carnitine in the body (Cardenas, 2016; Shafiei et al., 2020). Carnitine not involved in metabolism will be excreted as free carnitine through the urine. However, some carnitines that are not absorbed at the intestinal trim level will be entirely degraded by bacteria in the colon to produce trimethylamine, a quaternary amine that, after absorption of enterocytes, is oxidized in the liver by monooxygenase containing flavin 3 to form *Trimethylamine-N-Oxide* (TMAO) (Rolfes et al., 2020).

Given its essential role in fatty acid oxidation and energy metabolism, l-carnitine has been investigated as an ergogenic aid to improve exercise capacity in healthy athletic populations (Longo et al., 2016). Early research shows beneficial effects on acute physical performance, such as increased maximum oxygen consumption and higher power output. Subsequent studies have shown the positive impact of dietary supplementation with l-carnitine on the recovery process after exercise (Fielding et al., 2018).

Some forms of carnitine, including L-carnitine, acetyl-L-carnitine, and propionyl-L-carnitine, have slightly different functions and potential benefits. In this study, L-carnitine derived from animal products, namely red meat (beef served in the form of meatballs), was used. Beef, including red meat, contains iron, and iron deficiency occurs in endurance athletes, especially women. Low iron can interfere with oxygen delivery and physical performance. Vegetarianism, the desire for comfort, and the perception of health risks associated with red meat contribute to low bioavailable iron intake (Shahreza et al., 2023). Although the bioavailability of L-carnitine from food is relatively high, absorption from oral L-carnitine supplements is much lower. The bioavailability of L-carnitine from oral supplements (dose, 0.6 to 7 g) ranges between 5% and 25% of the total dose (Jiang et al., 2020).

The body can produce carnitine; most people get sufficient food under normal circumstances. However, certain conditions or food choices can lead to carnitine deficiency. This deficiency can interfere with fatty acid metabolism and energy production, potentially leading to muscle weakness and other health problems. Meat, fish, poultry, and dairy products are the richest sources of L-carnitine, while fruits, vegetables, and whole grains contain little. An omnivorous diet can produce 23 to 135 mg/day of L-carnitine for an average of 70 kg per person. L-carnitine in oral preparations is available in prescription form for primary and secondary L-carnitine deficiency therapies. L-carnitine is also available as a nutritional supplement, with additional doses typically ranging from 0.5 to 2 g/day (Pooyandjoo et al., 2016; Stephens et al., 2013). In general, L-carnitine appears to be well tolerated; no toxic effects have been reported concerning the intake of high doses of L-carnitine. However, L-carnitine supplementation may cause mild gastrointestinal symptoms, including nausea, vomiting, stomach cramps, and diarrhea. Supplements that provide more than three g/day can cause "fishy" body odor (Cardenas & Ochoa, 2023).

L-carnitine, available in the form of dietary supplements, is trending, as it has a good level of safety, antioxidant-type activity, and a suggested effect on energy metabolism pathways (Virmani & Cirulli, 2022). However, L-carnitine therapy has contraindications in patients with hyperglycemia problems. Therefore, L-carnitine supplements should be avoided in patients with diabetes Mellitus (Zare et al., 2015).

The importance of L-carnitine's impact on fatty acid oxidation and energy metabolism is highlighted by many studies on L-carnitine in improving exercise capacity in healthy athletes. It initially shows promising effects on acute physical performance, such as increased maximum oxygen consumption and higher power output. Subsequent studies have shown the positive impact of dietary supplementation with l-carnitine on the recovery process after exercise. This suggests that l-carnitine alleviates muscle injury (Stephens et al., 2013).

4. CONCLUSION

The 50 mg and 100 mg carnitine interventions did not affect lowering blood triglyceride levels. However, the increase in triglycerides in the 50 mg carnitine intervention group was higher than in the 100 mg carnitine intervention group. This is evidenced by the average rating of the 100 mg carnitine group being 17.32, lower than the 50 mg L-carnitine group, which was 21.68. This suggests that administration of more than 100 mg of carnitine for 12 days may affect the increase in triglycerides in the blood.

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