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Shared Mechanisms and Differential Effects of L-Carnitine Supplementation in Athletes and Cardiometabolically Impaired Individuals

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Abstract

Introduction and purpose:

L-carnitine is a key component of mitochondrial fatty acid transport and energy metabolism. Owing to its metabolic importance, it is widely studied in the context of exercise performance and cardiometabolic disorders. This review aims to assess current evidence on its metabolic actions, effectiveness in physical performance and recovery, and its therapeutic potential in cardiovascular and metabolic diseases.

Material and methods:

A literature search was performed in PubMed, Google Scholar and ResearchGate using the keywords. Studies were analyzed for mechanisms of action, supplementation effects, clinical outcomes and safety.

Results:

L-carnitine enhances mitochondrial β-oxidation, regulates the acyl-CoA/CoA ratio and reduces oxidative stress. In athletes, chronic supplementation may increase fat oxidation during moderate-intensity exercise and reduce markers of muscle damage, but it does not significantly improve VO_2max or endurance. In individuals with metabolic disorders, L-carnitine improves insulin sensitivity, lipid profile, metabolic flexibility and cardiac function. Supplementation increases TMAO levels, although its clinical importance remains uncertain.

Conclusions:

L-carnitine shows benefits for exercise recovery and significant improvements in metabolic and cardiovascular dysfunction. Its effects vary depending on baseline metabolic status and carnitine levels. Future research should focus on standardized dosing strategies, long-term effects, and clarifying the clinical relevance of TMAO elevation.

Keywords:

L-carnitine, fatty acid oxidation, mitochondrial function, exercise performance, cardiometabolic health, heart failure, muscle recovery, oxidative stress, supplementation, TMAO

1. Introduction

Carnitine is a water-soluble compound that plays a crucial role in energy metabolism. It facilitates the transport of long-chain fatty acids into the mitochondrial matrix, where they undergo β-oxidation in

order to produce energy. Carnitine is present in almost all tissues but the biggest amount can be found in cardiac and skeletal muscles which have high metabolic demand.

Approximately 75% of the body's L-carnitine supply is obtained from dietary sources—primarily red meat and other animal-derived products. The remaining part is synthesized endogenously from the essential amino acids: lysine and methionine mainly in the liver and kidneys [1]. However, skeletal and cardiac muscles, which store the largest amounts of carnitine, are unable to synthesize it and must obtain it from the bloodstream. Therefore, the appropriate concentration of carnitine is jointly regulated by endogenous synthesis, intestinal absorption, and efficient renal reabsorption.

Despite extensive studies on its physiological roles and potential benefits, l-carnitine remains a subject of ongoing debate. On one hand, it is crucial for mitochondrial fatty acid transport and energy metabolism, processes that directly influence exercise performance[2]. On the other hand, disturbances in carnitine metabolism have been associated with cardiac dysfunction following impaired transport of fatty acids into the mitochondria leading to energy deficiency within cardiomyocytes since the heart mainly depends on fatty acids for energy under physiological conditions [3]. Therefore, this review aims to integrate current knowledge from both exercise and clinical research to provide a comprehensive understanding of L-carnitine's role in linking physical performance with cardiometabolic health.

2. Material and methods

A comprehensive literature search was conducted using PubMed and Google Scholar, with additional sources from ResearchGate. Keywords included: L-carnitine, fatty acid oxidation, mitochondrial function, exercise performance, cardiometabolic health, heart failure, muscle recovery, oxidative stress, supplementation and TMAO. Peer-reviewed studies in English were reviewed. Publications were analyzed for biochemical mechanisms, ergogenic effects, cardiometabolic outcomes, safety, and supplementation protocols. Reference lists of selected papers were examined to identify further relevant research.

3. Current state of knowledge

3.1. Endogenous synthesis of L-carnitine

Carnitine is synthesized endogenously in humans primarily in the liver and kidneys from the amino acids lysine and methionine. Its biosynthesis involves four main enzymatic steps: hydroxylation of 6-N-trimethyllysine (TML) by trimethyllysine dioxygenase (TMLD), cleavage of the intermediate by 3-hydroxy-6-N-trimethyllysine aldolase (HTMLA), oxidation by 4-N-trimethylaminobutyraldehyde

dehydrogenase (TMABADH), and final hydroxylation by γ-butyrobetaine dioxygenase (BBD), leading to the formation of L-carnitine.

After being synthesized or absorbed from the diet, L-carnitine is transported through the bloodstream to target tissues, particularly skeletal and cardiac muscle. Transport across cellular membranes is mediated by OCTN2 (organic cation/carnitine transporter novel type 2), which actively facilitates L-carnitine uptake into cells—especially in organs with high energy demands. Defects in OCTN2 function result in primary carnitine deficiency, a condition characterized by severe metabolic disturbances due to impaired cellular carnitine uptake [4].

3.2. L-carnitine in fatty acid metabolism

L-carnitine is essential for proper metabolic function as it enables fatty acid β-oxidation. It facilitates the transfer of long-chain fatty acids into the mitochondrial matrix by converting them into acylcarnitine derivatives through the action of carnitine palmitoyltransferase I (CPT1) located on the outer mitochondrial membrane. These acylcarnitines are subsequently transported across the inner membrane by carnitine–acylcarnitine translocase (CACT) and reconverted into acyl-CoA by carnitine palmitoyltransferase II (CPT2), allowing their entry into the β-oxidation pathway for ATP synthesis [5].

L-carnitine plays a crucial role in maintaining acyl-CoA/CoA balance. It buffers excess acyl groups through the formation of acylcarnitines, which are subsequently exported from the mitochondria. This process preserves the pool of free CoA within the mitochondrial matrix and prevents the accumulation of potentially toxic acyl-CoA intermediates. By regulating the acyl-CoA to CoA ratio, L-carnitine supports the optimal activity of key metabolic enzymes such as pyruvate dehydrogenase and ensures flexibility between lipid and carbohydrate oxidation [6].

3.3. L-carnitine and physical activity

3.3.1. Effects on energy metabolism

Carbohydrates and fats are the main fuels for ATP production in skeletal muscle. While fat represents the largest energy reserve, its maximal oxidation rate is relatively low and declines at an exercise intensity of 60–70 % of maximal oxygen consumption (VO_2max), making glycogen the main fuel at higher intensities. Since glycogen stores are limited and their depletion is closely linked to fatigue during prolonged high-intensity exercise, enhancing fat metabolism during physical exercise is crucial, as it allows for more efficient utilization of the body's energy stores, preserves muscle glycogen, and delays

the onset of fatigue, thereby supporting sustained performance during prolonged exercise and metabolic adaptation to different exercise intensities [7].

L-carnitine increases the use of fats as an energy source during moderate-intensity exercise (50% -79% VO_2max), provided that its muscle concentration is significantly elevated through chronic supplementation combined with carbohydrates, which facilitates carnitine transport into muscles. This mechanism involves the transport of long-chain fatty acids into mitochondria, where they undergo β-oxidation, leading to increased fat oxidation and glycogen sparing. Studies have shown that chronic L-carnitine supplementation (2g twice daily for 24 weeks, taken with carbohydrates) elevates muscle carnitine content and significantly enhances fat oxidation, primarily through greater utilization of intramuscular lipids. These effects are not observed with short-term supplementation, as muscle carnitine levels do not increase.

During high-intensity exercise (>80% VO_2max), fat oxidation is limited, and metabolism shifts toward carbohydrate utilization; in this context, L-carnitine may improve performance and reduce lactate production, although it does not significantly increase fat oxidation [8].

Growing research indicates that L-carnitine plays an important role in regulating substrate utilization during exercise, particularly by promoting muscle glycogen sparing through enhanced fatty acid oxidation. Chronic L-carnitine supplementation, when combined with carbohydrates, increases intramuscular carnitine content and consequently promotes glycogen sparing during moderate-intensity exercise. This effect is primarily attributed to enhanced fatty acid oxidation, which reduces the use of glycogen as a primary energy source.

Systematic review emphasize that the glycogen-sparing effect depends on elevated muscle carnitine levels, which require long-term supplementation and specific dosing protocols involving concurrent carbohydrate intake[9]. However, this effect is not achieved at higher exercise intensities, where energy metabolism shifts toward greater carbohydrate utilization [10].

3.3.2. Effects on exercise performance and recovery

Current evidence does not support a significant effect of L-carnitine or its derivatives on aerobic performance in healthy individuals or trained athletes. Both acute and chronic supplementation protocols have shown no meaningful improvements in VO_2max, endurance duration, or other indicators of aerobic capacity. While some studies report slight reductions in perceived fatigue or minor benefits during high-intensity exercise, these changes are not accompanied by measurable gains in aerobic efficiency or exercise duration [1112].

However, L-carnitine and its derivatives have been shown to reduce exercise-induced muscle damage and oxidative stress. Supplementation with L-carnitine significantly decreases biomarkers of muscle

injury—such as creatine kinase (CK), myoglobin (Mb), and lactate dehydrogenase (LDH)—particularly within the first 24 hours following intense physical activity. It also alleviates delayed-onset muscle soreness (DOMS) and reduces perceived fatigue after exercise [13].

In addition to these effects, L-carnitine exhibits strong antioxidant properties. It lowers levels of malondialdehyde (MDA), a marker of lipid peroxidation, and enhances the activity of antioxidant enzymes like superoxide dismutase (SOD), thereby limiting the formation of reactive oxygen species and protecting muscle cells from oxidative damage [14].

These benefits have been observed in both physically active and older populations, regardless of sex or age. Furthermore, L-carnitine supports post-exercise muscle recovery by improving blood flow, reducing tissue hypoxia, and modulating inflammatory and oxidative responses. As a result, it facilitates faster restoration of muscle function and helps prevent declines in strength and power following intense exercise [1516].

3.3.3. Determinants of supplementation effectiveness

The effectiveness of L-carnitine supplementation depends on the dosage, form, and duration of administration. Regarding aerobic performance (VO_2max) and endurance duration, supplementation of L-carnitine or its derivatives (e.g., L-carnitine tartrate, propionyl-L-carnitine) do not produce significant improvements in VO_2max or extend time to exhaustion during moderate-intensity exercise[17]. Minor benefits have been reported during high-intensity exercise, such as increased peak power output or greater total work performed but these changes do not translate into measurable gains in aerobic parameters [18].

In contrast, chronic supplementation (typically 2–3 g/day for 3–5 weeks) reduces markers of muscle damage (CK, LDH, myoglobin), alleviates delayed onset muscle soreness (DOMS), and lowers exercise-induced oxidative stress. These benefits have been observed across different L-carnitine forms, including L-carnitine tartrate, and in both physically active individuals and older adults. Antioxidant and recovery effects appear most pronounced after several weeks of continuous supplementation [19].

In summary, the form (e.g., L-carnitine tartrate), dose (2–3 g/day), and duration (minimum 3–5 weeks) are critical determinants of L-carnitine's protective and antioxidant effects, whereas its impact on VO_2max and time to exhaustion during aerobic exercise remains negligible [2021].

The effectiveness of L-carnitine supplementation is influenced by several factors, including training adaptation status, dietary habits (particularly carbohydrate intake), baseline carnitine levels, and the presence of chronic diseases. The greatest benefits are observed in individuals with carnitine deficiency—such as those with genetic transporter defects, chronic kidney disease, liver disorders,

catabolic states, or certain cardiovascular conditions—where supplementation has been shown to improve muscle function and exercise performance [2223].

In contrast, in healthy and well-trained individuals, L-carnitine supplementation generally does not lead to meaningful improvements in physical performance, VO_2max, or intramuscular carnitine content. This lack of effect is likely due to adequate endogenous levels and limited muscle uptake under normal physiological conditions[24].

Co-administration of carbohydrates with L-carnitine can enhance its muscular uptake and may improve exercise metabolism; however, this effect is not consistent and depends on baseline metabolic status, training level, and individual differences in absorption and transport. The overall response to supplementation is highly variable and modulated by factors such as initial carnitine status, dietary pattern (e.g., vegetarian vs. omnivorous diet), exercise intensity, and the presence of metabolic or chronic diseases. The greatest benefits are typically seen in populations with deficiency or increased metabolic demand [2526].

3.4. The role of L-carnitine in metabolic and cardiovascular health

3.4.1. Effects on lipid and glucose metabolism

L-carnitine supplementation has been shown to improve both glucose and lipid metabolism in individuals with metabolic disorders. Regular intake causes reductions in fasting glucose, insulin concentration, and HOMA-IR, indicating enhanced insulin sensitivity, along with favorable changes in lipid profiles, such as decreased triglycerides, total cholesterol, and LDL-cholesterol levels [27]. These effects are most visible with daily doses of 2–3 g over a period of at least 8–12 weeks, particularly among individuals with type 2 diabetes, insulin resistance, or metabolic syndrome [28]. The improvement in insulin sensitivity is thought to result from enhanced oxidation of acyl derivatives in skeletal muscle, which promotes greater metabolic flexibility (the ability to shift from mainly utilizing fat for energy in a fasted state to relying on carbohydrates when stimulated by insulin) and limits the accumulation of harmful lipid intermediates [29].

In terms of lipid regulation, L-carnitine supplementation effectively lowers triglycerides, total cholesterol, and LDL-cholesterol, while its impact on HDL-cholesterol remains minimal or inconsistent. These lipid-lowering effects tend to be more pronounced with higher dosages (>1.5 g/day) and longer supplementation durations [30].

3.4.2. Cardioprotective effects

L-carnitine enhances cardiac function in individuals with metabolic disorders through mitochondrial and antioxidant mechanisms. It facilitates the transport of long-chain fatty acids into mitochondria, promoting β-oxidation and ATP production in cardiomyocytes—particularly under conditions of insulin resistance, diabetes, and heart failure . Moreover, L-carnitine helps to stabilize mitochondrial structure and function, protecting the heart from damage caused by metabolic stress and excess free fatty acids [31]. Its antioxidant actions include reducing the generation of reactive oxygen species (ROS), enhancing the activity of antioxidant enzymes such as superoxide dismutase (SOD2), and preventing cardiomyocyte apoptosis under conditions of hyperglycemia and chronic inflammation [3233].

L-carnitine may provide benefits to patients with heart failure and those recovering from myocardial infarction. Supplementation in individuals with chronic heart failure has been shown to improve left ventricular ejection fraction, cardiac output, reduce BNP/NT-proBNP levels, and limit cardiac chamber enlargement and left ventricular remodeling [3435].

In patients after acute myocardial infarction, early and long-term administration of L-carnitine may reduce left ventricular dilatation and the risk of ventricular arrhythmias as well as anginal symptoms. L-carnitine administration in these patients can decrease overall mortality (by approximately 27%) as well as the incidence of arrhythmias (by about 65%) and angina (by about 40%) [3637].

The quality of evidence is variable—most reported benefits come from studies with smaller sample sizes or lower methodological quality [3839]. Large, clinical trials are still required to precisely determine the role of L-carnitine in the treatment of heart failure and post–myocardial infarction care.

3.5. Potential risks of L-carnitine supplementation

The increase in trimethylamine N-oxide (TMAO) levels following L-carnitine supplementation has raised controversy due to its potential association with cardiovascular risk.

L-carnitine is metabolized by the gut microbiota to produce trimethylamine (TMA), which undergoes hepatic oxidation to form trimethylamine N-oxide (TMAO)—a compound regarded as a biomarker of atherosclerosis and cardiovascular events, including myocardial infarction and stroke [4041].

Studies have shown that higher plasma concentrations of TMAO correlate with an increased risk of cardiovascular incidents, insulin resistance, hypertension, and systemic inflammation [424344].

However, the mechanistic link between TMAO and the pathogenesis of cardiovascular disease remains unclear. Some studies indicate that a moderate increase in TMAO (<10 μmol/L) may represent a risk marker rather than a causal factor, particularly in individuals without chronic kidney disease or advanced heart failure [45].

Moreover, some interventional studies have demonstrated that an increase in TMAO levels after L-carnitine supplementation is not associated with deterioration of the lipid profile, oxidative stress markers, or inflammatory parameters in healthy subjects [46].

After discontinuation of supplementation, TMAO concentrations return to baseline without persistent metabolic alterations [47].

Long-term supplementation with L-carnitine at doses up to 2 g per day is considered safe in healthy adults, with no significant adverse effects reported in clinical studies, although data on higher doses remain limited [48]. Most studies have shown no negative impact on renal, hepatic, or hematological function in individuals without chronic diseases [4950]. In patients with carnitine deficiency, chronic kidney disease, or other metabolic disorders, supplementation is well tolerated and generally regarded as beneficial [51].

4. Discussion

4.1. Effects of L-carnitine supplementation in healthy vs. metabolically impaired individuals

The effects of L-carnitine in athletes and individuals with cardiometabolic disorders arise from shared biological mechanisms, primarily the enhancement of mitochondrial function through increased fatty acid oxidation, and antioxidant activity. L-carnitine serves as a key cofactor for the transport of long-chain fatty acids into mitochondria, enabling efficient β-oxidation and ATP production in both the skeletal muscles of athletes and the cardiomyocytes of individuals with metabolic diseases [5253].

In both populations, L-carnitine stabilizes mitochondrial membrane structure, limits the accumulation of toxic lipid intermediates, reduces oxidative stress, and supports cellular energy homeostasis . In athletes, these mechanisms translate more into improved recovery, reduced muscle damage, and enhanced tolerance to high-intensity exercise [5455].

In individuals with cardiometabolic disorders, L-carnitine improves metabolic flexibility, cardiac function, lipid profile, and glycemic control, which is particularly beneficial in conditions such as insulin resistance, heart failure, and diabetes [565758].

The greater benefits of L-carnitine supplementation observed in older adults or individuals with metabolic disorders are primarily attributed to carnitine deficiency and mitochondrial impairments, which are common in these populations and lead to disturbances in fatty acid oxidation, accumulation of toxic lipid intermediates, and reduced metabolic flexibility [59].

Moreover, in individuals with metabolic syndrome, diabetes, heart failure, or chronic kidney disease, L-carnitine supplementation improves the glucolipid profile, blood pressure, muscle mass, and cardiac function parameters—effects that are not typically observed in healthy, well-trained individuals [60616263].

In athletes, muscle carnitine levels are usually within the normal range, and compensatory mechanisms of muscular bioenergetics remain efficient; therefore, the effects of supplementation are largely limited to enhanced recovery and protection against oxidative stress [6465].

4.2. Limitations

This review is limited by the heterogeneity of available studies, which vary widely in dosage, duration, population characteristics, and outcome measures. Many clinical trials have small sample sizes or lack rigorous methodology. The literature is further constrained by inconsistent reporting of diet, baseline carnitine levels, and gut microbiota composition—factors that significantly influence supplementation outcomes. Additionally, the long-term effects of chronic L-carnitine intake, especially regarding TMAO dynamics, remain insufficiently studied.

4.3. Future Research

Future studies should focus on large, well-designed randomized controlled trials to establish standardized dosing strategies and clarify population-specific responses. Investigations into the interaction between L-carnitine and gut microbiota are essential to better understand TMAO production and its clinical significance. Long-term safety data, particularly in populations with cardiometabolic diseases, are also needed. Furthermore, research should aim to identify biomarkers predicting responsiveness to supplementation, which may help tailor its use to individuals most likely to benefit.

5. Conclusions

L-carnitine plays a well-established role in the transport of long-chain fatty acids into mitochondria and in energy metabolism. Its actions bridge the fields of exercise performance and cardiometabolic health by enhancing mitochondrial function, increasing fatty acid oxidation, and exerting antioxidant effects.

In the context of sports performance, the effects of supplementation are moderate and condition-dependent, influenced by factors such as dosage, duration, and training status. L-carnitine may support recovery, reduce oxidative stress and muscle damage, but it does not consistently improve aerobic capacity or endurance performance.

In individuals with metabolic or cardiovascular disorders, the benefits are more pronounced, including improvements in lipid and glucose profiles, metabolic flexibility, and cardiac function—particularly in those with carnitine deficiency or mitochondrial dysfunction.

However, the rise in TMAO levels following L-carnitine supplementation remains a subject of controversy, as its clinical relevance to cardiovascular risk is still uncertain.

L-carnitine appears to be a safe and promising metabolic and mitochondrial support, nevertheless future research should focus on large, well-designed randomized controlled trials to determine its optimal dosing strategies, long-term safety, and the clinical relevance of TMAO modulation. Clarifying these aspects is essential to define L-carnitine's precise role in both exercise performance and cardiometabolic health.

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