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L-Carnitine and Its Derivatives in Metabolic and Chronic Diseases: A Narrative Review

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ABSTRACT

Background. L-carnitine (LC) is a naturally occurring vitamin-like compound involved in mitochondrial fatty acid transport and cellular energy metabolism. Beyond its physiological role, LC and its derivatives have also been shown to exhibit antioxidant and anti-inflammatory properties.

Aim. This narrative review summarizes current evidence regarding the role of L-carnitine in human disease and its clinical applications, with particular emphasis on its therapeutic potential, safety profile, and major areas of clinical research.

Methods. A narrative review was conducted using PubMed, Cochrane Library, and Google Scholar. Systematic reviews, meta-analyses, and randomized controlled trials published between 2013 and 2025 were included to provide a comprehensive overview of the available evidence. Animal studies, conference abstracts, case reports, and non-English publications were excluded from this review. Twenty-nine quality sources were selected for analysis.

Results. Evidence from systematic reviews, meta-analyses, and randomized controlled trials suggests that L-carnitine may improve glucose metabolism, insulin sensitivity, lipid parameters, neuropathic symptoms, and sperm quality. The strongest evidence supports beneficial effects on metabolic outcomes and male reproductive health, while evidence supporting improvements in major cardiovascular and reproductive endpoints remains limited. L-carnitine is generally well tolerated at doses up to 2–3 g/day; however, long-term oral

supplementation may increase circulating trimethylamine-N-oxide (TMAO) concentrations, although the clinical implications remain uncertain.

Conclusions. Current evidence suggests a role of L-carnitine as an adjunctive nutritional intervention in selected patient populations. Nevertheless, further well-designed randomized controlled trials with long-term follow-up and clinically relevant endpoints are required to establish its therapeutic efficacy and long-term safety.

KEYWORDS L-carnitine; acetyl-L-carnitine; cardiovascular disease; diabetes mellitus; obesity; dyslipidemia; neuropathy; polycystic ovary syndrome; male infertility

1. Introduction

Interest in the therapeutic potential of L-carnitine has increased beyond its established role in primary carnitine deficiency, particularly in chronic diseases associated with mitochondrial dysfunction, oxidative stress, and inflammation.

L-carnitine (3-hydroxy-4-N-trimethylaminobutyrate) is an essential compound present in virtually all living cells. Its primary role is to transport long-chain fatty acids into the mitochondria, where they undergo β-oxidation to produce ATP. Inside the mitochondria, fatty acids are progressively degraded by acyl-CoA dehydrogenases, which enables energy production. In addition to facilitating fatty acid oxidation, L-carnitine promotes the removal of excess acyl groups, maintains intramitochondrial coenzyme A (CoA) homeostasis, supports mitochondrial function, and protects cells against oxidative damage and metabolic stress [1].

Recent evidence suggests that L-carnitine (LC) functions not only as a transporter of fatty acids but also as a potent antioxidant with anti-inflammatory properties. Its antioxidant activity has been attributed to several mechanisms, including modulation of mitochondrial function, reduced production of reactive oxygen species (ROS), and maintenance of cellular redox balance. The anti-inflammatory effects may be mediated, at least partially, through

inhibition of the nuclear factor-kappa B (NF-κB) signaling pathway, likely secondary to the suppression of ROS generation [1, 4, 13].

More than 99% of body carnitine is located intracellularly. It is found mainly in skeletal muscle, the myocardium, and the liver. Less than 1% circulates in plasma. Approximately 25% of total body carnitine is synthesized endogenously, while the remaining 75% is generally obtained from dietary intake. Meat, poultry, fish, and dairy products are the principal dietary sources, while plant-based foods contain considerably lower amounts. Carnitine concentrations vary according to sex and age, with generally higher levels in males and a positive correlation between age and circulating carnitine concentrations [2, 3].

The term "carnitine" refers to two stereoisomeric forms, L-carnitine and D-carnitine. Only the L-isomer is biologically active and used in clinical practice. In contrast, D-isomers are biologically inactive and may interfere with the activity of the L-isomer. Clinically relevant derivatives (esterified forms) include acetyl-L-carnitine (ALC) and propionyl-L-carnitine (PLC) [2, 5, 14].

This narrative review summarizes current evidence regarding the therapeutic role of L-carnitine and its derivatives in chronic diseases, with particular emphasis on clinical efficacy and its safety profile.

2. Clinical Outcomes

2.1. Cardiovascular Disease

Existing evidence indicates modest improvements in cardiometabolic parameters following LC supplementation. However, these benefits have not been demonstrated as major cardiovascular outcomes [13].

2.1.1. Lipids

Meta-analyses have demonstrated improvements in lipid metabolism following LC supplementation, although the magnitude of these effects varies across patient populations. Reported changes include reductions in triglycerides, ApoB-100, oxidized LDL cholesterol, and thiobarbituric acid reactive substances (TBARS), along with increases in HDL cholesterol and ApoA1 concentrations. However, in patients with T2DM, improvements

appear to be less consistent, with some analyses reporting reductions only in total and LDL cholesterol without significant changes in triglycerides or HDL cholesterol [5, 6, 14].

Across studies included in the meta-analyses, oral LC supplementation reduced plasma lipoprotein (a) [Lp(a)] concentrations by an average of 8.82 mg/dL, whereas intravenous administration showed no significant effect. This effect may result from enhanced mitochondrial fatty acid oxidation and reduced hepatic Lp(a) synthesis [12].

Evidence from randomized controlled trials generally supports these findings. In patients with coronary artery disease, supplementation with 1000 mg/day of LC for 12 weeks reduced triglyceride concentrations and increased HDL cholesterol and ApoA1 levels, whereas total and LDL cholesterol remained unchanged [13].

2.1.2. Blood Pressure

LC may improve blood pressure through favorable effects on endothelial function, nitric oxide bioavailability, and insulin sensitivity. Evidence from meta-analyses remains inconsistent. While some studies reported reductions in diastolic blood pressure with doses of at least 2 g/day; others demonstrated significant improvements only in systolic blood pressure. Similar findings have been observed with acetyl-L-carnitine supplementation in hypertensive individuals at high cardiovascular risk [14].

2.1.3. Heart Failure

Meta-analyses indicate that L-carnitine supplementation improves several surrogate measures of cardiac function in patients with chronic heart failure. Reported benefits include increases in left ventricular ejection fraction (LVEF), stroke volume, cardiac output, and diastolic function (E/A ratio), together with reductions in B-type natriuretic peptide (BNP) and N-terminal pro-B-type natriuretic peptide (NT-proBNP) concentrations. Improvements have also been observed in structural parameters of cardiac remodeling, including left ventricular end-diastolic and end-systolic dimensions and volumes. Despite these favorable effects on surrogate markers, supplementation has not consistently improved mortality, exercise capacity, or the incidence of adverse events. Comparable findings have also been reported in patients with dilated cardiomyopathy [10, 25].

2.1.4. Other Cardiovascular Conditions

Coronary artery bypass grafting (CABG) is associated with a transient postoperative decline in circulating LC concentrations, which may impair myocardial energy metabolism. In one study, oral supplementation with LC (50 mg/kg/day), initiated on the third postoperative day, restored preoperative plasma carnitine concentrations within 10 days. These findings suggest that postoperative LC supplementation may facilitate recovery of myocardial energy metabolism following CABG [26].

A multicenter randomized clinical trial evaluated patients undergoing maintenance hemodialysis who had previously received intravenous LC. Reduction or discontinuation of LC supplementation resulted in significant decreases in plasma and erythrocyte free carnitine concentrations together with increased BNP levels. However, the intervention had no significant effect on cardiac function. Additionally, LC has been shown to inhibit the formation of advanced glycation end products (AGEs), suggesting a potential role in vascular protection, particularly among high-risk populations such as patients undergoing chronic hemodialysis [12, 27].

2.2. Glucose Metabolism

LC supplementation appears to improve whole-body glucose disposal, especially non-oxidative pathways, resulting in increased glucose uptake and glycogen storage. LC also improves skeletal muscle metabolism, including increased glycogen content and decreased lactate accumulation. LC supplementation may also restore reduced metabolic flexibility in patients with impaired glucose tolerance (IGT). These metabolic effects appear particularly pronounced in individuals with impaired glucose tolerance [5, 11].

These mechanisms translate into modest but clinically relevant metabolic improvements. Meta-analyses demonstrate significant reductions in insulin resistance, assessed by HOMA-IR, with greater treatment effects observed after longer intervention periods. LC supplementation also significantly lowers fasting blood glucose in patients with T2DM, whereas its effects on HbA1c remain less consistent. The greatest benefits have generally been observed with doses ranging from 1000 to 2000 mg/day, although one randomized

controlled trial also demonstrated a significant reduction in HbA1c after 12 weeks of treatment [6, 14, 28].

2.3. Neuropathy

Current evidence suggests that the strongest benefits of L-carnitine supplementation in neuropathy have been observed with its derivatives, particularly acetyl-L-carnitine (ALC).

A Cochrane systematic review of four randomized controlled trials (907 participants) evaluated the efficacy of ALC in diabetic peripheral neuropathy. Three placebo-controlled trials assessed ALC at doses ranging from 1500 to 3000 mg/day, while one trial compared ALC (2000 mg/day) with methylcobalamin (1.5 mg/day). ALC supplementation was associated with a modest reduction in neuropathic pain compared with placebo, with the greatest benefit observed at doses exceeding 1500 mg/day. Lower doses were ineffective. Two placebo-controlled trials suggested improvement in vibration perception after 12 months of treatment. Functional outcomes were comparable between ALC and methylcobalamin, with no significant between-group differences [7].

Similar but less pronounced analgesic effects have also been reported in non-diabetic peripheral neuropathy [8].

2.4. Obesity

Umbrella meta-analyses have demonstrated that carnitine supplementation significantly reduces body weight, body mass index (BMI), fat mass, and waist circumference. The greatest effects were observed in interventions lasting less than 18 weeks, particularly in individuals with obesity with a baseline BMI ≥30 kg/m². However, no significant changes in body weight or BMI were observed among patients with diabetes [6, 9, 14].

One randomized controlled trial compared combined supplementation of LC with a multispecies synbiotic with LC plus placebo in women with obesity. Both interventions showed improvements in body composition, lipid profile, and glucose homeostasis. Significant reductions in triglycerides and fasting blood glucose, together with greater increases in HDL cholesterol, were observed only in the synbiotic co-supplementation group. These findings suggest that modulation of the gut microbiota may potentiate selected

metabolic effects of LC supplementation, although confirmation in larger studies is needed [18].

2.5. Polycystic Ovary Syndrome (PCOS)

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder affecting women of reproductive age, with an estimated prevalence ranging from 12% to 18%, depending on the diagnostic criteria and the population studied. The syndrome is characterized by reproductive, metabolic, and psychological manifestations [15].

The potential role of L-carnitine in PCOS has been attributed primarily to its antioxidant properties and its ability to reduce oxidative stress. Consequently, supplementation during the follicular phase has been proposed as a strategy to improve reproductive outcomes in women undergoing assisted reproductive treatment [13].

Current evidence does not support a clinically meaningful benefit of LC supplementation on reproductive outcomes. A randomized controlled trial found no improvement in oocyte or embryo quality and its maturation, fertilization, implantation, or pregnancy rates following assisted reproductive technology (ART). Similarly, systematic reviews and meta-analyses have reported no significant improvements in ovulation, clinical pregnancy, or live birth rates. The certainty of evidence, particularly for live birth outcomes, remains low [16, 17, 19].

In contrast, metabolic benefits appear more favorable. Supplementation with up to 3 g/day of LC may reduce BMI and improve insulin resistance, together with improvements in total cholesterol, LDL cholesterol, and triglyceride concentrations in several studies. Nevertheless, improvements in lipid parameters have not been observed consistently across all studies [16, 17].

2.6. Male Infertility

Infertility affects an estimated 48–186 million people worldwide, with up to one-third of these cases attributed to male infertility. Idiopathic male infertility refers to impaired fertility despite the absence of an identifiable underlying cause following standard clinical evaluation. Reduced sperm motility is a common feature of male infertility. Carnitines play a role in

sperm energy metabolism and mitochondrial function, thereby supporting sperm motility and viability, while their antioxidant properties may further protect spermatozoa against oxidative damage [20, 23].

Evidence from systematic reviews and randomized controlled trials indicates that LC supplementation improves sperm motility parameters and may also improve sperm concentration and morphology. The most consistent benefits were observed in patients with asthenozoospermia and severe idiopathic infertility. Combined L-carnitine/acetyl-L-carnitine supplementation appears to provide greater improvements in seminal parameters than either compound alone [20, 21, 22].

One randomized placebo-controlled trial evaluated a formulation containing LC, acetyl-L-carnitine, antioxidants, trace elements, and vitamins. Patients with higher seminal carnitine concentrations before therapy had higher progressive motility. Furthermore, supplementation also increased serum testosterone and luteinizing hormone concentrations. Despite improvements in semen quality, current evidence has not demonstrated consistent benefits for conception, clinical pregnancy, or live birth rates [20, 21, 22, 24].

2.7. Adverse Effects

Overall, LC supplementation is well tolerated, and reported adverse events are generally mild and predominantly gastrointestinal. Doses up to 2–3 g/day have consistently demonstrated a favorable safety profile. Higher doses (≥3 g/day) have been associated with an increased frequency of gastrointestinal symptoms, including nausea, abdominal discomfort, and diarrhea. Less commonly reported adverse effects include muscle weakness and seizures, particularly in susceptible individuals [3, 4, 9, 14].

A potential concern associated with long-term oral supplementation is increased TMAO production. LC can be metabolized by the gut microbiota to trimethylamine (TMA), which is oxidized in the liver to trimethylamine N-oxide (TMAO). Elevated circulating TMAO concentrations have been associated with atherosclerosis, impaired reverse cholesterol transport, enhanced foam cell formation, and an increased risk of cardiovascular disease. Interestingly, TMAO production appears to depend on gut microbiota composition. Vegans and vegetarians produce substantially lower TMAO concentrations following an oral L-carnitine challenge than omnivorous individuals. Chronic dietary exposure to carnitine may increase the ability of the gut microbiota to produce TMAO [4, 29].

Consequently, the overall impact of chronic oral LC supplementation on cardiovascular risk remains uncertain and should be evaluated in long-term studies assessing hard clinical endpoints.

3. Discussion

The available evidence indicates that LC and its derivatives may provide modest benefits across several metabolic, cardiovascular, neurological, and reproductive disorders. The most consistent findings relate to improvements in glucose metabolism, insulin sensitivity, selected lipid parameters, neuropathic symptoms, and sperm quality, whereas evidence supporting clinically meaningful cardiovascular and fertility outcomes remains limited.

The broad spectrum of reported clinical effects probably reflects the central role of L-carnitine in mitochondrial energy metabolism, suggesting that improvements observed across different diseases may originate from common metabolic mechanisms rather than disease-specific actions.

Interpretation of these findings should be cautious. Most randomized controlled trials and meta-analyses have focused primarily on surrogate biochemical or metabolic markers rather than patient-centered outcomes such as cardiovascular events, disease progression, live birth, or mortality. Moreover, substantial heterogeneity in study populations, supplementation regimens, treatment duration, and outcome measures limits the comparability of individual studies and the certainty of pooled estimates.

Another important consideration is the potential increase in circulating TMAO concentrations during long-term oral supplementation. Although elevated TMAO levels have been associated with increased cardiovascular risk in observational studies, causality has not been established, and the clinical relevance of this association remains uncertain.

This review has several limitations. As a narrative review, it was not conducted according to PRISMA guidelines, and selection bias cannot be excluded. Interpretation of the evidence is limited by heterogeneity in study populations, supplementation protocols, treatment duration, and outcome measures.

The available evidence suggests that the greatest benefits are observed in patients with metabolic disturbances or conditions associated with impaired mitochondrial energy metabolism, whereas supplementation appears less effective in otherwise healthy individuals. Overall, L-carnitine appears to be a useful adjunctive nutritional intervention for selected patient populations. Nevertheless, further large-scale randomized controlled trials are needed to establish its long-term efficacy and safety before its routine use can be recommended in clinical practice.

4. Conclusions

Current evidence suggests LC improves several metabolic and reproductive parameters, particularly glucose metabolism, insulin sensitivity, selected lipid markers, neuropathic symptoms, and sperm quality.

Taken together, it should be regarded as an adjunctive nutritional strategy rather than a stand-alone therapeutic intervention. Future well-designed randomized controlled trials should focus on clinically meaningful outcomes and identification of patient populations most likely to benefit from supplementation.

Disclosure

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