CLINICAL MONOGRAPH · ENERGY & NUTRITIONAL

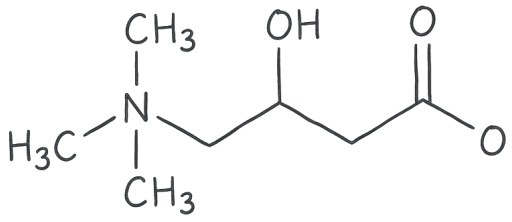
L-Carnitine

Mitochondrial transporter for fatty-acid metabolism

L-carnitine is a small molecule the body makes (and also gets from red meat and dairy) that moves long-chain fats into mitochondria, the cellular compartments that turn fat into energy [marcovina2013]. Without enough carnitine, fat cannot be burned efficiently, and toxic fatty-acid intermediates can build up.

Prescription levocarnitine, sold as Carnitor, is FDA-approved for people born without normal carnitine handling, for certain inherited metabolic diseases, and for kidney-failure patients on dialysis who lose carnitine through the dialyzer [fda_label_carnitor]. It is also used to treat valproic acid (an anti-seizure medication) toxicity, which depletes carnitine [lheureux2009] [eknoyan2003; lenzi2003].

Outside those approved uses, L-carnitine, acetyl-L-carnitine (ALC), and propionyl-L-carnitine (PLC) have been studied in heart failure after a heart attack, leg pain from peripheral artery disease, male infertility, Alzheimer disease, diabetic nerve pain, and age-related fatigue [marcovina2013; iliceto1995; brevetti1999]. Evidence in those off-label settings ranges from moderately positive to mixed. Many over-the-counter supplement versions exist; OTC supplements are not the same regulated product as prescription Carnitor [hudson2003; sima2005].



EVIDENCE POSTURE FDA APPROVED WELL STUDIED REVIEWED **2026-05-11**

State-licensed 503A | Pharmacist reviewed | Doctor led | Cold-chain ready | Patient choice preserved

Contents

Click any section to jump there. Page numbers update on render.

FOR CLINICIANS

L-carnitine (levocarnitine) is the obligate cofactor for long-chain fatty-acid translocation across the inner mitochondrial membrane via the carnitine palmitoyltransferase shuttle (CPT1 on the outer leaflet, carnitine-acylcarnitine translocase, and CPT2 on the inner leaflet) [bremer1990, marcovina2013] [fda503a]. Endogenous biosynthesis from lysine and methionine occurs primarily in liver, kidney, and brain; roughly 75% of body carnitine is dietary in non-vegetarian adults [vaz2002, rebouche2004]. Deficiency states fall into two categories [brevetti1995; brevetti1999; hiatt2001]. Primary carnitine deficiency reflects loss-of-function mutations in SLC22A5 (OCTN2), the high-affinity plasma-membrane carnitine transporter, producing cardiomyopathy, hypoketotic hypoglycemia, and skeletal myopathy [magoulas2012, longo2017, longo2019]. Secondary deficiencies arise from inborn errors of fatty-acid oxidation, hemodialysis-associated losses [eknoyan2003], valproate-pivalate conjugation [lheureux2009, lheureux2005], and certain antiretroviral nucleoside analogues [famularo1997].

Levocarnitine (Carnitor; Sigma-Tau/Leadiant) is FDA-approved for primary systemic carnitine deficiency, secondary deficiency due to specified inborn errors of metabolism, and the prevention and treatment of carnitine deficiency in end-stage renal disease patients on hemodialysis [fda_label_carnitor, dailymed_levocarnitine] [fda503a]. The National Kidney Foundation Carnitine Consensus Conference [eknoyan2003] codified the dialysis use case at IV 20 mg/kg post-dialysis with reassessment at 3, 6 months. Off-label clinical investigation includes (1) cardiac ischemia and post-MI ventricular remodeling, the CEDIM trial [iliceto1995] reported attenuated LV dilation at 6 and 12 months with oral L-carnitine 9 g/day initiated within 24 h of MI, and the DiNicolantonio (2013) meta-analysis [dinicolantonio2013] found a 27% reduction in all-cause mortality across 13 controlled trials of L-carnitine after acute MI; (2) heart failure with PLC [mancini1992, rizos2000]; (3) intermittent claudication where the PLC ester improved pain-free and maximum walking distance in multicenter trials; (4) male infertility, where L-carnitine and ALC improved sperm motility in randomized trials [lenzi2003] and ALC+PLC adjunctively improved sildenafil response after radical prostatectomy [cavallini2005]; (5) Alzheimer disease, where the Cochrane review [hudson2003] of ALC trials reported small benefits on cognitive scales without consistent effect on global function [pettegrew1995, spagnoli1991, salvioli1994]; (6) chronic diabetic peripheral neuropathy [sima2005]; (7) hepatic encephalopathy [malaguarnera2013]; (8) fatigue in elderly and centenarian populations [malaguarnera2007, malaguarnera2008] and multiple sclerosis [tomassini2004]; and (9) the TMAO, CV-risk hypothesis, where gut-microbiota-derived trimethylamine-N-oxide from carnitine and choline metabolism has been associated with atherosclerosis in observational and mechanistic work [koeth2013, wang2011], clinical relevance to therapeutic L-carnitine use remains an open question.

Compounded 503A levocarnitine is dispensed only on patient-specific prescription for clinical contexts the manufactured Carnitor product cannot meet, typically custom-strength IV infusion protocols, IM injections, sublingual troches when oral GI intolerance precludes the labeled solution or tablet, or compounded multi-ingredient blends with B-vitamins or amino acids. OTC L-carnitine and acetyl-L-carnitine dietary supplements are widely sold under DSHEA and are not interchangeable with the prescription drug [fda503a] [hiatt2011; delaney2013].

Why Personalized L-Carnitine

Carnitor's labeled dosing was set for narrow populations: 1 to 3 g/day oral for primary or secondary carnitine deficiency, and 20 mg/kg IV post-dialysis for end-stage renal disease. It was not calibrated for the valproate-toxicity patient who cannot keep oral solution down, for the post-MI patient on the CEDIM 9 g/day schedule, for the male-infertility regimen pairing L-carnitine with acetyl-L-carnitine, or for the dialysis patient whose carnitine losses, residual renal function, and tolerance for IV push do not match the trial average. Body pools live in skeletal and cardiac muscle, depend on OCTN2 transport, and turn over differently in valproate exposure, hemodialysis, and inherited transporter defects. Those are not interchangeable patients on one dose schedule.

That gap is what a compounding pharmacy fills. The molecule is the same levocarnitine the FDA reviewed for Carnitor. What changes is the preparation: a custom-strength IV infusion when the labeled vial concentration does not fit the protocol, an IM injection when peripheral IV access is poor, a sublingual troche when the oral solution triggers nausea, diarrhea, or the fishy-odor complaint that makes patients stop taking it, or a multi-ingredient blend with B-vitamins or amino acids when the clinical plan calls for it. Strengths and routes sit outside what Sigma-Tau or Leadiant manufacture, because the manufactured product was built for the labeled indications, not the off-label or atypical case the prescriber is actually treating.

This is the older arrangement that pre-dates mass manufacturing. A physician writes the prescription for a named patient. A licensed pharmacist prepares it. Modern 503A oversight, state inspection, named-on-label dispensing, and a recall path keep it honest.

Quick Facts About L-Carnitine

Category: Quaternary ammonium amino-acid derivative; mitochondrial long-chain fatty-acid transporter

Common aliases: Levocarnitine (USAN/INN); 3-hydroxy-4-(trimethylammonio)butanoate; vitamin BT (historical); acetyl-L-carnitine (ALC) and propionyl-L-carnitine (PLC) are short-chain acyl esters

Biological role: Obligate cofactor for the carnitine palmitoyltransferase (CPT1/CPT2) shuttle that imports long-chain acyl-CoA across the inner mitochondrial membrane for β-oxidation; buffers the cellular acyl-CoA/CoA ratio

Endogenous synthesis: Synthesized in liver, kidney, and brain from lysine and methionine via four enzymatic steps; ~75% of body carnitine is normally dietary (red meat and dairy)

FDA-approved branded product: Carnitor and Carnitor SF (levocarnitine), oral solution, tablets, and intravenous injection, approved for primary systemic carnitine deficiency, secondary deficiency due to inborn errors of metabolism, and the prevention/treatment of carnitine deficiency in end-stage renal disease patients on hemodialysis

Routes: Oral solution, oral tablet/capsule, intravenous, intramuscular, sublingual troche (compounded)

Evidence posture: Mechanism and biochemistry well established. FDA-approved use in primary/secondary deficiency and dialysis-associated deficiency is supported by replicated trials. Off-label uses (cardiac ischemia, intermittent claudication, male infertility, Alzheimer disease, diabetic neuropathy, fatigue) range from well-studied to mixed.

Compounded under: 503A, patient-specific prescription only; common preparations include custom-strength IV infusions, IM injections, sublingual troches, and amino-acid/B-vitamin blends

Regulatory note: Numerous OTC L-carnitine and acetyl-L-carnitine products are marketed as dietary supplements under DSHEA; OTC supplements are not interchangeable with prescription levocarnitine (Carnitor)

SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY

L-Carnitine described in this monograph is a 503A compounded preparation. Every dose is made on a prescription, for a named patient, by a licensed pharmacist. It is not a stocked, mass-manufactured product.

- **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
- **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
- **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
- **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
- **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is L-Carnitine?

L-carnitine, chemically 3-hydroxy-4-(trimethylammonio)butanoate, is a small quaternary ammonium zwitterion with a molecular weight of 161 daltons. The L-isomer (levocarnitine) is the only biologically active form; the D-isomer is not endogenous and competitively inhibits carnitine uptake, which is why prescription product specifies the L-enantiomer exclusively [bremer1990, fda_label_carnitor].

Endogenous L-carnitine is synthesized from protein-bound lysine and methionine through a four-enzyme pathway distributed across liver, kidney, and brain [vaz2002]. Trimethyllysine hydroxylase, hydroxytrimethyllysine aldolase, trimethylaminobutyraldehyde dehydrogenase, and gamma-butyrobetaine hydroxylase generate L-carnitine in sequence. Skeletal muscle and cardiac muscle hold the largest tissue pools (~95% of body carnitine) but lack the final hydroxylation step and rely on plasma uptake via the OCTN2 transporter encoded by SLC22A5 [magoulas2012, longo2017].

Acetyl-L-carnitine (ALC) and propionyl-L-carnitine (PLC) are short-chain acyl esters of carnitine [mancini1992]. They occur naturally as products of the carnitine acetyltransferase and carnitine acyltransferase reactions, which buffer the mitochondrial acetyl-CoA/CoA pool. As exogenous therapies, ALC has been studied primarily in central nervous system contexts (Alzheimer disease, diabetic neuropathy, fatigue) and PLC primarily in vascular contexts (intermittent claudication, heart failure) [pettegrew1995; sima2005; brevetti1999].

How L-Carnitine Works

L-carnitine's central physiological role is to enable long-chain fatty-acid β-oxidation. Long-chain acyl-CoA esters cannot cross the inner mitochondrial membrane unassisted. Carnitine palmitoyltransferase 1 (CPT1) on the outer mitochondrial membrane transfers the acyl group from coenzyme A to carnitine, producing acylcarnitine. The carnitine-acylcarnitine translocase (CACT) then exchanges acylcarnitine into the matrix in trade for free carnitine moving outward. CPT2 on the inner leaflet transfers the acyl group back onto matrix CoA, releasing free carnitine for re-export and generating acyl-CoA that enters the β-oxidation spiral [bremer1990, marcovina2013].

Beyond fatty-acid transport, carnitine buffers the cellular acyl-CoA/CoA ratio through reversible esterification reactions catalyzed by carnitine acyltransferases. This buffering function disposes of accumulating acyl groups during metabolic stress (fasting, ischemia, exercise, inborn errors of fatty-acid oxidation) and maintains free coenzyme A available for other reactions [marcovina2013, vaz2002].

When carnitine pools are insufficient, whether from genetic transporter defects, hemodialysis losses, drug-induced depletion (notably valproate, which conjugates with carnitine via pivalate-style esterification and is also a CPT1 inhibitor), or competitive inhibition by accumulating acylcarnitines in fatty-acid oxidation

defects, long-chain fatty-acid oxidation fails, hypoketotic hypoglycemia and cardiomyopathy develop, and toxic acyl-CoA intermediates accumulate [magoulas2012, lheureux2009, lheureux2005].

Biological Role of L-Carnitine

Carnitine is essential to whole-body energy economy: long-chain fatty acids supply roughly 60, 70% of resting cardiac ATP production and a large fraction of skeletal-muscle energy during sustained exercise, and the carnitine shuttle is the rate-limiting committed step for that substrate flow [bremer1990, marcovina2013].

The body maintains carnitine homeostasis through a balance of dietary intake (predominantly red meat and dairy in omnivores; markedly lower in strict vegetarians and vegans), endogenous biosynthesis (~25% of daily requirement in non-vegetarian adults), and aggressive renal tubular reabsorption [vaz2002, rebouche2004]. Total body carnitine pool is approximately 100 mmol in adults, with >95% in skeletal and cardiac muscle and the remainder distributed across liver, kidney, plasma, and other tissues.

Plasma free L-carnitine in healthy adults ranges from approximately 25, 50 µM; the acylcarnitine/free-carnitine ratio is normally <0.4 and rises in fatty-acid oxidation disorders, dialysis-associated deficiency, and valproate use. Newborn screening programs in the United States and most developed countries include carnitine and acylcarnitine profiling by tandem mass spectrometry, which detects primary carnitine deficiency and most fatty-acid oxidation defects [magoulas2012, longo2019].

Detailed Mechanism of L-Carnitine

Carnitine is taken up into most peripheral tissues by the high-affinity sodium-dependent OCTN2 transporter (SLC22A5), expressed on enterocytes, renal tubular epithelium, cardiomyocytes, skeletal muscle, and placenta [longo2017, magoulas2012]. Loss-of-function mutations in SLC22A5 produce primary systemic carnitine deficiency, with plasma carnitine <5 µM (normal range 25, 50 µM) and severe intracellular depletion. Carnitine is filtered freely at the glomerulus and roughly 95, 99% reabsorbed via OCTN2 in the proximal tubule; impaired reabsorption is the basis for renal carnitine wasting in OCTN2 disease.

Hemodialysis removes free carnitine and short-chain acylcarnitines (molecular weight <200 daltons, modest protein binding, distribution into total body water) at clearances approaching the dialyzer urea clearance. Sustained loss over months to years drives a dialysis-associated carnitine disorder characterized by low total and free plasma carnitine, an elevated acylcarnitine/free-carnitine ratio, and clinical manifestations that include erythropoietin-resistant anemia, intradialytic hypotension, skeletal muscle weakness, and cardiomyopathy in some patients. The NKF Carnitine Consensus Conference [eknoyan2003] codified intravenous levocarnitine 20 mg/kg post-dialysis as the standard replacement regimen, with reassessment of clinical response at 3, 6 months.

Valproic acid causes carnitine depletion through three mechanisms: (1) formation of valproylcarnitine that is excreted in urine and depletes the free-carnitine pool; (2) inhibition of carnitine biosynthesis and renal tubular reabsorption; and (3) inhibition of CPT1 with secondary impairment of long-chain fatty-acid oxidation. Acute valproate overdose and chronic high-dose valproate use can precipitate hyperammonemic encephalopathy and hepatotoxicity through these pathways, and intravenous levocarnitine reverses or attenuates the toxicity in case series and observational data summarized by Lheureux and colleagues [lheureux2009, lheureux2005].

Acetyl-L-carnitine crosses the blood-brain barrier more efficiently than L-carnitine, supports neuronal acetyl-CoA generation, and serves as an acetyl donor for acetylcholine biosynthesis, the proposed basis for its investigation in cognitive disorders [pettegrew1995, hudson2003]. Propionyl-L-carnitine is preferentially taken up by skeletal and cardiac muscle and is converted to propionyl-CoA, which enters the citric acid cycle through succinyl-CoA as an anaplerotic substrate, the proposed basis for its preferential study in ischemic vascular indications [brevetti1999, mancini1992].

The TMAO hypothesis [koeth2013, wang2011] proposes that gut microbial metabolism of dietary carnitine and phosphatidylcholine generates trimethylamine, which the hepatic flavin monooxygenase FMO3 oxidizes to trimethylamine-N-oxide. Plasma TMAO correlates with atherosclerotic burden in observational cohorts and accelerates atherosclerosis in apoE-/- mice. The clinical implications for prescription levocarnitine therapy at supplemental doses are not established; the TMAO literature predominantly concerns dietary carnitine intake and gut microbiota composition rather than short-course replacement therapy.

L-Carnitine Research History

Carnitine was isolated from muscle extracts by Gulewitsch and Krimberg in 1905 and identified as a growth factor for the mealworm Tenebrio molitor in 1948, the basis for its historical (and now disfavored) name vitamin BT. The mitochondrial fatty-acid-transport function was established by Fritz, Bremer, and others through the 1960s and codified in Bremer's 1990 review [bremer1990].

Clinical recognition of human carnitine deficiency began with case reports of muscle and systemic deficiency in the 1970s. The molecular basis of primary deficiency, OCTN2/SLC22A5 mutations, was established in the late 1990s [magoulas2012, longo2017]. Levocarnitine (Carnitor) received FDA orphan-drug approval for primary carnitine deficiency in 1985 (oral) and 1992 (intravenous), with the indication expanded to include end-stage renal disease patients on hemodialysis in 1999 [fda_label_carnitor].

Cardiovascular investigation began in the 1980s and 1990s. The CEDIM trial [iliceto1995] reported attenuated left-ventricular remodeling at 12 months with oral L-carnitine 9 g/day for the first 5 days and 6 g/day for the next 12 months in 472 adults randomized within 24 hours of an acute anterior myocardial infarction. Propionyl-L-carnitine was developed as a tissue-targeted ester for ischemic vascular disease and tested in heart failure [mancini1992, rizos2000] and intermittent claudication [brevetti1995].

Acetyl-L-carnitine investigation in Alzheimer disease began with European trials in the late 1980s and produced a modest signal across multiple small randomized trials [pettegrew1995, spagnoli1991, salvioli1994]; the 2003 Cochrane review by Hudson and Tabet [hudson2003] concluded that ALC produced small benefits on some cognitive measures without consistent effect on global function or activities of daily living. Diabetic peripheral neuropathy data came from two pooled trials reported by Sima and colleagues [sima2005] [hiatt2011]. Male-infertility data developed through several Italian and international trials [lenzi2003, cavallini2005]. Geriatric fatigue and centenarian trials by Malaguarnera and colleagues [malaguarnera2007, malaguarnera2008] and MS-fatigue work [tomassini2004] addressed fatigue-related contexts. The dialysis-associated indication was operationalized by the NKF Carnitine Consensus Conference [eknoyan2003] [brevetti1999; hiatt2001].

The gut-microbiota, TMAO axis emerged with Wang et al. 2011 on dietary choline [wang2011] and Koeth et al. 2013 on L-carnitine [koeth2013] in Nature Medicine, linking dietary L-carnitine intake to trimethylamine-N-oxide generation and atherosclerosis in mice and observational human cohorts. The DiNicolantonio (2013) meta-analysis [dinicolantonio2013] of L-carnitine after acute MI was published in the same period and reported a 27% reduction in all-cause mortality across 13 trials and 3,629 participants, with the authors framing the apparent benefit-risk balance as favorable while acknowledging the TMAO mechanistic concern [delaney2013].

L-Carnitine Timeline

1905 • Gulewitsch and Krimberg isolate carnitine from muscle extracts

1948 • Carter and colleagues identify carnitine as the mealworm growth factor 'vitamin BT'

1960s • Fritz and Bremer establish carnitine's role in mitochondrial long-chain fatty-acid transport [bremer1990]

1985 • FDA approves oral levocarnitine (Carnitor) for primary carnitine deficiency under orphan-drug designation [fda_label_carnitor]

1990 • Bremer publishes definitive review of intracellular carnitine metabolism in J Clin Chem Clin Biochem [bremer1990]

1991 • Spagnoli et al [spagnoli1991]. publish long-term acetyl-L-carnitine trial in Alzheimer disease in Neurology

1992 • Mancini et al [mancini1992; fda_label_carnitor]. publish controlled trial of propionyl-L-carnitine in congestive heart failure; FDA approves intravenous Carnitor

1994 • Salvioli and Neri publish acetyl-L-carnitine trial in mental decline in the elderly [salvioli1994]

1995 • Iliceto et al. publish CEDIM trial, oral L-carnitine attenuates left-ventricular remodeling after acute anterior MI; Pettegrew et al [iliceto1995]. publish acetyl-L-carnitine MRS substudy in Alzheimer disease; Brevetti et al [pettegrew1995; brevetti1995]. publish PLC double-blind dose-titration trial in intermittent claudication

1997 • Famularo et al. report acetylcarnitine deficiency in AIDS patients on nucleoside analogues; Brevetti et al [famularo1997; brevetti1997]. report PLC quality-of-life effects in claudication

1999 • Brevetti et al [brevetti1999; fda_label_carnitor]. publish European multicenter PLC trial in intermittent claudication; Carnitor indication extended to ESRD hemodialysis

2000 • Rizos publishes three-year survival data for L-carnitine in dilated cardiomyopathy; Brass publishes review of supplemental carnitine and exercise [rizos2000; brass2000]

2001 • Hiatt et al [hiatt2001]. publish PLC trial showing improved exercise performance and functional status in claudication

2002 • Vaz and Wanders publish review of mammalian carnitine biosynthesis in Biochem J [vaz2002]

2003 • Hudson and Tabet publish Cochrane review of acetyl-L-carnitine for dementia; NKF Carnitine Consensus Conference (Eknoyan et al.) publishes practice recommendations for levocarnitine in dialysis [hudson2003; eknoyan2003]

2003 • Lenzi et al [lenzi2003]. publish L-carnitine and acetyl-L-carnitine RCT in idiopathic asthenozoospermia in Fertility and Sterility

2004 • Tomassini et al [tomassini2004; rebouche2004]. compare ALC with amantadine for MS-related fatigue; Rebouche reviews carnitine and acetyl-L-carnitine kinetics

2005 • Sima et al. publish pooled ALC trials in chronic diabetic peripheral neuropathy; Cavallini et al [sima2005]. publish ALC+PLC+sildenafil RCT after radical prostatectomy; Lheureux et al [cavallini2005; lheureux2005]. publish first Critical Care review of carnitine in valproate toxicity

2007 • Malaguarnera et al [malaguarnera2007]. report L-carnitine RCT in centenarian fatigue in Am J Clin Nutr

2008 • Malaguarnera et al [malaguarnera2008]. report ALC trial in elderly fatigue

2009 • Lheureux and Hantson publish second review of carnitine in valproate toxicity in Clin Toxicol [lheureux2009]

2011 • Wall et al. demonstrate that chronic oral L-carnitine plus carbohydrate raises muscle carnitine content and alters fuel metabolism during exercise; Wang et al [wall2011]. link gut-microbiota choline metabolism to TMAO and CV disease in Nature; Hiatt et al [wang2011; hiatt2011]. publish PLC plus monitored exercise in PAD

2012 • Magoulas and El-Hattab review systemic primary carnitine deficiency in Orphanet J Rare Dis [magoulas2012]

2013 • Koeth et al. report gut-microbiota metabolism of L-carnitine to TMAO and atherosclerosis in Nature Medicine; Marcovina et al [koeth2013]. review translation of mitochondrial carnitine biology to metabolic therapy; DiNicolantonio et al [marcovina2013]. publish meta-analysis of L-carnitine after acute MI; Delaney et al [dinicolantonio2013; delaney2013; malaguarnera2013]. publish PLC claudication meta-analysis; Malaguarnera reviews ALC in hepatic encephalopathy

2017 • Longo et al [longo2017]. publish functional and molecular studies in primary carnitine deficiency

2019 • Longo et al [longo2019]. publish review of carnitine inborn errors of metabolism

Clinical Contexts for L-Carnitine

Primary systemic carnitine deficiency (OCTN2/SLC22A5) FDA APPROVED

FDA-approved indication; carnitine replacement is the standard of care.

Primary carnitine deficiency is an autosomal recessive disorder caused by loss-of-function mutations in SLC22A5 (the OCTN2 transporter), producing cardiomyopathy, hypoketotic hypoglycemia, and skeletal myopathy [magoulas2012; longo2017]. Lifelong oral or intravenous levocarnitine restores tissue carnitine pools and prevents the cardiac and metabolic complications of the disease [fda_label_carnitor]. Newborn screening detects the condition presymptomatically in most U.S. states via low free carnitine on tandem mass spectrometry [longo2019].

Branded product: Carnitor

Secondary carnitine deficiency from inborn errors of metabolism FDA APPROVED

FDA-approved indication for the intravenous formulation.

Inborn errors of fatty-acid oxidation and organic acidemias (medium-chain acyl-CoA dehydrogenase deficiency, very-long-chain acyl-CoA dehydrogenase deficiency, propionic acidemia, methylmalonic acidemia, isovaleric acidemia, and others) can produce secondary carnitine deficiency through accumulation of acylcarnitines and renal loss [longo2019]. Acute and chronic levocarnitine replacement is part of the standard metabolic-disease management regimen [fda_label_carnitor].

Branded product: Carnitor injection

Carnitine deficiency in ESRD on hemodialysis FDA APPROVED

FDA-approved indication; NKF Consensus Conference codified the dosing regimen.

Hemodialysis removes carnitine and produces a deficiency phenotype that includes erythropoietin-resistant anemia, intradialytic hypotension, skeletal-muscle weakness, and (in some patients) cardiomyopathy. The NKF Carnitine Consensus Conference recommends intravenous levocarnitine 20 mg/kg administered slowly into the venous return of the dialyzer after each session, with reassessment of clinical response at 3, 6 months and continuation only on documented response [eknoyan2003; fda_label_carnitor]. CMS reimbursement requires documented deficiency or specific clinical indication.

Branded product: Carnitor injection

Valproic acid, induced hepatotoxicity, hyperammonemia, and overdose WELL STUDIED

Widely used off-label; supported by case series, observational data, and poison-center guidance.

Valproate depletes free carnitine, inhibits long-chain fatty-acid oxidation, and can precipitate hyperammonemic encephalopathy and hepatotoxicity. Intravenous levocarnitine 100 mg/kg loading then 15 mg/kg every 4 hours is the conventional rescue regimen in symptomatic toxicity. The Critical Care and Clin Toxicol reviews by Lheureux and Hantson summarize the evidence base, which is observational rather than randomized but consistently supportive in severe toxicity [lheureux2005; lheureux2009].

Cardiac ischemia and post-MI left-ventricular remodeling WELL STUDIED

Off-label; multiple controlled trials and a positive meta-analysis with the TMAO observation as a counterweight.

The CEDIM trial randomized 472 adults to oral L-carnitine 9 g/day for 5 days then 6 g/day for 12 months versus placebo within 24 h of acute anterior MI and reported attenuated left-ventricular dilation at 6 and 12 months on serial echocardiography [iliceto1995]. The DiNicolantonio (2013) meta-analysis of 13 controlled trials (3,629 participants) reported a 27% reduction in all-cause mortality, a 65% reduction in ventricular arrhythmias, and a 40% reduction in anginal symptoms after acute MI [dinicolantonio2013]. The Koeth (2013) and Wang (2011) work on the gut-microbiota, TMAO axis is a mechanistic counterweight: chronic dietary carnitine exposure generates TMAO that may accelerate atherosclerosis in apoE-/- mice and correlates with CV events in observational cohorts [koeth2013; wang2011]. The clinical implications of TMAO biology for short- to medium-term levocarnitine therapy after MI remain unresolved.

Heart failure with reduced ejection fraction (PLC) EMERGING

Off-label; small randomized trials with mixed signals.

Propionyl-L-carnitine 1.5, 3 g/day orally has been studied as adjunctive therapy in chronic heart failure [mancini1992]. Mancini (1992) reported improved exercise duration in a controlled trial. Rizos (2000) reported a survival advantage at three years in 80 patients with dilated cardiomyopathy on PLC versus placebo on background therapy [rizos2000]. Trial sizes are small and standard-of-care has evolved substantially in the intervening decades; modern guidelines do not include PLC.

Intermittent claudication (peripheral artery disease) WELL STUDIED

Off-label; PLC ester improves walking distance in multiple multicenter trials and a meta-analysis.

Propionyl-L-carnitine 1, 2 g twice daily orally has been investigated in adults with intermittent claudication from peripheral arterial disease [brevetti1995; brevetti1997; hiatt2011]. Brevetti (1995) reported dose-titration efficacy on pain-free walking distance. The Brevetti (1999) European multicenter trial in 245 patients reported significant improvements in maximum walking distance and quality-of-life measures versus placebo [brevetti1999]. Hiatt (2001) reported improved functional status [hiatt2001]. Hiatt (2011) tested PLC against monitored exercise. The Delaney (2013) systematic review and meta-analysis of randomized PLC claudication trials reported a moderate but consistent improvement in pain-free and maximum walking distance [delaney2013].

Male infertility (idiopathic asthenozoospermia) WELL STUDIED

Off-label; randomized trials report improvement in sperm motility parameters.

Lenzi (2003) randomized 100 men with idiopathic asthenozoospermia to combined L-carnitine 2 g/day plus acetyl-L-carnitine 1 g/day or placebo for 6 months and reported improvements in sperm concentration, motility, and acrosin activity. Cavallini (2005) randomized men after bilateral nerve-sparing radical retropubic prostatectomy to acetyl-L-carnitine plus propionyl-L-carnitine with sildenafil versus sildenafil alone and reported improved erectile-function outcomes [cavallini2005]. Andrology guidelines list carnitine and acetyl-L-carnitine among nutraceutical options for idiopathic male-factor infertility [lenzi2003].

Alzheimer disease and age-related cognitive decline EMERGING

Off-label; Cochrane review identified small benefits on some scales without consistent global effect.

Acetyl-L-carnitine 1.5, 3 g/day orally has been studied in mild-to-moderate Alzheimer disease and age-related cognitive decline [pettegrew1995; spagnoli1991]. Pettegrew (1995) reported phosphorus magnetic-resonance spectroscopy and neuropsychological-test changes in a small placebo-controlled cohort. Spagnoli (1991) reported long-term ALC effects on cognitive scales. Salvioli (1994) tested ALC in non-Alzheimer mental decline [salvioli1994]. The 2003 Cochrane review by Hudson and Tabet identified small benefits on some cognitive measures without consistent global effect on activities of daily living; current Alzheimer guidelines do not include ALC [hudson2003].

Chronic diabetic peripheral neuropathy WELL STUDIED

Off-label; pooled randomized trials report improved pain and nerve-conduction outcomes.

Sima and colleagues (2005) analyzed two parallel randomized placebo-controlled trials of acetyl-L-carnitine 500 mg or 1000 mg three times daily orally for 52 weeks in 1,257 patients with chronic diabetic peripheral neuropathy [sima2005]. Pooled analysis showed improvements in vibratory perception, pain scores, and sural-nerve regeneration markers on nerve biopsy, with greater effect at the higher dose. ALC is not FDA-approved for diabetic neuropathy and is not a first-line therapy in modern diabetic-neuropathy guidelines.

Hepatic encephalopathy EMERGING

Off-label; small randomized trials and reviews report modest benefit.

Acetyl-L-carnitine has been studied as adjunctive therapy in minimal and overt hepatic encephalopathy [malaguarnera2013]. Malaguarnera (2013) reviewed a small randomized-trial series reporting improvements in psychometric performance and ammonia levels with oral ALC; trial sizes are small and replication is limited.

Fatigue (elderly, centenarian, multiple sclerosis) EMERGING

Off-label; small randomized trials with positive signals on patient-reported fatigue.

Malaguarnera (2007) randomized 66 centenarians to L-carnitine 2 g/day versus placebo for 6 months and reported reductions in total fat mass, increases in total muscle mass, and reductions in physical and mental fatigue [malaguarnera2007]. Malaguarnera (2008) reported ALC effects on fatigue in elderly outpatients [malaguarnera2008]. Tomassini (2004) compared ALC and amantadine in a small crossover trial in MS-related fatigue and reported ALC tolerability comparable to amantadine with comparable fatigue improvements [tomassini2004].

Exercise performance and muscle carnitine loading EMERGING

Off-label; physiological evidence of muscle loading with co-administered carbohydrate; clinical-outcome data limited.

Wall (2011) demonstrated that 24 weeks of oral L-carnitine 2 g/day plus 80 g carbohydrate twice daily raised skeletal-muscle total carnitine content by approximately 21% and altered fuel utilization during low- and high-intensity exercise in healthy adults [wall2011]. Brass (2000) reviewed supplemental carnitine and exercise and concluded that performance benefits in non-deficient adults are inconsistent [brass2000]. Most exercise-performance work uses OTC supplement preparations rather than prescription levocarnitine.

Antiretroviral-associated neurotoxicity EMERGING

Off-label; case-series evidence of acetylcarnitine deficiency and clinical improvement with replacement.

Famularo (1997) reported acetylcarnitine deficiency in patients with HIV on nucleoside analogue reverse-transcriptase inhibitors who developed peripheral neuropathy, with clinical improvement on acetyl-L-carnitine replacement [famularo1997]. Modern antiretroviral regimens have largely replaced the implicated agents (stavudine, didanosine, zalcitabine) and the contemporary relevance is therefore limited; the historical literature remains a mechanistic data point on acquired acetylcarnitine depletion.

FDA-Approved Uses of L-Carnitine

Brand	Indication	Year	Route
Carnitor (levocarnitine) oral solution and tablets	Treatment of primary systemic carnitine deficiency	1985	Oral
Carnitor (levocarnitine) injection	Acute and chronic treatment of patients with an inborn error of metabolism that results in secondary carnitine deficiency; prevention and treatment of carnitine deficiency in end-stage renal disease patients on hemodialysis	1992	Intravenous

Levocarnitine (Carnitor; originally Sigma-Tau, now Leadiant Biosciences) is the FDA-approved prescription form of L-carnitine. The oral solution and tablets are approved for the treatment of primary systemic carnitine deficiency (first approved 1985). The intravenous injection is approved for acute and chronic treatment of patients with an inborn error of metabolism that results in secondary carnitine deficiency (1992 approval) and for the prevention and treatment of carnitine deficiency in end-stage renal disease patients on hemodialysis (indication expanded 1999) [fda_label_carnitor, dailymed_levocarnitine].

The labeled treatment for ESRD-associated carnitine deficiency derives from the National Kidney Foundation Carnitine Consensus Conference recommendation of intravenous levocarnitine 20 mg/kg administered slowly into the venous line of the dialyzer after each hemodialysis session, with reassessment

at 3, 6 months and continuation only if a documented response to therapy occurs [eknoyan2003]. Use in valproic acid, induced hepatotoxicity, hyperammonemia, and overdose is widely accepted and incorporated into poison-center guidance [lheureux2009, lheureux2005] although the FDA-approved labeling does not formally include valproate toxicity as a labeled indication for Carnitor.

Numerous OTC L-carnitine and acetyl-L-carnitine products are marketed in the United States as dietary supplements under the Dietary Supplement Health and Education Act (DSHEA). These products are not regulated as drugs and are not interchangeable with prescription Carnitor; ingredient quality, label-claim accuracy, and indication framing differ materially.

Compounded L-Carnitine (503A)

Compounded levocarnitine is dispensed under 503A only on a patient-specific prescription written by a licensed prescribing physician for an identified patient. The manufactured Carnitor product (oral solution, oral tablets, and intravenous injection) is available for the labeled indications, and a compounded preparation is appropriate only when the prescribing clinician documents that the manufactured product cannot meet a specific clinical need [fda503a] [fda_label_carnitor].

Common 503A compounded carnitine preparations include: (1) custom-strength intravenous levocarnitine for IV infusion protocols when the patient's clinical regimen requires a concentration, volume, or excipient profile that the manufactured 1 g per 5 mL injection cannot provide; (2) intramuscular levocarnitine where the clinician documents that intravenous access is impractical and the patient cannot tolerate oral therapy; (3) sublingual troches in patients with severe gastrointestinal intolerance to the labeled oral solution or who require taste-masked or sucrose-free formulations; and (4) multi-ingredient blends combining levocarnitine with B-vitamins (B12, B6, B-complex), other amino acids, or alpha-lipoic acid where the prescribing clinician documents a clinical rationale that an FDA-approved single-ingredient product cannot meet [fda_label_carnitor].

The honest framing for patients and prescribers: (a) FDA-approved Carnitor exists and should be the first-line consideration for the labeled indications; (b) numerous OTC L-carnitine and acetyl-L-carnitine dietary supplements are widely available under DSHEA, are not regulated as drugs, and are not interchangeable with prescription Carnitor or with compounded preparations; (c) compounded 503A levocarnitine is patient-specific, not direct-to-consumer, and is not a generic substitute for either the manufactured drug or the OTC supplements [fda_label_carnitor].

L-Carnitine Formulations and Routes

Form	Concentration	Description
Manufactured oral solution (reference product)	100 mg/mL (1 g per 10 mL)	Carnitor and Carnitor SF oral solution. SF formulation is sucrose-free for patients with carbohydrate-intolerance considerations.
Manufactured oral tablet (reference product)	330 mg	Carnitor 330 mg tablet; alternative to oral solution for patients who prefer solid dosage form.
Manufactured intravenous injection (reference product)	200 mg/mL (1 g per 5 mL single-dose vial)	Carnitor injection for IV slow bolus or for administration into the venous return of a hemodialysis circuit.
Compounded sterile intravenous solution	Custom (commonly 200 mg/mL or diluted for infusion bags)	Sterile injectable solution prepared under USP <797> for IV infusion protocols when the manufactured concentration, volume, or excipient profile is not suitable for the patient's prescribed regimen.
Compounded intramuscular injection	Custom (typically 200 mg/mL)	Sterile injectable solution prepared under USP <797> for IM administration when documented oral intolerance and inability to use IV access justify the route.
Compounded sublingual troche	Typically 250, 1000 mg per troche	Sublingual troche prepared under USP <795> for patients with severe oral-route gastrointestinal intolerance to the manufactured oral solution or tablet.
Compounded multi-ingredient blend (oral, IV, or IM)	Custom	Combination preparations of levocarnitine with B-vitamins, additional amino acids, or alpha-lipoic acid where the prescribing clinician documents a patient-specific rationale that FDA-approved single-ingredient products cannot meet.

Routes used in published literature: oral, intravenous, intramuscular, sublingual, troche.

L-Carnitine Dosing

Route	Population	Range	Duration	Study type
oral	Adults with primary or secondary carnitine	1, 3 g/day in divided doses; titrated to clinical	Indefinite while clinically beneficial	FDA-approved labeled regimen

Route	Population	Range	Duration	Study type
	deficiency (labeled regimen)	response and plasma carnitine concentrations		
oral	Pediatric primary carnitine deficiency (labeled regimen)	50, 100 mg/kg/day in divided doses (typical starting); titrated to a maximum of 3 g/day per labeling	Indefinite while clinically beneficial	FDA-approved labeled regimen
intravenous	ESRD on hemodialysis (NKF consensus regimen)	20 mg/kg slow IV into the venous return after each dialysis session	Reassess clinical response at 3, 6 months; continue only on documented response	NKF Consensus Conference recommendation
intravenous	Valproate-induced hepatotoxicity / hyperammonemia / overdose (off-label rescue)	100 mg/kg IV loading dose (up to 6 g), then 15 mg/kg IV every 4 hours until clinical and biochemical improvement	Until ammonia and hepatic indices normalize	Observational, case series, poison-center guidance
oral	Adults after acute anterior MI (CEDIM regimen)	L-carnitine 9 g/day for the first 5 days, then 6 g/day for 12 months	12 months	Randomized double-blind placebo-controlled (CEDIM)
oral	Adults with intermittent claudication (PLC trial regimens)	Propionyl-L-carnitine 1, 2 g twice daily orally	6 months to 1 year in trial protocols	Multicenter randomized controlled trials
oral	Men with idiopathic asthenozoospermia (Lenzi regimen)	L-carnitine 2 g/day plus acetyl-L-carnitine 1 g/day	6 months	Randomized double-blind placebo-controlled
oral	Adults with mild-to-moderate Alzheimer disease (ALC trial regimens)	Acetyl-L-carnitine 1.5, 3 g/day in divided doses	6 months to 1 year in trial protocols	Randomized controlled trials and Cochrane review
oral	Adults with chronic diabetic peripheral neuropathy (Sima 2005 regimen)	Acetyl-L-carnitine 500 mg or 1000 mg three times daily	52 weeks	Pooled analysis of two randomized placebo-controlled trials

Route	Population	Range	Duration	Study type
oral	Centenarians with physical and mental fatigue (Malaguarnera regimen)	L-carnitine 2 g/day	6 months	Randomized double-blind placebo-controlled

Doses listed reflect FDA labeling or published clinical-trial protocols, not RonanRx prescribing recommendations. The prescribing doctor selects route, dose, frequency, and ester (L-carnitine, acetyl-L-carnitine, or propionyl-L-carnitine) based on the patient's indication, clinical context, and goals [rebouche2004].

Levocarnitine oral bioavailability is approximately 15% from oral solution and is dose-dependent (saturable intestinal transport) [fda_label_carnitor]. Intravenous administration bypasses this limitation. For chronic deficiency states the labeled oral regimen is typically titrated against clinical response and plasma carnitine [eknoyan2003]. For the dialysis indication the NKF post-dialysis IV protocol is the operative standard. Off-label regimens in the cardiac, vascular, neurological, and reproductive contexts reflect the cited trial protocols and should not be treated as universally established.

Doses listed should not be presented to patients as instructions. Patient instructions originate from the prescribing physician's prescription, not from this educational page.

L-Carnitine Safety

Levocarnitine has been generally well tolerated across decades of clinical use in deficiency states and in the off-label cardiac, vascular, neurological, and reproductive trials. The most common reported adverse effects are gastrointestinal, nausea, vomiting, abdominal cramping, and diarrhea, typically dose-dependent and most prominent with higher oral doses or oral solution administered without food. A characteristic transient fishy body odor (trimethylamine) is reported in some patients and is generated by gut-microbiota metabolism of unabsorbed carnitine [37][36].

Seizures have been reported in patients with and without pre-existing seizure disorders receiving levocarnitine, both oral and intravenous; the labeled warning notes that an increase in seizure frequency or new seizure onset has been observed in patients with prior seizure history. Intravenous administration is associated with rare hypotension, especially in hemodialysis patients receiving rapid infusion [37][8].

The TMAO observation [36][35] is a long-term cardiovascular consideration rather than an acute safety signal. Chronic dietary L-carnitine exposure can generate gut-microbiota-derived TMAO that has been associated with atherosclerosis in animal models and observational human cohorts. Whether short- to medium-term therapeutic levocarnitine in approved indications carries clinically meaningful TMAO-mediated CV risk is unresolved; the DiNicolantonio (2013) meta-analysis of L-carnitine after acute MI reported reduced all-cause mortality despite the TMAO mechanistic concern [13].

Contraindications

The labeled contraindications for Carnitor are limited. Known hypersensitivity to levocarnitine or any excipient in the prescribed formulation is a contraindication for that preparation. The D-isomer of carnitine (DL-carnitine, racemic) is not appropriate for therapeutic use because it competitively inhibits OCTN2-mediated uptake of the active L-isomer; prescription product is L-carnitine only [37].

Drug interactions

Valproic acid depletes carnitine through valproylcarnitine excretion, inhibition of biosynthesis, and CPT1 inhibition; this is the basis for the off-label use of levocarnitine in valproate toxicity rather than a contraindication to combined use [910]. Pivalate-containing antibiotics (pivampicillin, pivmecillinam) and certain antiretroviral nucleoside analogues (stavudine, didanosine, zalcitabine) can produce acquired acetylcarnitine deficiency through analogous mechanisms [11].

Anticoagulant interaction: case reports describe increased INR in patients on warfarin after starting oral L-carnitine; clinically meaningful interaction is uncommon but anticoagulant monitoring is reasonable when initiating or stopping therapy.

Thyroid: pharmacological doses of L-carnitine have been reported to attenuate thyroid hormone action at the cellular level in some studies; this is a theoretical consideration in hyperthyroid states rather than an established clinical interaction at typical levocarnitine doses.

Adverse events

Common adverse events reported in clinical trials and post-marketing surveillance: gastrointestinal symptoms (nausea, vomiting, abdominal cramps, diarrhea), particularly with higher oral doses; transient fishy body odor (trimethylaminuria-like) from gut-microbiota metabolism of unabsorbed carnitine; and mild myasthenia in uremic patients receiving the dialysis-deficiency regimen [378] [36].

Uncommon adverse events: increased seizure frequency or new seizures in patients with prior seizure history (labeled warning); hypotension with rapid IV administration in hemodialysis patients; hypersensitivity reactions; and rare reports of arrhythmia [36]. Long-term cardiovascular concern from gut-microbiota-derived TMAO is a separate consideration discussed in the safety overview.

↗ Monitoring L-Carnitine Therapy

For chronic levocarnitine therapy in deficiency states, monitoring includes plasma free carnitine and the acylcarnitine/free-carnitine ratio (by tandem mass spectrometry), clinical assessment of disease-specific markers (cardiomyopathy on echocardiogram for primary deficiency, glycemic and lipid markers in fatty-acid oxidation disorders), and tolerability surveillance for GI symptoms and body-odor complaints [fda_label_carnitor].

For the dialysis indication, the NKF Consensus Conference recommends reassessment of clinical response at 3, 6 months, symptomatic improvement in erythropoietin-resistant anemia, intradialytic hypotension, or skeletal-muscle symptoms, with continuation only on documented response [eknoyan2003].

For valproate-induced toxicity rescue, monitoring follows the standard toxicology workup: serum ammonia, hepatic transaminases, valproate level, blood gas, and clinical mental status [lheureux2009].

L-Carnitine in Special Populations

L-Carnitine Evidence Quality

Carnitine biochemistry, the mitochondrial fatty-acid-transport function, the OCTN2 transporter biology, and the metabolic consequences of deficiency are well established by decades of biochemical, genetic, and clinical research [vaz2002; magoulas2012; longo2017; brevetti1999]. FDA-approved indications, primary systemic deficiency, secondary deficiency from inborn errors of metabolism, and ESRD-associated deficiency on hemodialysis, are supported by replicated trial data and consensus practice recommendations [fda_label_carnitor, eknoyan2003] [bremer1990; hiatt2001].

Off-label evidence varies by indication [longo2019; marcovina2013; brevetti1995]. Strongest: post-MI cardiac remodeling (CEDIM plus DiNicolantonio meta-analysis, with the TMAO mechanistic counterweight) [iliceto1995, dinicolantonio2013, koeth2013]; intermittent claudication with PLC (multiple multicenter trials and meta-analysis); diabetic peripheral neuropathy with ALC (pooled trials, n=1,257) [sima2005]; male infertility (multiple RCTs in idiopathic asthenozoospermia) [lenzi2003, cavallini2005]; and valproate-toxicity rescue (observational and case-series evidence consistently supportive) [lheureux2009, lheureux2005]. Moderate-to-mixed: Alzheimer disease (Cochrane review identifies small benefits without consistent global effect) [hudson2003, pettegrew1995, spagnoli1991]; heart failure with PLC (small trials, mixed) [mancini1992, rizos2000]; hepatic encephalopathy [malaguarnera2013]; fatigue (small randomized trials) [malaguarnera2007, malaguarnera2008, tomassini2004]; exercise performance (physiological loading demonstrated; clinical-outcome benefit inconsistent in non-deficient adults) [wall2011, brass2000].

The TMAO hypothesis [koeth2013, wang2011] is a long-term mechanistic concern affecting interpretation of chronic carnitine therapy but has not produced trial-level evidence of harm at typical therapeutic doses; the largest cardiac meta-analysis to date [dinicolantonio2013] reported net mortality benefit after acute MI despite the mechanistic concern [delaney2013].

Major L-Carnitine Clinical Studies

Study	Design	Participants	Duration	Finding
Effects of L-carnitine administration on left ventricular remodeling after acute anterior myocardial infarction (CEDIM)	Randomized double-blind placebo-controlled multicenter	472	12 months	Oral L-carnitine (9 g/day for 5 days, then 6 g/day) initiated within 24 hours of acute anterior MI attenuated progressive left-ventricular dilation on serial echocardiography at 6 and 12 months versus placebo [iliceto1995].
L-carnitine in the secondary prevention of cardiovascular disease: systematic review and meta-analysis	Systematic review and meta-analysis of 13 controlled trials	3,629 pooled	varied	L-carnitine after acute MI was associated with a 27% reduction in all-cause mortality, a 65% reduction in ventricular arrhythmias, and a 40% reduction in anginal symptoms [dinicolantonio2013].
Intestinal microbiota metabolism of L-carnitine, a nutrient in red meat, promotes atherosclerosis	Mechanistic (mouse, human cohort) and observational	varied	varied	Gut-microbiota metabolism of dietary L-carnitine generates trimethylamine-N-oxide (TMAO), which accelerated atherosclerosis in apoE-/- mice and correlated with cardiovascular events in observational human cohorts [koeth2013].
European multicenter study on propionyl-L-carnitine in intermittent claudication	Randomized double-blind placebo-controlled multicenter	245	6 months	Oral PLC 2 g/day improved maximum walking distance and quality-of-life measures versus placebo in adults with Fontaine II PAD [brevetti1999].
Propionyl-L-carnitine in intermittent claudication: double-blind, placebo-controlled, dose titration, multicenter study	Randomized double-blind placebo-controlled dose-titration multicenter	varied	varied	Dose-titration confirmed PLC 2 g/day as the effective regimen for improvement in pain-free walking distance in claudication [brevetti1995].

Study	Design	Participants	Duration	Finding
A systematic review and meta-analysis of propionyl-L-carnitine effects on exercise performance in patients with claudication	Systematic review and meta-analysis	pooled	varied	PLC produced consistent improvements in pain-free and maximum walking distance across randomized claudication trials [delaney2013].
Use of carnitine and acetyl-carnitine in the treatment of idiopathic asthenozoospermia (Lenzi 2003)	Randomized double-blind placebo-controlled	100	6 months	Combined oral L-carnitine 2 g/day plus acetyl-L-carnitine 1 g/day improved sperm concentration, motility, and acrosin activity versus placebo [lenzi2003].
Acetyl-L-carnitine plus propionyl-L-carnitine improve efficacy of sildenafil after bilateral nerve-sparing radical retropubic prostatectomy	Randomized controlled trial	varied	varied	Adjunctive ALC+PLC improved erectile-function response to sildenafil after radical prostatectomy versus sildenafil alone [cavallini2005].
Acetyl-L-carnitine improves pain, nerve regeneration, and vibratory perception in chronic diabetic neuropathy	Pooled analysis of two randomized double-blind placebo-controlled trials	1257	52 weeks	ALC 500 mg or 1000 mg three times daily improved vibratory perception, pain scores, and sural-nerve regeneration markers in chronic diabetic peripheral neuropathy, with greater effect at the higher dose [sima2005].
Long-term acetyl-L-carnitine treatment in Alzheimer's disease (Spagnoli 1991)	Randomized double-blind placebo-controlled	varied	12 months	Long-term ALC produced significant effects on selected cognitive scales versus placebo in mild-to-moderate Alzheimer disease [spagnoli1991].
Acetyl-L-carnitine effects in early Alzheimer's disease (Pettegrew 1995)	Randomized double-blind placebo-controlled with 31P-MRS substudy	varied	12 months	ALC produced changes in 31P-MRS brain metabolic markers and on selected neuropsychological measures in early Alzheimer disease [pettegrew1995].
		pooled	varied	

Study	Design	Participants	Duration	Finding
Acetyl-L-carnitine for dementia (Cochrane review)	Systematic review and meta-analysis			ALC produced small benefits on some cognitive measures across pooled trials, without consistent effect on global function or activities of daily living [hudson2003].
Three-year survival of patients with heart failure caused by dilated cardiomyopathy and L-carnitine administration	Randomized placebo-controlled	80	3 years	L-carnitine produced a survival advantage at 3 years versus placebo in adults with dilated cardiomyopathy on background therapy [rizos2000].
Controlled study of propionyl-L-carnitine in congestive heart failure	Randomized double-blind placebo-controlled	varied	varied	PLC improved exercise duration and selected hemodynamic markers in chronic heart failure [mancini1992].
L-carnitine treatment in centenarians (Malaguarnera 2007)	Randomized double-blind placebo-controlled	66	6 months	L-carnitine 2 g/day reduced physical and mental fatigue, increased lean mass, and reduced fat mass in centenarians [malaguarnera2007].
Chronic oral ingestion of L-carnitine and carbohydrate increases muscle carnitine content in humans	Randomized controlled	varied	24 weeks	L-carnitine 2 g/day plus carbohydrate raised skeletal-muscle total carnitine by approximately 21% and altered fuel utilization during low- and high-intensity exercise [wall2011].
Practice recommendations for the use of L-carnitine in dialysis-related carnitine disorder (NKF Carnitine Consensus Conference)	Consensus practice recommendation	n/a	n/a	Consensus regimen of IV levocarnitine 20 mg/kg post-dialysis with reassessment at 3, 6 months, continuation only on documented response [eknoyan2003].
Science review: carnitine in the treatment of valproic acid-induced toxicity	Narrative review of observational evidence	n/a	n/a	Levocarnitine is supported as rescue therapy for valproate-induced hepatotoxicity, hyperammonemic encephalopathy, and overdose,

Study	Design	Participants	Duration	Finding
				based on case-series and observational data [lheureux2005; lheureux2009].
Systemic primary carnitine deficiency: clinical manifestations, diagnosis, and management	Review	n/a	n/a	Primary deficiency (SLC22A5 mutations) presents with cardiomyopathy, hypoketotic hypoglycemia, and skeletal myopathy; lifelong levocarnitine replacement is the standard of care [magoulas2012].

L-Carnitine Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Oral bioavailability of levocarnitine from the labeled oral solution is approximately 15% in healthy adults, with saturable intestinal transport; higher single doses produce proportionally lower absorbed fractions [rebouche2004, fda_label_carnitor]. Intravenous administration bypasses this limitation and is used in deficiency states with severe depletion.

Distribution is into total body water with high tissue accumulation in skeletal and cardiac muscle via OCTN2-mediated transport. Plasma protein binding is minimal (<5%). Free L-carnitine in plasma is normally 25, 50 μM; the acylcarnitine/free-carnitine ratio is <0.4 in healthy adults.

Elimination is predominantly renal, with extensive tubular reabsorption via OCTN2 such that >95% of filtered carnitine is normally retained in non-deficient adults. Hemodialysis removes free carnitine and short-chain acylcarnitines at clearances approaching dialyzer urea clearance, which is the basis for the dialysis-associated deficiency. Plasma half-life after IV administration in healthy adults is approximately 17.4 hours [rebouche2004, fda_label_carnitor].

Pharmacodynamics

The pharmacodynamic effect of levocarnitine is restoration of tissue free-carnitine pools sufficient to support the carnitine palmitoyltransferase shuttle and to buffer the acyl-CoA/CoA ratio under the metabolic stress of the clinical context. In deficiency states this is a true replacement effect with measurable plasma and tissue carnitine restoration. In off-label cardiac, vascular, and reproductive contexts the proposed pharmacodynamic mechanism is presumed enhancement of fatty-acid oxidation under ischemic, oxidative, or substrate-limited conditions [bremer1990, marcovina2013].

Acetyl-L-carnitine and propionyl-L-carnitine differ from L-carnitine in tissue distribution and metabolic fate. ALC crosses the blood-brain barrier more efficiently and supplies acetyl-CoA in neurons. PLC is preferentially taken up by skeletal and cardiac muscle and provides propionyl-CoA as an anaplerotic substrate for the citric acid cycle. The ester selection in off-label trial design reflects these tissue and substrate considerations [pettegrew1995, brevetti1999].

↓↑ Comparing L-Carnitine Formulations

Manufactured Carnitor is supplied as oral solution 100 mg/mL (1 g per 10 mL, with sucrose-free SF formulation available), oral tablet 330 mg, and intravenous injection 200 mg/mL (1 g per 5 mL single-dose vial). The oral solution is the typical pediatric and starting adult formulation; tablets are an alternative for adult preference; the injection is reserved for the IV indications including the NKF post-dialysis regimen [fda_label_carnitor, dailymed_levocarnitine, eknoyan2003].

Compounded preparations vary in concentration, container closure, and excipient profile relative to the manufactured product. Sterile injectable compounded levocarnitine is dispensed only when the prescriber documents a clinical reason that the manufactured concentration or excipient is not appropriate. Sublingual troches, IM injection, and multi-ingredient blends are dispensed only when the prescriber documents an analogous patient-specific rationale.

Acetyl-L-carnitine and propionyl-L-carnitine are not FDA-approved drug products in the United States. They are widely sold as dietary supplements under DSHEA and are also available as 503A compounded preparations on patient-specific prescription. Trial-grade ALC and PLC have been used in the cited cardiac, vascular, neurological, and reproductive trials; OTC supplement-grade products are not standardized to those trial preparations.

🌡 L-Carnitine Storage and Handling

Manufactured Carnitor oral solution and tablets are stored at controlled room temperature (20, 25 °C) in the original container with appropriate light protection per labeling. Carnitor injection is stored at controlled room temperature; once a single-dose vial is opened, any unused portion should be discarded [fda_label_carnitor].

Compounded sterile injectable preparations are stored per the pharmacy's stability data and beyond-use dating under USP <797>. Compounded sublingual troches are stored at controlled room temperature per USP <795>. Patients receive labeling specific to the preparation dispensed.

L-Carnitine Compounding & Operations

503A compounding

RonanRx prepares levocarnitine under 503A on patient-specific prescriptions in state-licensed compounding pharmacies. Sterile injectable preparations follow USP General Chapter <797>; non-sterile sublingual troche and oral preparations follow USP General Chapter <795>. Active pharmaceutical ingredient is sourced from FDA-registered API suppliers with documented certificates of analysis, and ingredient suitability for the intended preparation pathway is verified before use.

Compounded levocarnitine is dispensed only when the prescribing physician documents a patient-specific clinical need that the manufactured Carnitor oral solution, oral tablet, or intravenous injection cannot meet [fda_label_carnitor]. Common documented reasons include: a required IV concentration or volume not available in the manufactured 200 mg/mL injection; severe oral-route gastrointestinal intolerance to the oral solution or tablet; the need for an IM route in patients without practical IV access; or a clinically appropriate multi-ingredient blend that no FDA-approved single-ingredient product matches [fda503a].

Pharmacist review

Each prescription for compounded levocarnitine undergoes pharmacist review prior to dispensing. The review confirms a documented patient-specific clinical reason that manufactured Carnitor is not appropriate; absence of contraindications and significant drug interactions (including review of concurrent valproate, anticoagulants, and antiretroviral agents); seizure-history flag where present; appropriate route, concentration, and beyond-use date assignment; and a prescribed regimen consistent with the labeled indication or with a documented evidence-based off-label protocol [fda503a].

RonanRx does not fill prescriptions that read as routine substitution of compounded for manufactured Carnitor without documented clinical rationale, consistent with FDA guidance on compounded copies of commercially available drugs [fda_label_carnitor].

Quality and traceability

Active pharmaceutical ingredient is sourced from FDA-registered facilities with documented certificates of analysis. Each batch of compounded levocarnitine carries a lot number traceable to API source, compounding date, beyond-use date, sterility and endotoxin test result (for sterile preparations), and dispensing pharmacist of record. Finished product lot records are retained per state board of pharmacy retention requirements.

Cold chain

Manufactured Carnitor does not require cold-chain handling; controlled room temperature storage is standard. Most compounded levocarnitine preparations follow the same controlled room-temperature

convention; refrigerated handling is used only when stability data for a specific compounded preparation requires it. Patients receive preparation-specific storage instructions on the dispensing label.

Frequently Asked Questions About L-Carnitine

Is L-carnitine FDA-approved?

Yes, levocarnitine (Carnitor) is FDA-approved as an oral solution, oral tablet, and intravenous injection for primary systemic carnitine deficiency, secondary carnitine deficiency from inborn errors of metabolism, and the prevention and treatment of carnitine deficiency in end-stage renal disease patients on hemodialysis [fda_label_carnitor]. Numerous OTC L-carnitine and acetyl-L-carnitine dietary supplements are also sold under DSHEA; OTC supplements are not the same regulated product as prescription Carnitor [dailymed_levocarnitine].

Why is there compounded levocarnitine if Carnitor is FDA-approved?

Compounded 503A levocarnitine is dispensed only when the prescribing physician documents a patient-specific clinical need that the manufactured Carnitor product cannot meet, for example, a custom IV concentration for an infusion protocol, an IM route where IV access is impractical, a sublingual troche for severe oral intolerance, or a multi-ingredient blend with B-vitamins or amino acids where the clinician documents a clinical rationale [fda_label_carnitor]. It is not a generic substitute for Carnitor or for OTC supplements [fda503a].

What is the difference between L-carnitine, acetyl-L-carnitine, and propionyl-L-carnitine?

L-carnitine is the parent molecule and the form approved as Carnitor. Acetyl-L-carnitine (ALC) is the acetyl ester, crosses the blood-brain barrier more efficiently, and has been studied in Alzheimer disease, diabetic neuropathy, and fatigue [pettegrew1995; sima2005]. Propionyl-L-carnitine (PLC) is the propionyl ester, is preferentially taken up by muscle, and has been studied in intermittent claudication and heart failure [brevetti1999; mancini1992]. ALC and PLC are sold OTC and as 503A compounded preparations; they are not FDA-approved drugs in the United States.

What did the CEDIM trial show about carnitine after a heart attack?

CEDIM randomized 472 adults to oral L-carnitine 9 g/day for 5 days then 6 g/day for 12 months versus placebo within 24 hours of an acute anterior myocardial infarction [iliceto1995]. The trial reported attenuated progressive left-ventricular dilation on serial echocardiography at 6 and 12 months. A 2013 meta-analysis of 13 controlled trials and 3,629 participants reported a 27% reduction in all-cause mortality across L-carnitine trials after acute MI [dinicolantonio2013].

Does L-carnitine cause heart disease through TMAO?

The Koeth 2013 study in Nature Medicine reported that gut-microbiota metabolism of dietary L-carnitine generates trimethylamine-N-oxide (TMAO), which accelerated atherosclerosis in apoE-/- mice and correlated with cardiovascular events in observational human cohorts [koeth2013; wang2011; dinicolantonio2013]. The clinical implications for prescription therapeutic levocarnitine at typical doses are not established, and the largest cardiac meta-analysis to date reported net mortality benefit after acute MI despite the mechanistic concern. The TMAO hypothesis is an active research question rather than an established clinical contraindication.

Is L-carnitine useful for energy or weight loss in healthy adults?

Evidence for performance, energy, or weight-loss benefit in healthy non-deficient adults is inconsistent. Wall (2011) demonstrated that 24 weeks of oral L-carnitine 2 g/day plus carbohydrate raises skeletal-muscle total carnitine by approximately 21% and alters exercise fuel use, but Brass (2000) and subsequent reviews concluded that clinical performance benefits in non-deficient adults are not consistent [wall2011; brass2000]. Compounded 503A levocarnitine is not dispensed for generic 'energy' or 'weight loss' indications without a documented patient-specific clinical need.

What are the most common side effects?

Gastrointestinal symptoms (nausea, vomiting, abdominal cramping, diarrhea) are the most common, particularly with higher oral doses or oral solution without food [fda_label_carnitor]. A transient fishy body odor from gut-microbiota metabolism of unabsorbed carnitine is reported in some patients [eknoyan2003]. The labeled warnings include increased seizure frequency or new seizures in patients with prior seizure history. Intravenous administration is associated with rare hypotension, particularly in hemodialysis patients receiving rapid infusion.

Does RonanRx sell L-carnitine directly to patients?

No. Compounded 503A levocarnitine requires a patient-specific prescription written by a licensed physician for an identified patient and a pharmacist review before dispensing. RonanRx is not a direct-to-consumer storefront. OTC L-carnitine and acetyl-L-carnitine supplements are widely available through dietary-supplement retail channels; those products are regulated under DSHEA and are not the same as prescription Carnitor or compounded preparations [fda503a; fda_label_carnitor].

References

1. *[bremer1990]* Bremer J. *The role of carnitine in intracellular metabolism*. Journal of Clinical Chemistry and Clinical Biochemistry. 1990. PMID 2199593. (accessed 2026-05-11)
2. *[vaz2002]* Vaz FM, Wanders RJ. *Carnitine biosynthesis in mammals*. Biochemical Journal. 2002. PMID 11802770. (accessed 2026-05-11)

3. *[rebouche2004]* Rebouche CJ. *Kinetics, pharmacokinetics, and regulation of L-carnitine and acetyl-L-carnitine metabolism*. Annals of the New York Academy of Sciences. 2004. PMID 15591001. (accessed 2026-05-11)
4. *[marcovina2013]* Marcovina SM, Sirtori C, Peracino A, Gheorghiade M, Borum P, Remuzzi G, Ardehali H. *Translating the basic knowledge of mitochondrial functions to metabolic therapy: role of L-carnitine*. Translational Research. 2013. PMID 23138103. (accessed 2026-05-11)
5. *[magoulas2012]* Magoulas PL, El-Hattab AW. *Systemic primary carnitine deficiency: an overview of clinical manifestations, diagnosis, and management*. Orphanet Journal of Rare Diseases. 2012. PMID 22989098. (accessed 2026-05-11)
6. *[longo2017]* Frigeni M, Balakrishnan B, Yin X, Calderon FRO, Mao R, Pasquali M, Longo N. *Functional and molecular studies in primary carnitine deficiency*. Human Mutation. 2017. PMID 28841266. (accessed 2026-05-11)
7. *[longo2019]* Almannai M, Alfadhel M, El-Hattab AW. *Carnitine Inborn Errors of Metabolism*. Molecules. 2019. PMID 31500110. (accessed 2026-05-11)
8. *[eknoyan2003]* Eknoyan G, Latos DL, Lindberg J; National Kidney Foundation Carnitine Consensus Conference. *Practice recommendations for the use of L-carnitine in dialysis-related carnitine disorder. National Kidney Foundation Carnitine Consensus Conference*. American Journal of Kidney Diseases. 2003. PMID 12666074. (accessed 2026-05-11)
9. *[lheureux2005]* Lheureux PE, Penaloza A, Zahir S, Gris M. *Science review: carnitine in the treatment of valproic acid-induced toxicity - what is the evidence?*. Critical Care. 2005. PMID 16277730. (accessed 2026-05-11)
10. *[lheureux2009]* Lheureux PE, Hantson P. *Carnitine in the treatment of valproic acid-induced toxicity*. Clinical Toxicology (Phila). 2009. PMID 19280426. (accessed 2026-05-11)
11. *[famularo1997]* Famularo G, Moretti S, Marcellini S, Trinchieri V, Tzantzoglou S, Santini G, Longo A, De Simone C. *Acetyl-carnitine deficiency in AIDS patients with neurotoxicity on treatment with antiretroviral nucleoside analogues*. AIDS. 1997. PMID 9030365. (accessed 2026-05-11)
12. *[iliceto1995]* Iliceto S, Scrutinio D, Bruzzi P, D'Ambrosio G, Boni L, Di Biase M, Biasco G, Hugenholtz PG, Rizzon P. *Effects of L-carnitine administration on left ventricular remodeling after acute anterior myocardial infarction: the L-Carnitine Ecocardiografia Digitalizzata Infarto Miocardico (CEDIM) Trial*. Journal of the American College of Cardiology. 1995. PMID 7608438. (accessed 2026-05-11)
13. *[dinicolantonio2013]* DiNicolantonio JJ, Lavie CJ, Fares H, Menezes AR, O'Keefe JH. *L-carnitine in the secondary prevention of cardiovascular disease: systematic review and meta-analysis*. Mayo Clinic Proceedings. 2013. PMID 23597877. (accessed 2026-05-11)
14. *[mancini1992]* Mancini M, Rengo F, Lingetti M, Sorrentino GP, Nolfe G. *Controlled study on the therapeutic efficacy of propionyl-L-carnitine in patients with congestive heart failure*. Arzneimittelforschung. 1992. PMID 1445476. (accessed 2026-05-11)
15. *[rizos2000]* Rizos I. *Three-year survival of patients with heart failure caused by dilated cardiomyopathy and L-carnitine administration*. American Heart Journal. 2000. PMID 10650325. (accessed 2026-05-11)
16. *[brevetti1995]* Brevetti G, Perna S, Sabba C, Martone VD, Condorelli M. *Propionyl-L-carnitine in intermittent claudication: double-blind, placebo-controlled, dose titration, multicenter study*. Journal of the American College of Cardiology. 1995. PMID 7594063. (accessed 2026-05-11)
17. *[brevetti1997]* Brevetti G, Perna S, Sabba C, Martone VD, Di Iorio A, Barletta G. *Effect of propionyl-L-carnitine on quality of life in intermittent claudication*. American Journal of Cardiology. 1997. PMID 9070558. (accessed 2026-05-11)
18. *[brevetti1999]* Brevetti G, Diehm C, Lambert D. *European multicenter study on propionyl-L-carnitine in intermittent claudication*. Journal of the American College of Cardiology. 1999. PMID 10551714. (accessed 2026-05-11)

19. *[hiatt2001]* Hiatt WR, Regensteiner JG, Creager MA, Hirsch AT, Cooke JP, Olin JW, Gorbunov GN, Isner J, Lukjanov YV, Tsitsiashvili MS, Zabelskaya TF, Amato A. *Propionyl-L-carnitine improves exercise performance and functional status in patients with claudication*. American Journal of Medicine. 2001. PMID 11382369. (accessed 2026-05-11)
20. *[hiatt2011]* Hiatt WR, Creager MA, Amato A, Brass EP. *Effect of propionyl-L-carnitine on a background of monitored exercise in patients with claudication secondary to peripheral artery disease*. Journal of Cardiopulmonary Rehabilitation and Prevention. 2011. PMID 20861750. (accessed 2026-05-11)
21. *[delaney2013]* Delaney CL, Spark JI, Thomas J, Wong YT, Chan LT, Miller MD. *A systematic review and meta-analysis of propionyl-L-carnitine effects on exercise performance in patients with claudication*. Vascular Medicine. 2013. PMID 23321261. (accessed 2026-05-11)
22. *[lenzi2003]* Lenzi A, Lombardo F, Sgrò P, Salacone P, Caponecchia L, Dondero F, Gandini L. *Use of carnitine therapy in selected cases of male factor infertility: a double-blind crossover trial*. Fertility and Sterility. 2003. PMID 12568837. (accessed 2026-05-11)
23. *[cavallini2005]* Cavallini G, Modenini F, Vitali G, Koverech A. *Acetyl-L-carnitine plus propionyl-L-carnitine improve efficacy of sildenafil in treatment of erectile dysfunction after bilateral nerve-sparing radical retropubic prostatectomy*. Urology. 2005. PMID 16286128. (accessed 2026-05-11)
24. *[pettegrew1995]* Pettegrew JW, Klunk WE, Panchalingam K, Kanfer JN, McClure RJ. *Clinical and neurochemical effects of acetyl-L-carnitine in Alzheimer's disease*. Neurobiology of Aging. 1995. PMID 7723928. (accessed 2026-05-11)
25. *[spagnoli1991]* Spagnoli A, Lucca U, Menasce G, Bandera L, Cizza G, Forloni G, Tettamanti M, Frattura L, Tiraboschi P, Comelli M, et al. *Long-term acetyl-L-carnitine treatment in Alzheimer's disease*. Neurology. 1991. PMID 1944900. (accessed 2026-05-11)
26. *[salvioli1994]* Salvioli G, Neri M. *L-acetylcarnitine treatment of mental decline in the elderly*. Drugs Under Experimental and Clinical Research. 1994. PMID 7813389. (accessed 2026-05-11)
27. *[hudson2003]* Hudson S, Tabet N. *Acetyl-L-carnitine for dementia*. Cochrane Database of Systematic Reviews. 2003. PMID 12804452. (accessed 2026-05-11)
28. *[sima2005]* Sima AAF, Calvani M, Mehra M, Amato A; Acetyl-L-Carnitine Study Group. *Acetyl-L-carnitine improves pain, nerve regeneration, and vibratory perception in patients with chronic diabetic neuropathy: an analysis of two randomized placebo-controlled trials*. Diabetes Care. 2005. PMID 15616239. (accessed 2026-05-11)
29. *[malaguarnera2007]* Malaguarnera M, Cammalleri L, Gargante MP, Vacante M, Colonna V, Motta M. *L-Carnitine treatment reduces severity of physical and mental fatigue and increases cognitive functions in centenarians: a randomized and controlled clinical trial*. American Journal of Clinical Nutrition. 2007. PMID 18065594. (accessed 2026-05-11)
30. *[malaguarnera2008]* Malaguarnera M, Gargante MP, Cristaldi E, Colonna V, Messano M, Koverech A, Neri S, Vacante M, Cammalleri L, Motta M. *Acetyl L-carnitine (ALC) treatment in elderly patients with fatigue*. Archives of Gerontology and Geriatrics. 2008. PMID 17658628. (accessed 2026-05-11)
31. *[malaguarnera2013]* Malaguarnera M. *Acetyl-L-carnitine in hepatic encephalopathy*. Metabolic Brain Disease. 2013. PMID 23389620. (accessed 2026-05-11)
32. *[tomassini2004]* Tomassini V, Pozzilli C, Onesti E, Pasqualetti P, Marinelli F, Pisani A, Fieschi C. *Comparison of the effects of acetyl L-carnitine and amantadine for the treatment of fatigue in multiple sclerosis: results of a pilot, randomised, double-blind, crossover trial*. Journal of the Neurological Sciences. 2004. PMID 14759641. (accessed 2026-05-11)

33. *[wall2011]* Wall BT, Stephens FB, Constantin-Teodosiu D, Marimuthu K, Macdonald IA, Greenhaff PL. *Chronic oral ingestion of L-carnitine and carbohydrate increases muscle carnitine content and alters muscle fuel metabolism during exercise in humans*. Journal of Physiology. 2011. PMID 21224234. (accessed 2026-05-11)

34. *[brass2000]* Brass EP. *Supplemental carnitine and exercise*. American Journal of Clinical Nutrition. 2000. PMID 10919968. (accessed 2026-05-11)

35. *[wang2011]* Wang Z, Klipfell E, Bennett BJ, Koeth R, Levison BS, Dugar B, Feldstein AE, Britt EB, Fu X, Chung YM, Wu Y, Schauer P, Smith JD, Allayee H, Tang WHW, DiDonato JA, Lusis AJ, Hazen SL. *Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease*. Nature. 2011. PMID 21475195. (accessed 2026-05-11)

36. *[koeth2013]* Koeth RA, Wang Z, Levison BS, Buffa JA, Org E, Sheehy BT, Britt EB, Fu X, Wu Y, Li L, Smith JD, DiDonato JA, Chen J, Li H, Wu GD, Lewis JD, Warrier M, Brown JM, Krauss RM, Tang WHW, Bushman FD, Lusis AJ, Hazen SL. *Intestinal microbiota metabolism of L-carnitine, a nutrient in red meat, promotes atherosclerosis*. Nature Medicine. 2013. PMID 23563705. (accessed 2026-05-11)

37. *[fda_label_carnitor]* U.S. Food and Drug Administration. *Carnitor (levocarnitine) — FDA Drugs@FDA application page (NDA 019257) for oral solution, tablets, and injection*. Drugs@FDA. 2024. https://www.accessdata.fda.gov/scripts/cder/daf/index.cfm?event=overview.process&ApplNo=019257 (accessed 2026-05-11)

38. *[dailymed_levocarnitine]* U.S. National Library of Medicine DailyMed. *Levocarnitine (Carnitor) — DailyMed product labeling*. DailyMed. 2024. https://dailymed.nlm.nih.gov/dailymed/search.cfm?query=levocarnitine&pagesize=20 (accessed 2026-05-11)

39. *[fda503a]* U.S. Food and Drug Administration. *Section 503A of the Federal Food, Drug, and Cosmetic Act*. https://www.fda.gov/drugs/human-drug-compounding/section-503a-federal-food-drug-and-cosmetic-act (accessed 2026-05-11)

How to Access L-Carnitine

Compounded L-Carnitine is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed L-Carnitine, sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)