

l-Carnitine (LC), acetyl-l-carnitine (ALC), propionyl-l-carnitine (PLC)

Carnitine (LC) is a quaternary ammonium compound biosynthesized from the amino acids lysine and methionine. Vitamin C is essential to the synthesis of LC. Carnitine is synthesized in the liver, and in the brain and kidney, from lysine and methionine, with catalysts vitamins B6, B3, C, and folate. In our natural diet, carnitine is found in meat and in milk products.

Betaine (trimethylglycine) is another quaternary ammonium compound that is biologically active as an osmolite, stabilizing osmotic pressure in cells.

- Carnitine is a betaine derivative (this is one more example of the amazing biological activity of betaine, another ingredient in your Diphasic A.M.) of beta hydroxy butyrate.
- Betaine has another protective function – it protects the brain against the toxic effects of ammonia produced in the liver (but it does not do this as well as carnitine).
- Carnitine increases the conversion of ammonia to urea.
- To protect the brain from excess ammonia, we use carnitine, taurine, citrulline, and betaine.

The primary function of LC is transporting fatty acids from the cytosol into the mitochondria during the breakdown of lipids for the generation of metabolic energy. In the mitochondrial matrix, fatty acids are broken down through beta oxidation to acetyl-CoA for entry into the citric acid cycle. Fatty acids must first be activated before binding to the LC molecule to form acyl-carnitine.

- Carnitine increases liver co-enzyme A (Co-A-SH).
- Carnitine stabilizes the Co-A-SH/Acetyl-CoA ratio.
- Mitochondria are impermeable to fatty acyl-CoA, but not to fatty acyl-carnitine --- LC is essential for translocation of long-chain fatty acids into skeletal muscle mitochondria for beta oxidation. Beta oxidation, in turn, feeds acetyl-CoA into the TCA.
- Carnitine will increase both alpha-hydroxybutyrate and beta-hydroxybutyrate
- Carnitine is essential for the transport of long chained fatty acids into the mitochondria: Thus Carnitine depletion causes intracellular lipid accumulation.

Maebashi, N. et al, "Lipid-lowering Effect Of Carnitine in Patients with Type IV Hyperlipoproteinaemia." Lancet 2:805-807, 1978.

- Carnitine improves fat metabolism in the heart (and other organs and tissues).
- Carnitine reduces triglycerides and cholesterol; it significantly increases high-density lipo-proteins.
- Carnitine increases the oxidation of fatty acids, and thereby increases ketone formation (this is one of the facts seized upon by the unscrupulous health food industry as "proof" that carnitine will "burn fat").
- Carnitine is a buffer for acetyl groups, which are present in excess in some tissues during ketosis and as a result of hypoxic muscle activity.

Orally ingested LC is poorly absorbed and poorly assimilated by muscle cells. Combining LC intake with enough carbohydrate to increase serum insulin results in greater whole body retention of LC.

Oral dose of 2 grams LC at one time is the maximum absorbed.

Choline conserves urinary carnitine.

LC supplementation reduces severity of physical and mental fatigue and increases cognitive functions in geriatric subjects.

LC increases glucose oxidation in Type II diabetics. LC increases glucose storage in both Type II diabetics and controls. In Type II diabetics, plasma lactate significantly decreases during LC infusion.

- Carnitine reduces lactic acid production.
- Carnitine appears to improve insulin resistance. It decreases the spike in glucose concentration after glucose administration, and decreases the associated insulin secretion.

LC is a substantial antioxidant, protecting against lipid peroxidation of phospholipid membranes, and against oxidative stress induced at the myocardial and endothelial cell level.

Carnitine improves heart muscle exercise tolerance.

Carnitine prevents cardiac arrhythmias, including the occurrence of ventricular fibrillation in the early phases of ischemia.

Carnitine prevents angina.

Carnitine is a vaso-dilator of coronary blood vessels (and lowers blood pressure).

Carnitine (because of its role in supplying fatty acids to the heart muscle) is valuable in the prevention of chronic heart failure.

Fritz, I. et al, "Specificity of Carnitine Actions of Fatty Acid Oxidation by Heart Muscle." Am. J. Physiol. 202:117-121, 1962.

Thomsen, et al: "Improved Stress Tolerance of the Ischemic Human Myocardium After Carnitine Administration." J. Amer. Cardiology 39:289, 1977.

There are conflicting reports in the literature as to whether carnitine improves athletic performance. Some studies say yes, some studies say that it does not improve exercise performance in normals, but does definitely increase exercise performance in cardiac patients.

Regarding its role as a protector against cardiovascular disease, it has been shown that carnitine:

- protects myocardial infarct patients against cardiac necrosis.
- improves fat metabolism in the heart (as well as other organs)
- decreases lipid peroxides in the heart.
- improves heart muscle exercise tolerance.
- decreases angina pain
- is a vasodilator of coronary blood vessels, and lowers blood pressure.
- decreases the elevated LDH levels in myocardial infarct patients.
- decreases left ventricle enlargement.
- decreases the incidence of arrhythmias, including the occurrence of ventricular fibrillation in the early stages of ischemia.
- decreases peripheral vascular disease.
- decreases congestive heart failure.

- has a dramatic impact on decreasing triglycerides. It decreases elevated cholesterol as well, but has a far greater effect on triglycerides. Carnitine also increases high density lipoproteins.
- The corollary to the preceding statement is that studies have shown that a deficiency of carnitine is a causative factor in increasing triglycerides, both in the liver and in the serum.

The literature shows a link between mitral valve prolapse and both carnitine and magnesium deficiencies. Carnitine and magnesium need is most pronounced in Anaerobic patients.

Carnitine supplementation of 3 grams daily is specifically beneficial for Grave's, especially in patients who test Anaerobic.

- When the particular thyroid autoimmune reaction involves hyperthyroid function, carnitine supplementation is beneficial, partly because it influences the fatty acids in the mitochondrial membranes. Carnitine works as a peripheral antagonist of thyroid hormone action, but not an inhibitor of thyroid gland function, by decreasing T3 and T4 entry into the nucleus of cells (including neurons, hepatocytes, and fibroblasts, and likely most other cell types as well). Carnitine does not antagonize the physiological negative feedback of thyroid hormones on TSH secretion.
- Not only does carnitine control the mechanism of hyperthyroidism, but hyperthyroidism depletes the body deposits of carnitine. Thus, for these 2 reasons, carnitine is essential supplementation for all hyperthyroid patients. There are many symptoms and conditions that correlate directly with the level of Free T4 in hyperthyroid patients, and all these symptoms and conditions are improved dramatically by carnitine supplementation. They include: asthenia, dyspnea (increased respiratory rate), nervousness, palpitations, hand tremor, elevated heart rate, and exaggerated patellar reflex.
- Carnitine also reverses the abnormal labs associated with hyperthyroid, including: decreasing elevated liver enzymes ALT, AST, and GGT, decreasing elevated serum ferritin, decreasing elevated sex hormone binding globulin, and increasing depressed creatine phosphokinase. (Carnitine has no effect on the hyperthyroid abnormal labs that include depressed TSH, low cholesterol, and elevated urine hydroxyproline.)
- Muscle weakness is characteristic of both hyper- and hypothyroidism. In both hyper- and hypothyroidism, the carnitine content of muscles is low, and is restored to normal after normalization of thyroid function.

- If any autoimmune thyroid patient, including a hypothyroid Hashimoto's autoimmune thyroiditis, makes a sudden shift into hyperthyroid function, with elevated respiratory rate and heart rate, you will need to supplement with carnitine, 1000 mg 3 times daily.

MS patients with fatigue respond well to supplementation with carnitine.

- Carnitine supplementation (3-6 g daily)= ↓ fatigue in MS patients on immunosuppression therapy

I suspect that glutathione is actually neutral as regards Anaerobic/Dysaerobic anabolic/catabolic balance. Of the 3 amino acids involved, one is anti-Anaerobic and one is anti-Dysaerobic. I have used glutathione quite heavily in Anaerobic patients for many years only because it is the one "antioxidant" that does not have a specific anti-Dysaerobic effect. (Others include carnitine and acetyl-L-carnitine, and probably propionyl-L-carnitine, PQQ, carnosine, and lipoic acid under certain circumstances, and maybe anatabine (along with selenium, of course).

- Carnitine (2g/ os daily) = ↓ liver enzymes, ↓ [CHOL], & ↓ [TG] in chronic hepatitis C, & ↓ fatigue
- Carnitine has been shown to increase the survival of hypoxic mice.
- Carnitine protects membrane structures.
- Carnitine has been shown to decrease drug-induced seizures in mice, while preventing the increase in lactic acid and the decreased ATP and decreased phosphocreatine typical of seizures.

Carnitine has been shown to decrease lipid-induced immune suppression.

- Carnitine increases lymphocyte proliferation following mitogenic stimulation, and increases white blood cell motility.
- Continuing with studies that show carnitine's benefit on the immune system: there have been many studies showing that carnitine decreases inflammatory cytokines, decreases interleukins, and decreases tumor necrosis factor in mice with induced sarcoma. In particular, carnitine supplementation of 1,000 mg daily for 12 weeks decreased IL-6 by 61%, and consequently, reduced CRP by 29%. Carnitine supplementation also slightly reduces TNF- α . Carnitine supplementation has no effect on IL-10.

- Carnitine deficiency results in the hyper-activation of CD4+ T cells and enhanced cytokine production. Specifically as relates to intestinal inflammatory diseases, carnitine decreases IL-1 β and IL-6. There may be an association between Crohn's disease and mutations in the L-carnitine transporter genes.
- IL-6 ---Chronic Fatigue Syndrome = need alpha linolenic acid, Mg, carnitine
- Carnitine has similar immunomodulatory effects as glucocorticoids. Carnitine suppresses TNF-alpha and Interleukin-12 release from human monocytes, while at the same time it activates glucocorticoid receptors. Furthermore, this suppression of excess inflammatory cytokines is blocked by administering a glucocorticoid inhibitor --- showing that at least part of carnitine's immunomodulatory benefits are by potentiating the anti-inflammatory effects of glucocorticoids.

Interestingly, carnitine also antagonizes excessively activated glucocorticoid receptors. Carnitine actually increases bone density (while excess glucocorticoid and excess thyroid decrease bone mineralization).

Carnitine was shown to be beneficial in reducing chronic fatigue.

Carnitine supplementation (along with vitamin B12) was shown to decrease anorexia.

In rats, carnitine decreases the age-associated decline in mitochondrial function and general metabolic activity.

Biotin-deficient rats are a good animal model for human multiple carboxylase deficiency, and have low plasma free LC levels.

Carnitine causes hypertrophy of Type I muscle fibers; it increases exercise tolerance.

- LC has a role entirely separate from its transport of long-chain fatty acids from the cytosol into the mitochondria. LC functions as an acetyl group buffer at the onset of exercise, when the rate of acetyl-CoA generation is greater than its utilization in the TCA cycle. The buffering of excess acetyl-CoA by LC under demand of exercise significantly reduces free LC.

For example, following a few minutes of high intensity exercise, skeletal muscle free LC content is reduced from 75% of the total muscle LC pool at rest to around 20%, with almost all the reduction being attributed to the formation of ALC. This increase in ALC formation during high intensity

exercise occurs to a greater extent in Type I muscle fibers. It is directly related to the increase in muscle acetyl-CoA, suggesting that the rate of acetyl-CoA formation from pyruvate oxidation exceeds its utilization by the TCA cycle, leading to excess accumulation of acetyl-CoA.

Thus, during high intensity exercise (and other circumstances of increased pyruvate decarboxylase (PDC) flux) LC buffers the excess acetyl groups formed, ensuring a viable pool of free Co-A-SH to be maintained for the continuation of the PDC and TCA cycle reactions. If the activity of the PDC were maximal at high exercise intensity, and its rate of pyruvate oxidation were supported solely by the Co-A-SH available in the muscle at rest, then the entire pool of muscle Co-A-SH would become acetylated within one second of the initiation of the muscle contraction, hypothetically resulting in the immediate and complete inhibition of the PDC reaction and of the TCA cycle.

- In addition to maintaining a viable pool of Co-A-SH, the accumulation of ALC itself provides a store of acetyl groups, which is readily available for transacetylation back to its acetyl-CoA for utilization by the TCA cycle. So, in the latter half of 4 hours of exercise at 55% VO₂max, muscle ALC content returns to near resting values, which is paralleled by a decrease in PDC activity and therefore acetyl group delivery from pyruvate.
- Increasing muscle total LC content alleviates the decline in fat oxidation rates routinely observed during high intensity exercise, and concomitantly reduces muscle glycogen utilization. Trained human and equine athletes have a higher capacity to utilize fatty acids during exercise, and appear to have a higher total muscle LC content compared to untrained controls. Increasing skeletal muscle LC content delays fatigue development by 25% during electrical stimulation in rat muscle.
 - Central nervous system and muscle involvement in an adolescent patient with riboflavin-responsive multiple acyl-CoA dehydrogenase deficiency (MADD) ----- reported as an adolescent case of late-onset riboflavin-responsive multiple acyl-CoA dehydrogenase deficiency (MADD) characterized by intermittent nausea and depressive state as early symptoms.

Progressive muscle weakness occurs and blood creatine kinase level is found to be elevated.

Branched-chain organic acidurias ----- branched-chain organic acidurias are a group of disorders that result from an abnormality of specific enzymes involving the catabolism of BCAA (leucine, isoleucine, valine). All these disorders present in neonates as a neurologic distress of the intoxication type, with either ketosis or ketoacidosis and hyperammonemia. Most of these

disorders present with severe dehydration, leuconeutropenia, and thrombopenia. All these disorders can be diagnosed by identifying acylcarnitine and other organic acid compounds in plasma and urine. These disorders are amenable to treatment by removing toxic compounds and by using special diets and carnitine.

With LC supplementation, Anemia of Chronic Disease has improved

Suberate = high = need carnitine, B2, choline, CoQ10.

Be careful with carnitine since excess may decrease reabsorption of amino acids in the proximal tubule of the kidneys.

- Carnitine is derived from lysine. Carnitine may improve growth by sparing lysine and enhancing FA oxidation = also protects from ammonia.

Sweaty feet odor, fatigue, recurrent infections, ADD = signs of LC need.

- NUTRI-SPEC practitioners have been benefiting for years from the true value of carnitine. As part of your Activator, carnitine has given a metabolic boost to every single one of your nutrition patients. Carnitine is also a major component of your Formula ES because of its beneficial effects not only on myocardial energy production, but also because it is one of the most effective ways to lower elevated triglycerides (one of the few primary risk factors for heart attacks and strokes).

Carnitine is also a critical component of the Oxygenic A that you give to all your patients with an Anaerobic Metabolic Imbalance. Why? Your Anaerobic patients show a deficiency of oxidative metabolism reflected in their over-dependence on anaerobic glycolysis. In particular (as reflected by their high urinary surface tension and low oxidation index), these Anaerobic patients are deficient in the ability to oxidize fatty acids. In reversing the deficient oxidation of fatty acids, carnitine improves cellular energy production, with the resultant benefit of decreasing the excess lactic acid build up typical of Anaerobic patients (while, of course, it lowers the surface tension and improves the oxidation index).

Carnitine is also a critical constituent of your Oxygenic G that you give all your glucogenic patients. A glucogenic imbalance is typified by a desperate need for more energy production via the beta hydroxy butyric acid metabolic pathway. In facilitating the oxidation of fatty acids, carnitine feeds this need of your glucogenic patients perfectly.

Carnitine contributes in a major way to the power of your Diphasic Nutrition Plan. It is found in your Go Power, or Diphasic A.M. supplement

that will protect all your patients against pathological hyperplasia when taken in the morning (in harmony with the anti-anabolic phase of your patients' diphasic metabolic cycle).

- ALC and PLC are two naturally occurring LC derivatives formed by carnitine acetyltransferase. There are beneficial effects of ALC and PLC, including management of peripheral arterial disease, heart and cerebral ischemia, and congestive heart failure, as well as Type II diabetes (an independent risk factor for CVD). ALC is particularly known to have neuroprotective benefits.

- Acetyl-L-Carnitine (ALC) is an acetylated form of LC. During strenuous exercise, a large portion of LC and unused acetyl-CoA are converted to ALC and CoA inside mitochondria. The ALC is transported outside the mitochondria where it converts back to LC and acetyl-CoA. The LC is cycled back into the mitochondria with acyl groups to facilitate fatty acid utilization.
 - o Excess acetyl-CoA will block fatty acid utilization. Excess acetyl-CoA causes more carbohydrate to be used for energy at the expense of fatty acids. This shift toward carbohydrate and away from fatty acid metabolism occurs by different mechanisms inside and outside the mitochondria. ALC transport decreases acetyl-CoA inside the mitochondria, but increases it outside.
- A portion of LC is converted to ALC after ingestion in humans.
- ALC is superior to LC in terms of bioavailability. ALC has lower blood concentration than LC after ingestion, but that is possibly because ALC is hydrolyzed more in blood, or that it is entering cells (brain or muscle) more efficiently from the blood than LC. (LC is not absorbed into cells unless there is an insulin spike such as from a carbohydrate load.)
- Glucose metabolism increases with administration of either ALC or LC.
- ALC improves insulin resistance.
- ALC inhibits TNF- α -induced insulin resistance.
- ALC has the ability to cross the BBB where it acts as a powerful antioxidant. (It may have a neuroprotective effect in Parkinson's.)
 - Cerebral ATP depleted by hyperammonemia and hyperglutaminemia is restored by acetyl-L-carnitine.
 - ALC corrects the brain's deficiency of the cholinergic system associated with hyperammonemia.
- ALC may be beneficial in dementia, particularly dementia affecting the cholinergic neurotransmitter system. ALC has activity on cholinergic neurons, and affects membrane stabilization and enhances mitochondrial function.
 - Evidence for a central cholinergic deficit in congenital ornithine transcarbamylase (OTC) deficiency --- muscarinic cholinergic binding site distribution shows a wide-spread loss of M1 sites, consistent with

cholinergic cell loss. These alterations are similar to those reported in Alzheimer's disease, suggesting that the severe cognitive dysfunction in congenital OTC deficiency may at least partly result from a muscarinic cholinergic lesion. Treatment with acetyl-L-carnitine results in partial recovery of the developmental choline acetyltransferase deficit.

- Forget the brain memory products (pun intended). For your information --- ALC excess can be excitotoxic --- accelerating fatty acid oxidation in brain cells.
- Lipoic acid plus acetyl L-carnitine does an even better job of increasing glutathione than either alone.
- Lipoic acid and acetyl-L-carnitine are 2 mitochondrial antioxidants studied in a chronic rotenone-induced cellular model of Parkinson's Disease (PD). Both nutrients were found protective against mitochondrial dysfunction, oxidative damage, and accumulation of alpha-synuclein and ubiquitin. Most notably, it was found that combined LA and ALC worked at 100 to 1,000 times fold lower concentrations than they did individually. Pretreatment with combined LA and ALC increased mitochondrial biogenesis and decreased production of ROS through the upregulation of peroxisome proliferator-activated receptor-gamma coactivator 1 alpha as a possible underlying mechanism. This study provided important evidence that combining mitochondrial antioxidants at optimal doses might be an effective and safe prevention strategy for PD and other neuro-degenerative conditions.
- Agents that have shown to be beneficial in animal models of PD include CoQ10, creatine, Gingko biloba, nicotinamide, and acetyl-L-carnitine.
- Long-term administration of ALC to aged rats restores a synaptic pattern in the hippocampus comparable to that of young rats.
- LC acts as a carrier of fatty acids across the inner mitochondrial membrane for subsequent beta oxidation. ALC is the acetyl derivative of LC that possesses slight cholinergic-mimetic activity. Its success in sports medicine depends on its ability to stimulate CNS functions.
- Chronic Fatigue Syndrome (CFS) in the elderly: administering ALC reduced both physical and mental fatigue and improved the cognitive status as well as physical functions.
- Chronic fatigue in MS patients is better treated by 2 grams of ALC daily than with amantidine (the most widely used drug to treat MS-related fatigue).

- Fibromyalgia Syndrome (FMS): ALC decreases the total myalgic score compared to a placebo after 10 weeks. (The ALC group and the placebo group showed equal improvement for the first 6 weeks.)
- Fatty acids are the major oxidation fuel for the heart, while glucose and lactate provide the remaining need. Fatty acids in heart cytoplasm are transformed to long-chain acyl-CoA and transferred into the mitochondrial matrix by the action of 3 LC-dependent enzymes to produce acetyl-CoA through the beta-oxidation pathway. Another source of mitochondrial acetyl-CoA is from the oxidation of carbohydrates. The pyruvate dehydrogenase (PDH) complex, the key irreversible rate-limiting step in carbohydrate oxidation, is modulated by the intra-mitochondrial ratio acetyl-CoA/CoA. An increased ratio results in the inhibition of PDH activity. A decreased ratio can relieve the inhibition of PDH as shown by the transfer of acetyl groups from the acetyl-CoA to carnitine, forming ALC. This activity of LC and the modulation of the intra-mitochondrial acetyl-CoA/CoA ratio affects glucose oxidation.

Evidence suggests that LC exerts a protective effect in heart ischemia and hypertrophy. The actions of LC and PLC cannot be explained as exclusively dependent on the stimulation of fatty acid oxidation, but rather on a marked increase in glucose oxidation, via a relief of PDH inhibition caused by the elevated acetyl-CoA/CoA ratio. Enhanced pyruvate flux through PDH is beneficial for the cardiac cells since less pyruvate is converted to lactate thus preventing the acidification of the intracellular compartment. In addition, LC decreases tissue levels of acyl moieties, a mechanism particularly important in the ischemic phase.

- Propionyl-L-Carnitine (PLC) is naturally occurring derivative of LC that plays an important role in the metabolism of both carbohydrates and lipids, leading to an increase in ATP generation. PLC is also a potent anti-radical agent and thus may protect tissues from oxidative damage. PLC has a protective effect in cardiac and endothelial dysfunction, preventing the progression of atherosclerosis. PLC improves some of the cardio-metabolic alterations accompanying insulin resistance.
- LC and PLC reduce angina.
- PLC is effective in congestive heart failure in association with its effects on both cardiac muscle and skeletal muscle.
- PLC improves the contraction of isolated and aerobic perfused rabbit hearts. PLC improves energy metabolism and myocardial contractility in several different experimental models of congestive heart failure (CHF). The benefits of PLC are most apparent in situations of high energy demand such as those induced by increased work load. PLC may also benefit CHF patients by improving skeletal muscle metabolism by increasing pyruvate flux into the Krebs Cycle and by decreasing lactate production. In a randomized study of 50 patients with mild CHF, PLC increased the maximal exercise time, reduced lactate production, and improved left ventricular ejection fraction.
- PLC stimulates energy production in ischemic muscles by increasing citric acid cycle flux and by stimulation of pyruvate dehydrogenase activity. Its free radical scavenging activity may also be beneficial.
- In peripheral arterial disease patients, PLC improves coagulative fibrinolytic homeostasis in endothelium, and positively affects blood viscosity improvements in maximum walking distance. The improved walking distance correlates positively with increased mitochondrial oxidative ATP synthesis. Oral PLC, 1-3 grams daily, significantly improves mean maximum walking distance in arterial obstructive disease by 73% in patients with intermittent claudication.
- PLC, unlike LC and ALC, has anti-inflammatory activity in some models of vascular inflammation in rodents. In a study of rat paw edema induced by platelet activating factor, PLC effectively inhibited the edema while LC, ALC, as well as indomethacin and phenylbutazone were ineffective. The extent of inhibition produced by PLC was comparable to that of betamethasone or sodium salicylate.
- PLC has a specific protective role in the vascular component of the inflammatory process, while LC and ALC are ineffective.

- Ferrous ions induce an increase of MDA formation and a reduction of mitochondrial oxygen consuming and calcium transporting capillaries in the heart. LC and ALC fail to prevent mitochondrial damage. PLC significantly improves mitochondrial function, but fails to reduce MDA formation. This protective effect is specific for PLC, as LC and ALC do not modify mitochondrial damage.
- Recovery from heart ischemia is enhanced by LC, and even more with ALC. PLC was not effective in the first minutes of reperfusion, but during the whole reperfusion phase was beneficial. It is seen that LC and ALC and PLC have protective effects against intracellular PH decreases during ischemia and improve the energetic state of the heart, leading to increased ischemia tolerance.
- Patients with peripheral arterial disease were perfused with LC, ALC, and PLC. ALC and PLC produced significant increases in plasma levels of adenosine and ATP, while LC induced less relevant changes. The compounds did not affect the adenosine/inosine ratio. Peak plasma levels of adenosine preceded those of ATP. It is suggested that the pharmacological activity of PLC, ALC, and LC may be mediated in part by an interference with the endogenous purine system.
- ALC and PLC, but not LC, decrease the cytoplasmic calcium level in endothelial cells, protecting them from oxidative damage, from glucose/glucose oxidase, and from oxidized low-density lipoproteins.
- When cardiac function in isolated rat hearts was impaired by ischemia, subsequent perfusion with PLC increased their rate of recovery. Both PLC and to a lesser extent its taurine amide, and also ALC, significantly restored cardiac function in 15 minutes after 90 minutes of ischemia. LC was ineffective. PLC also increased tissue ATP and creatine phosphate. PLC and ALC reduced fatty acid peroxidation (as shown by decreased MDA), and were more effective than LC in preventing the production of superoxide.

In myocytes, PLC alone stimulated palmitate oxidation, but in rat heart tissue, both PLC and LC did so, while ALC was actually inhibitory. Possible mechanisms for the protective action of PLC against ischemia include an increased rate of cellular transport, stimulation of fatty acid oxidation, and a reduction of free radical formation.

- Sexual dysfunction in men following treatments for prostate cancer showed benefits from oral phosphodiesterase type 5 (PDE5) inhibitors. There is also evidence that PDE5 inhibitors are more effective in combination with ALC and PLC.

- Sexual dysfunction after bilateral prostatectomy was improved in almost all cases with PLC and ALC added to sildenafil, more than sildenafil alone.
- Testosterone replacement significantly increases the prostate volume and free and total testosterone levels, and significantly lowers serum LH, while ALC and PLC do not, but are more active than testosterone in improving male aging symptoms. (Two grams per day of each.)
- LC, ALC, and PLC all stimulate gastric acid secretion in rats. The addition of atropine to the perfusion only partially antagonized the effects. The mechanism of the gastric secretory stimulation was found to be the ALC and PLC partly by inhibition of acetylcholine esterase. But as the increase of gastric acid secretion was blocked only partially by atropine, while completely abolished by experimental blockade of post synaptic sympathetic receptors, it is suggested that the effect of carnitines in stimulating gastric secretion is determined by cholinergic and partly by adrenergic mechanisms.
- LC has a positive effect on bone mass. Administration of LC or PLC is capable of increasing serum osteocalcin that decreases with age, leading to osteoporosis.
- PLC has neuroprotective effects similar to ALC, and could be useful in the treatment of neurodegenerative diseases. Both ALC and PLC attenuate forebrain ischemia-induced neuronal damage, while increasing ATP and glutathione, as well as decreasing thiobarbituric acid-reactive substances (TBARS), and nitric oxide in hippocampal tissues.
- Chronic Fatigue Syndrome: 59% of patients improved on 2 grams of ALC; 63% improved on 2 grams of PLC; 37% improved on a combination of 2 grams ALC + 2 grams PLC. ALC significantly improved mental fatigue; PLC improved general fatigue. Attention concentration improved in all groups. Pain complaints did not decrease in any group. In the ALC group, but not in the other groups, the changes in plasma LC correlated with clinical improvement.
- The anti-inflammatory effects of LC, ALC, and PLC are associated not with the reduction of prostaglandins, thromboxane, and leukotriene B4, but with the increased production of prostacyclin (PGI₂). Thus, the ratios between PGI₂ and prostaglandins, thromboxane, and leukotrienes B4 tended to be higher, particularly in young animals fed LC, ALC, and PLC.
- LC, ALC, and PLC all decrease the number of peritoneal carrageenan-elicited macrophages.

- Urinary excretion of methylmalonic acid was increased 200-fold in vitamin B12 deficient rats. Urinary ALC excretion was increased in the vitamin B12 deficient animals by 70%. This increase in urinary ALC excretion correlates with the degree of metabolic impairment as measured by the urinary methylmalonic acid elimination. Urinary PLC excretion averaged 11 nmols/day in control rats and 120 nmols/day in the vitamin B12 deficient group. Thus, vitamin B12 deficiency is associated with the redistribution of carnitine toward ALC and PLC.