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Review

History of L-carnitine: Implications for renal disease *

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Abstract

L-Carnitine (LC) plays an essential metabolic role that consists in transferring the long chain fatty acids (LCFAs) through the mitochondrial barrier, thus allowing their energy-yielding oxidation. Other functions of LC are protection of membrane structures, stabilizing a physiologic coenzyme-A (CoA)-sulfate hydrate/acetyl-CoA ratio, and reduction of lactate production. On the other hand, numerous observations have stressed the carnitine ability of influencing, in several ways, the control mechanisms of the vital cell cycle. Much evidence suggests that apoptosis activated by palmitate or stearate addition to cultured cells is correlated with de novo ceramide synthesis. Investigations in vitro strongly support that LC is able to inhibit the death planned, most likely by preventing sphingomyelin breakdown and consequent ceramide synthesis; this effect seems to be specific for acidic sphingomyelinase. The reduction of ceramide generation and the increase in the serum levels of insulin-like growth factor (IGF)-1, could represent 2 important mechanisms underlying the observed antiapoptotic effects

could represent 2 important mechanisms underlying the observed antiapoptotic effects of acetyl-LC. Primary carnitine deficiency is an uncommon inherited disorder, related to functional anomalies in a specific organic cation/carnitine transporter (hOCTN2). These conditions have been classified as either systemic or myopathic. Secondary forms also are recognized. These are present in patients with renal tubular disorders, in which excretion of carnitine may be excessive, and in patients on hemodialysis. A lack of carnitine in hemodialysis patients is caused by insufficient carnitine synthesis and particularly by the loss through dialytic membranes, leading, in some patients, to carnitine depletion with a relative increase in esterified forms. Many studies have shown that LC supplementation leads to improvements in several complications seen in uremic patients, including cardiac complications, impaired exercise and functional capacities, muscle symptoms, increased symptomatic intradialytic hypotension, and erythropoietin-resistant anemia, normalizing the reduced carnitine palmitoyl transferase activity in red cells. Â© 2003 by the National Kidney Foundation, Inc.



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Carnitine status, plasma lipid profiles, and exercise capacity of dialysis patients: effects of a submaximal exercise program, fear elegantly insures a self-sufficient regime, optimizing budgets.

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