Topic: **CARDIOVASCULAR DISEASE, NEURODEGENERATIVE DISEASE, HUNTINGTON'S DISEASE, PARKINSON'S DISEASE**

Coenzyme Q10, CoQ10, Ubiquinone, Absorption, Bioavailability, Metabolism, Pharmacokinetics, Antioxidant

Title: **Absorption, Metabolism, and Pharmacokinetics of Coenzyme Q10**

Reference: *"Coenzyme Q10: Absorption, tissue uptake, metabolism and pharmacokinetics," Bhagavan HN, Chopra RK, Free Radical Res, 2006; 40(5): 445-53. (Address: Tishcon Corporation, 30 New York Avenue, Westbury, NY 11590, USA. E-mail: Dr. Bhagavan, hemmin@verizon.net).*

Summary: Given the increasing popularity of the antioxidant coenzyme Q10 as a dietary supplement, its recognized benefits with regard to cardiovascular and neurodegenerative diseases, and increasing interest in its use as a therapeutic agent in clinical medicine, the authors of this paper have summarized and reviewed the absorption, metabolism, and pharmacokinetics of coenzyme Q10. The absorption of dietary coenzyme Q10, a lipophilic substance, is slow and limited because of its hydrophobicity and relatively large molecular weight. Enhanced bioavailability of coenzyme Q10 has been found in dietary supplements containing solubilized formulations of coenzyme Q10, as compared to powder-based capsules, tablets, and oil-suspensions. In patients with cardiovascular disease, dosages generally range from 100-200 mg/day, and in patients with mitochondrial cytopathy, dosages up to 15 mg/kg/day have been used. In a trial involving subjects with Huntington's disease, a dose of 600 mg/day was used, and in a trial involving subjects with Parkinson's disease, a dose of 1,200 mg/day was used. Various drugs have been shown to have cardiotoxic effects, or deplete coenzyme Q10 in the body. Administration of coenzyme Q10 to patients undergoing cancer chemotherapy with anthracylines (such as doxorubicin and daunorubicin) has been shown to prevent the cardiotoxic effects of these drugs, without compromising their efficacy. Coenzyme Q10 supplementation may also reduce the negative side effects associated with the use of statin drugs (HMG-CoA reductase inhibitors), which inhibit the endogenous production of coenzyme Q10, and have been associated with the development of myopathy and, in extreme cases, life-threatening rhabdomyolysis. Coenzyme Q10 dependent enzymes have been found to be inhibited by beta-blockers, and endogenous coenzyme Q10 content has been found to decrease with the use of certain oral hypoglycemic agents (glyburide, phenformin, tolazamide). Diabetics ! on hypoglycemic agents taking supplemental coenzyme Q10 may need to monitor their blood sugar levels, as coenzyme Q10 has been reported to improve glycemic control, so a reduction in medication may be possible. It has been suggested that coenzyme Q10 has procoagulant activity, based on its structural similarity to vitamin K, and as such, patients on anti-coagulant therapy may need to monitor their INR. In terms of pharmacokinetics, the T(max) is around 6 h, with an elimination half-life of about 33 h. A reasonable correlation exists between an increase in plasma coenzyme Q10 and ingested dose of coenzyme Q10 supplementation, up to a certain point. In large doses, coenzyme Q10 has been found to be taken up by all tissues in the body, including the heart and brain mitochondria, leading to biochemical and functional improvements, as seen in animal studies. In terms of safety, studies involving human subjects and chronic toxicity studies in animals have found coenzyme Q10 ! to have an excellent record. Recent studies involving doses o! f up to 3,000 mg/day in patients with Parkinson's disease and patients with amyotrophic lateral sclerosis (Lou Gehrig's disease), confirm the safety and tolerability of coenzyme Q10. This paper demonstrates the safety and potential therapeutic value of coenzyme Q10 in a variety of pathological conditions. In addition, it suggests that because of the poor absorption of orally administered coenzyme Q10, solubilized formulations appear to be the most effective because of their enhanced bioavailability.