NovaQ10

Nutrient Interactions

Vitamin E

Alpha-tocopherol (vitamin E) and coenzyme Q10 are the principal fat-soluble antioxidants in membranes and lipoproteins. When alpha-TOH neutralizes a free radical, such as a lipid peroxyl radical (LOO·), it becomes oxidized itself, forming alpha-TO·, which can promote the oxidation of lipoprotein lipids under certain conditions in the test tube. When the reduced form of coenzyme Q10 (CoQ10H2) reacts with alpha-TO·, alpha-TOH is regenerated and the semiquinone radical (CoQ10H·) is formed. It is possible for CoQ10H· to react with oxygen (O2) to produce superoxide anion radical (O2·-), which is a much less oxidizing radical than LOO·. However, CoQ10H· can also reduce alpha-TO· back to alpha-TOH, resulting in the formation of fully oxidized coenzyme Q10 (CoQ10), which does not react with O2 to form O2·- (See Reaction Scheme) (4, 5).

Deficiency

Symptoms of coenzyme Q10 deficiency have not been reported in the general population, so it is generally assumed that normal biosynthesis and a varied diet provides sufficient coenzyme Q10 for healthy individuals (6). It has been estimated that dietary consumption contributes about 25% of plasma coenzyme Q10, but there are currently no specific dietary intake recommendations for coenzyme Q10 from the Institute of Medicine or other agencies (7). The extent to which dietary consumption contributes to tissue coenzyme Q10 levels is not clear.

Primary coenzyme Q10 deficiency is a rare, autosomal recessive disorder caused by genetic defects in coenzyme Q10 biosynthesis. The resultant low tissue levels of coenzyme Q10 severely compromise neuronal and muscular function. Oral coenzyme Q10 supplementation has been shown to improve neurological and muscular symptoms in some patients with primary coenzyme Q10 deficiency (8). Coenzyme Q10 levels have been found to decline gradually with age in a number of different tissues (1, 9), but it is unclear whether this age-associated decline constitutes a deficiency (see Disease Prevention). Decreased plasma levels of coenzyme Q10 have been observed in individuals with diabetes, cancer, and congestive heart failure (see Disease Treatment). Lipid lowering medications that inhibit the activity of HMG-CoA reductase, a critical enzyme in both cholesterol and coenzyme Q10 biosynthesis, decrease plasma coenzyme Q10 levels (see HMG-CoA reductase inhibitors (statins) under Drug Interactions), although it remains unclear whether this has clinical or symptomatic implications.