Coenzyme Q10 for Heart Failure: the Controversy

As a component of the electron transport chain, CoQ10 plays a key role in energy production, and is therefore essential for all energy-dependent process, including myocardial contraction. Myocardial concentrations of CoQ10 are significantly lower in patients with congestive heart failure (CHF) than in healthy people, but these levels can be increased by oral administration of CoQ10. Numerous clinical trials have shown that CoQ10 supplementation improves symptoms, increases left ventricular ejection fraction, and decreases the number of hospitalizations in patients with CHF. In other studies, however, CoQ10 was of no benefit.

The conflicting results in the different studies might be explained in part by differences in patient populations. One of the negative studies was conducted at an inner-city hospital and a Veterans Affairs hospital. The participants in that study may have had a relatively high prevalence of alcoholism (alcoholic heart disease) and dietary inadequacies. Those factors, combined with the use of diuretics, could have led to clinically significant deficiencies of magnesium, thiamine, or other nutrients that are needed for normal cardiac function. The effectiveness of CoQ10 may be diminished when deficiencies of other cardioprotective nutrients are present.


Nattokinase for Hypertension

Eighty-six patients with prehypertension or stage 1 hypertension (systolic blood pressure of 130–159 mm Hg) were randomly assigned to receive, in double-blind fashion, 1 capsule per day of nattokinase (2,000 fibrinolytic units per capsule) or placebo for eight weeks. Compared with placebo, nattokinase significantly decreased mean systolic and diastolic blood pressure by 5.5 mm Hg (p < 0.05) and 2.84 mm Hg (p < 0.05), respectively.

Comment: Nattokinase is a fibrinolytic enzyme found in natto (fermented soybeans). The results of the present study indicate that nattokinase is an effective treatment for mild hypertension. While nattokinase treatment decreased plasma renin activity to some extent, it had no effect on angiotensin-converting enzyme activity. Therefore, the mechanism of action of nattokinase as an antihypertensive agent is not clear. Although it is a fibrinolytic enzyme, nattokinase does not appear to cause abnormal bleeding. It is not known, however, whether it is safe to administer nattokinase to people taking anticoagulant or platelet-inhibiting medications.


Don't Push the Vitamin D Too Hard

This study examined the relation between serum vitamin D and 25-hydroxyvitamin D (25(OH)D) in normal subjects after oral administration of vitamin D3 (cholecalciferol) or after exposure to ultraviolet-B irradiation. Values for serum vitamin D and 25(OH)D were aggregated from six studies (one acute and five steady state). In three of the steady state studies, vitamin D was administered for 18–26 weeks in doses of 0–11,000 IU/day; in two studies, subjects received solar or ultraviolet-B irradiation. Rapid conversion of vitamin D to 25(OH)D was observed at low vitamin D concentrations, and a much slower rate of conversion was seen at higher concentrations. Thus, at typical vitamin D inputs and serum concentrations, there is very little cholecalciferol in the body, and 25(OH)D constitutes the bulk of the vitamin D reserves. However, at supraphysiologic inputs (that is, at vitamin D concentrations > 15 nmol/L, which is approximately equivalent to 2,000 IU/day or more and corresponds to a serum 25(OH)D concentration of about 88 nmol/L [35.2 ng/ml]), large quantities of vitamin D are stored as cholecalciferol, presumably in body fat, and are slowly released to be converted to 25(OH)D. This occurs because at high vitamin D concentrations, hepatic 25-hydroxylases become saturated and the reaction switches from first order to zero order.

Comment: In recent years, some investigators have proposed that optimal serum 25(OH)D levels are around 90–100 nmol/L or higher. The results of this study suggest that the conversion of vitamin D to 25(OH)D slows at 25(OH)D concentrations below 90–100 nmol/L. Some practitioners have observed that it is often difficult to increase serum 25(OH)D levels to the purported optimal range, even when large doses of the vitamin are administered. That observation is consistent with the conclusion that follows from the present study: serum 25(OH)D is a less reliable indicator of vitamin D status when large doses are being given than when smaller doses are being used. Continually increasing the vitamin D dose in an attempt to reach a target serum 25(OH)D level might lead to excessive accumulation of vitamin D in the tissues, potentially causing vitamin D toxicity.


L-Glutamine for Acute Pancreatitis

Forty-four patients (mean age, 43 years) with severe acute pancreatitis (two-thirds of cases due to biliary disease, 23% due to alcohol) were randomly assigned to receive, beginning after hospital admission, standard parenteral nutrition (control group) or an isonitrogenous, isocaloric parenteral nutrition formula that supplied L-glutamine in the form of L-alanyl-L-glutamine (0.4 g per kg of body weight per day). The number of patients who developed infections was significantly lower in the L-glutamine group than in the control group (41% vs. 73%; p = 0.03). The mortality rate was nonsignificantly lower in the L-glutamine group than...