Coenzyme Q10 and Cancer

Thomas Newman and Stephen Hulley proposed (JAMA 1996 Jan.3)[1] that since fibrate and statin cholesterol-lowering drugs cause cancer in rodents, they might cause unexplained cancers in patients when taken for a long time; and that their use for patients not at high cardiac risk could therefore be unwise. Meta analysis of randomized clinical trials suggests that lipid lowering drugs likely increases non cardiovascular mortality, lending plausibility to the Newman/Hulley proposal.[2]

In reply, I (1) offer a mechanism to explain how statin drugs may cause cancer, and (2) propose inferences for future protection and therapy against cancer and much more. Lovastatin and its statin analogues such as Simvastatin reduce liver synthesis of cholesterol by inhibiting activity of the liver enzyme 3 hydroxy 3 methylglutaryl coenzyme A (HMG CoA) reductase, which is required for the conversion of HMG-CoA to mevalonic acid. Biosynthesis of cholesterol is a multi-reaction pathway that requires mevalonic acid.[3] But cholesterol is not the only product dependent on mevalonic acid: body synthesis of Coenzyme Q10 also depends on it. Inhibiting HMG CoA reductase in order to slow body cholesterol synthesis would therefore be expected to elevate risk of conditions against which CoQ10 protects.[4]

Over the past 25 years, the vitamin-like substance Coenzyme Q10–synthesized in the liver and other cells from lower number CoQs, and ingested in many foods[5]–has been reported to be effective in reducing various cancers and metastases, even in patients for whom all conventional treatments had failed.[6,7] Some of these are alive and well 15 years later without any trace of cancers. AIDS patients showed striking response to therapy with CoQ10; the HIV virus appears to induce a deficiency of CoQ10.[8]

And CoQ10 has no established side effect at any dose level[9]. (Several recent tests used 390 milligrams/day; it is important to increase gradually from a smaller starting quantity).

Lovastatin lowers CoQ10 in laboratory rats,[10] a likely explanation the increased incidence of cancer in the statin drug tests cited by Newman and Hulley.[1] In patients on CoQ10, starting concurrent Lovastatin lowered CoQ10 by 44% to 75% (this finding was confirmed in Italy by G.P. Littarru). The condition of every patient worsened. One required open heart surgery. Another was referred for a heart transplant. Her life was saved by CoQ10 at 200 mg/day,[3] confirming CoQ10's efficacy against certain cardiac conditions. CoQ10 is as essential for survival and health as oxygen, food and water. Present in every body cell, it is often called ubiquinone.[11] Serum levels decline rapidly with age, producing many of the symptoms of aging; a deficiency of 25% is associated with illness, a deficit of 75% with death in animals.[5]

It is widely prescribed in Canada and Denmark. In Japan 12 percent of the population take physician-prescribed CoQ10 at 100-300 mg/day for high blood pressure and cardiac conditions.[5] However, it is largely ignored by medical doctors in the United States. Available over the counter, it is neither effectively promoted nor highly profitable for anyone. There are three possible mechanisms of action for CoQ10's effectiveness against cancer

(1) In its reduced form it is an antioxidant and protects against free radical damage.[12,13]

(2) It is intimately involved in synthesis of adenosine triphosphate (ATP), the basic energy molecule of every cell and thus in generation of 95 percent of the body energy. Karl Folkers and Cyril Bowers demonstrated in a series of papers in the *Journal of Medicine* in the 1970s, that oral CoQ10 administered to

diabetic patients boosts their bioenergetics. It is the role of CoQ10 in bioenergetics (rather than its antioxidant activity) which make it so effective in health and survival.

(3) CoQ10 is a non-specific stimulant of the host defense system.[14] Blood levels of T lymphocytes increased when it was given with pyridoxine (vitamin B_6) or alone; the ratio of T4/T8 T lymphocytes also improved with or without B_6. Contrast this with the energy loss and immune system weakening caused by conventional cancer therapies. It is widely accepted that if a large intake of a nutrient cures a disease, a moderate regular intake of the same nutrient may prevent that disease

From the above evidence and reasoning it follows that (1) Statin drugs could cause cancer in humans when used for decades, by lowering body Coenzyme Q10;[15] (2) Intake of CoQ10 at 50 to 100 mg/day could protect millions against cardiac conditions and cancers. For best results, the supplement needs to be accompanied by substantial quantities of the other members of the antioxidant team in a well-rounded, above RDA program of nutrient supplementation. Also necessary is a diet including moderate quantities of baked or boiled red meat and heavy in fresh, raw or lightly steamed vegetables. (3) High dose CoQ10 may defeat many cancers and their metastases without adverse side effects in humans as well as rats.

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Chronic Sulfite Toxicity

Several years ago while using orthomolecular therapy I developed pigmentation characteristic of Addison's disease. I had suffered from anxiety, cognitive difficulties, fatigue, allergies and rhinitis for many years.

A weekly cysteine supplement was required and an acute cysteine deficiency was alleviated within several months by supplementing B vitamins and 500 mg ni-

acin four times a day. A secondary deficiency resulting from cysteine supplementation required lysine and threonine. Pancreatin and lipase further depleted magnesium and biotin.

Two years ago I began to include lentils in my daily diet. Lentils provide a natural source of sulfite oxidase cofactor and contain a high ratio of lysine and threonine. Three early symptoms of sulfite oxidase activity were the occurrence of a sore throat that responded to manganese and vitamin C but not to vitamin C alone, muscle pain responding to selenium supplements and morning fatigue responding to phosphate. Thiamine deficiency and a requirement for niacin, riboflavin, B_{12} and folate are inherent in sulfite toxicity.

The use of a pharmaceutical sulfite oxidase cofactor requires immediate supplements of selenium, manganese, phosphate and thiamine in addition to niacin and B vitamins. Flax seed oil and cofactors for superoxide dismutase, glutathione peroxidase and glycan synthesis prevent oxidation of ocular tissues. Silica and cofactors for monoterpin synthesis are often required.

During the last two years using orthomolecular supplements the intermittent cognitive disfunction repeatedly responded to selenium, manganese, phosphate, thiamine and niacin. A remarkable improvement in memory resulted from the use of betaine, vitamin C and transmethylation cofactors. Anxiety, allergies, rhinitis and exercise tolerance continued to improve during the duration of treatment with sulfite oxidase cofactor.

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