

Lowering Cholesterol with Lovastatin: The Wrong Approach

A Survey of Usually Overlooked Literature

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Abstract

(1) *Lovastatin and its "-statin" analogues do much more than lower cholesterol. Inhibiting HMG CoA reductase activity, their mechanism of action (a) increases lipoprotein(a), (b) lowers coenzyme Q10, (c) increases risk of conditions against which Coq10 protects, (d) increases risk of arterial damage and (e) suppresses the immune system.*

(2) *Artificially lowering cholesterol is wrong.*

Key words

Co-Enzyme Q10, Oxysterols

I. Lovastatin (Mevacor) and its analogues such as simvastatin lower liver synthesis of cholesterol by inhibiting activity of the liver enzyme 3-hydroxy-3-methylglutaryl-coen-zyme A (HMG-coA) reductase, which is required for the conversion of HMG-coA to mevalonic acid. Biosynthesis of both cholesterol and Co-Enzyme Q10 is a multireaction pathway that requires mevalonic acid.¹

(1) These drugs not only increase lipoprotein(a),² suggested to be an independent risk factor for arterial damage in people with high concentrations of Lp(a) who didn't inherit familial hypercholesterolemia.³⁴ (But see my coming article on the importance of vitamins B6 and C.⁵) The drug raised Lp(a) the most in people who had inherited the highest concentrations.²

(2) The drugs lower biosynthesis of the vitaminlike substance Coenzyme Q10 from lower-numbered CoQs ingested in many foods. Although little known in this country, CoQ10 is as essential for survival and health as oxygen, food and water; because of its multiple services in the body it is often called ubiquinone-10. Serum levels decline with age; a deficiency of 25% is associated with illness,

a deficit of 75% with death in animals.⁶

CoQ10 is a powerful antioxidant, and overwhelming international evidence gathered over a quarter-century confirms that it is indispensable for human cardiac function in other ways as well. It is deficient in cardiac compared to healthy patients ($p < .01$).⁷¹¹ CoQ10 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's energy. In congestive heart failure digitalis, diuretics and vasodilators gave 25% survival after six years; CoQ10 without drugs, 75%.⁹

Lovastatin lowered CoQ10 in laboratory rats.¹² In patients taking CoQ10, starting concurrent lovastatin lowered it by 44%-75%;¹ this finding was confirmed in Italy by G.P. Littarru.¹ 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 per day.¹ Dr. Folkers believes, nevertheless, that the combination of the two is "scientifically sound" [personal communication 1992].

CoQ10 protects the body against gum disease, cancer and allergies (it dispatched all my allergies), among many others, and in double blind tests it improved intellectual ability.⁶ Consequently, taking lovastatin for a long time is likely to worsen the listed afflictions, lower pregnenolone and DHEA (see below), possibly cause muscular dystrophies [A. Hoffer personal communication 1992], and make patients more stupid—unless CoQ is also taken. Better, why not take the CoQ10 and see if the medicine can be phased out?

CoQ10 has "no known side effects at any dose level" [K. Folkers personal communication 1991]. However, anecdotal evidence suggests starting with a small quantity [P. Bruwer interview 1992] and gradually increasing it. I started with 10 mg/day and gradually increased to 50 (at the age of 70), most of it in the morning to avoid interfering with the night's

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sleep. The increase in stamina was remarkable and permanent; my stamina now is greater than 20 to 50 years ago, suggesting I was always deficient in CoQ10.

It requires a prescription in Japan, where 12 million (over 10 percent of the population) take it daily for cardiac disease and high blood pressure, usually at 100-300 mg/day. Our FDA (U.S. Food and Drug Administration), following the dictates of the amoral, extremely powerful international drug cartel,¹³ is bending every effort to get every natural substance with health-promoting qualities including CoQ10 out of health food stores onto prescription status at many times higher prices.

Then the drug companies can make big claims for it, and they and the physicians and hospitals that will use it can reap big profits on it. CoQ10 offers "unfair" competition where it is because it (a) isn't patentable and so (b) is inexpensive and not profitable, hence little used (what gets used is what pays, not work works), and (c) often helps make people well so they are no longer patients.

(3) Suppression of HMG-coA reductase activity, which has been known since the early 1960s, is one of the mechanisms by which oxidized cholesterol molecules called oxysterols (see below) (a) damage arterial walls and (b) suppress the immune system by inhibiting macrophage function.^{14,15}

The multiple ailments caused by lowering CoQ10 will be treated by other profitable drugs, creating new iatrogenic diseases to be treated by still other dangerous but profitable drugs. Deaths of people on long-term lovastatin will be wrongly blamed on "high cholesterol."

II. The entire approach of lowering cholesterol is faulty. The Framingham Project found a significant correlation between total cholesterol and heart-attack risk only in males from their low 30s to their early 60s—not over one-third of the adult population.¹⁶ "Lowering cholesterol 1 percent lowers heart attack risk 2 percent" was accurate only at the far right end of the distribution for those particular males.¹⁶ Extrapolating these findings to everyone violates more than common sense.

Further, results of cholesterol lowering trials use only the numerator without the denominator. So an absolute rate increase of 6.7 percent is represented as a "relative risk"

elevation of 500 percent. The term has no meaning. Authors of such work criticize others for using the procedure¹⁷ but use it themselves to bamboozle the public into believing present methods are working, so that the drug companies, doctors and researchers can continue their profitable though futile ways—and the public be damned.¹⁸ For the editors and peer reviewers of medical journals to allow this rubbish to be printed is a dereliction of their trust.¹⁷

Why is cholesterol-lowering wrong? This lipid does not participate in the initial arterial injury; it may not pile up in arteries or accumulate in the blood for months after such injury.¹⁹ It was caught at the scene of the crime like a schoolboy seen throwing the last snowball after a window was already broken. Not only is cholesterol essential for innumerable body functions, four-fifths is generated in the body.

Rising levels of cholesterol, uric acid²⁰ and others are among the body's defensive responses to arterial injury. They are signs of disease like a fever, not the disease or "risk factors" themselves.²¹ And so artificially lowering cholesterol, like contriving to reduce a fever with aspirin—from Darwinian medicine—hinders the body's efforts to protect itself!

Dean Ornish's patients got better exercising, lowering stress and eating a vegetarian diet without supplements. Carefully matched, randomly selected control patients on the American Heart Association diet taking their heart specialists' prescribed drugs got worse: their arteries continued to narrow. Ornish attributed the improvement in test patients to their lowered cholesterol.²²

But since rising cholesterol is a sign of disease like a fever, not its cause, falling cholesterol cannot improve health—any more than a dropping fever cures a patient of pneumonia. His patients were exposed to the same environmental sources of oxysterols as everyone else.²³ The test patients got better because (i) they put little if any oxidized cholesterol into their mouths;^{24,25} (ii) they prevented oxysterols' formation in their bodies from animal protein;¹⁹ (iii) their diet had to provide at least antioxidant vitamins C, beta-carotene and CoQ10 to handle oxysterols from all sources.²³

Consider the terminology. Cholesterol car-

ried by LDL (low-density lipoprotein) cholesterol is labeled "bad" because it moves lipids to arterial walls; that carried by HDL (high-density lipoprotein) cholesterol, called "good," shuttles them to the liver for disposal. But why would nature create LDLs if they kill us? LDLs perform necessary functions: for one, they carry beta-carotene and CoQ10. Too little LDL may not carry enough of those valuable antioxidants to inhibit growth of tumor cells,²⁶ helping explain why artificially lowering LDL cholesterol promotes cancer.

And LDLs function as the steroid-forming precursor to deliver anti-aging pregnenolone, the precursor of progesterone and DHEA— all of them required for good health.²⁶ Further, cholesterol itself serves as an antioxidant to help rid the body of toxins from the environment and diet.²⁷ This helps explain why the body makes cholesterol rise after arterial damage and makes it drop as plaques are reversed, and further helps explain why excessive lowering of cholesterol promotes cancer.

All cholesterol is benign until it is oxidized.^{24,27,28} For details on how cholesterol in the body gets oxidized, see the Multi-Source Oxysterol Injury Hypothesis of Atherogenesis²³ and my article comparing the importance of vitamins B6 and C.⁵ Margarine and other partially hydrogenated polyunsaturated fatty acids (PUFAs)-containing food products also generate oxysterols in the body.²⁹ To learn what certain pioneering physicians have done to protect patients against oxysterols and the reasons why their therapy works, see my review article.²³

Lowering cholesterol naturally by the supplements and diet regimen described there elevates mood. But reducing it excessively by the chemical manipulation of drugs decreases the brain neurotransmitter serotonin, increasing hostility and agitated mental states. Victims are more depression, suicide and accident prone. It is safe, in sum, to predict that proper clinical trials will show lovastatin sickens and kills. To have begun massive worldwide use of the drug before long-term safety trials, after the same procedure proved to be disastrous with clofibrate strikes me as unholy haste to reap the big bucks.

Conclusion

After the 200-percent increase in cardiac deaths among test patients using diuretics in

the Oslo Heart Trial³⁰ and the time-bomblike findings in Finland,³¹ the National Health Institution of Helsinki called for a moratorium on use of cholesterol lowering drugs.³¹ In view of those results and the evidence and reasoning here presented, such a moratorium should be made universal and permanent.

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