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CHOLESTEROL: A PATIENT-SPECIFIC NUTRITIONAL APPROACH

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INTRODUCTION

When asked how he had been so successful in blazing a new path of thought Einstein replied, "I did it solely by challenging axioms." It is the intent of this article to challenge the universally accepted idea that dietary intake of cholesterol is correlated directly with cholesterolemia and with myocardial infarction. A patient-specific approach to the problem of hypercholesterolemia and coronary artery disease is offered.

An axiom, though based upon scientifically verifiable facts, is not itself provable. In other words, an axiom is an extrapolation of the facts, i. e.; it involves "jumping to conclusions." From certain scientifically measurable facts it was once axiomatically accepted that the earth was flat and the sun revolved around the earth. An extrapolation of facts always involves making assumptions; assumptions which in this case clearly were inaccurate.

A statistically significant correlation between hypercholesterolemia and myocardial infarction has been demonstrated. From this irrefutable fact it is accepted as self-evident the assumption that dietary cholesterol intake leads to elevated cholesterol and thence to heart attack. It must be understood that this is indeed an assumption. Nowhere, to the author's knowledge, has a cause and effect relationship been established between cholesterol intake and either cholesterolemia or atherosclerosis.

If high cholesterol and cardiovascular disease do not result from eating cholesterol, then where do they come from? Numerous studies have shown that it is aberrant lipid metabolism, not over-consumption, that causes cholesterol problems. This knowledge allows the clinical nutritionist to approach patients with something more than advice to quit eating cholesterol. By specific testing, the metabolic imbalance that has deranged lipid metabolism in each individual patient can be identified. Having defined the individual's underlying biochemical imbalances, the clinician can prescribe a specific nutrition regimen designed to restore normal metabolism, rather than treating high cholesterol as a disease entity per se. It is thus possible to correct the cause of the problem, more than merely minimizing its effects.

RESEARCH REFUTING THE CHOLESTEROL AXIOM

Following are a number of little known facts regarding cholesterol which are in direct conflict with commonly accepted assumptions.

Cholesterol is a requirement of every living cell and we cannot live without it. It is the building block of sex hormones. Fifteen percent of the dry weight of the human brain is cholesterol (5, 9).

The body synthesizes 2,000 mg. daily of this essential substance. In comparison, even a high cholesterol diet provides only about 800 mg. Furthermore, when large quantities of cholesterol are ingested the body simply synthesizes less such that an excess is avoided. Animal studies which induced atheroma with dietary cholesterol used the human equivalent of 15,000 mg. of cholesterol a day (1).

Animals fed a diet consisting of 81% animal fat, but with concurrent high levels of protein, vitamins and minerals showed no pathological changes in the aorta or the heart (2, 8).

Studies of primitive African cultures have shown no correlation between dietary intake and atherosclerosis even among 400 men of the Masai tribe who ate meat and milk exclusively. A strong correlation does exist, however, between atherosclerosis and consumption of refined sugar and flour (6, 7).

In 1914 only 15% of all heart disease was atherosclerotic in nature; today that has risen to over 90%. Over 50% of adult Americans now die of cardiovascular disease. Yet the only significant change in dietary patterns in Western countries over the last 100 years has not been in fat consumption but in refined sugar and flour intake (15).

Hundreds of millions of dollars have been spent on research attempting to prove that eating foods high in cholesterol increases the risk of heart attack. No such evidence has been produced (3).

Even the existence of a statistical correlation between hypercholesterolemia and myocardial infarction does not necessarily establish a cause and effect relationship. How do we explain the countless patients in our practices whose x-rays show arteriosclerosis of the aorta, yet who have normal serum cholesterol? How do we explain the patients who have cholesterol levels over 300 yet show no evidence of cardiovascular disease? How do we explain to the heart attack victim with normal cholesterol that he is a statistical fluke?

To conclude, based on epidemiological studies, that serum cholesterol and heart attack are cause and effect seems a careless assumption. But to extrapolate from those statistics that dietary cholesterol causes cardiovascular disease represents a rather blatant "jumping to conclusions."

UNDERSTANDING HYPERCHOLESTEROLEMIA

So what does it actually mean when a patient has high serum cholesterol? The answer to this question lies in a study not of cholesterol, but of the individual and his or her unique body chemistry. Hypercholesterolemia is merely a symptom arising from one of two possible underlying metabolic imbalances.

Nutri-Spec has developed the means by which a clinician can obtain complete evaluation of a patient's body chemistry using objective test procedures. Urine and saliva chemistries as well as clinical tests are employed in identifying the patient's metabolic imbalances (11). The Nutri-Spec system represents a radical departure from disease-specific methods of diagnosis and treatment, in favor of a purely patient-specific approach.

Nutri-Spec has defined five fundamental metabolic balance systems, the operations of which are involved in maintaining homeostasis of all physiological processes. All pathology reflects a loss of homeostasis associated with aberration in one or more of the five fundamental balances (12). Every patient's symptoms, therefore, have a nutrition component and will benefit from restoration of metabolic balance (13, 14).

When studied in this light, atherosclerosis is seen as a dysfunction in two of the fundamental balances, namely, water/electrolyte balance and anaerobic/dysaerobic balance. While a discussion of water/electrolyte balance lies outside the theme of this article, anaerobic/dysaerobic imbalance fully explains the vast majority of high cholesterol problems.

Anaerobic/dysaerobic balance concerns not only the problems of oxidative energy production, but also represents the two opposite abnormalities of lipid metabolism. An anaerobic patient has insufficient fatty acid activity and excess sterols; the dysaerobic patient has excess fatty acids and insufficient sterols.

Cholesterol is a sterol fat. An excess, therefore, represents an anaerobic imbalance, while low levels correspond to a dysaerobic condition. However, there is a vital fact about cholesterol of which most are not cognizant; its biological role is played only at the cellular level. This means that serum cholesterol levels say absolutely nothing about a patient's cholesterol status (10).

Hypercholesterolemic patients can be either anaerobic or dysaerobic. If anaerobic, their cells are so saturated with cholesterol that it has now begun to accumulate in the serum. A dysaerobic patient actually has low cellular cholesterol due to excess fatty acid activity there. Serum levels rise as the cholesterol is unable to penetrate the cells.

Clinically this means that there is no treatment for high cholesterol per se. Effective therapy is contingent upon determining the patient's fundamental biochemical imbalance. Having done so, the clinician can confidently prescribe the diet and supplements specific to the individual patient's needs.

The anaerobic patient responds to one or more of the following supplements: negative valence sulfur, vitamin B6, magnesium (orotate or aspartate), L-carnitine, copper, and proteolytic enzymes (bromelaine, pancreatin). Dietary recommendations include avoidance of sugar, alcohol, fermented foods, and sterol fats.

If dysaerobic, the patient's supplemental needs will be met from the following: glycerol, choline, inositol, potassium (orotate or citrate), bioflavenoids, and niacin. The diet must avoid free fatty acids and trans fatty acids (vegetable oils, margarine, salad dressing, fried foods, canned meats), and include sterol fats (--- Yes, the diet must include high-cholesterol foods such as eggs).

CASE HISTORY

A 45-year old male presented with serum cholesterol of 324, and a blood pressure of 138/94. Nutri-Spec testing revealed a dysaerobic imbalance as well as a water/ electrolyte imbalance. He was put on a salt restricted diet and supplemented with mineral dispersing agents for his water/electrolyte imbalance. Being dysaerobic, he was given the appropriate diet and supplementation.

In five weeks his cholesterol had dropped to 184. His blood pressure had dropped to 121/77.

This case history reiterates our point that there is no disease-specific nutrition treatment for hypercholesterolemia. The patient was treated not for his disease, but per his biochemical imbalances. His cholesterol, being merely an effect of underlying causes, responded dramatically when those causes were corrected.

SUMMARY

Objective clinical testing procedures are the only consistently efficacious means to implement patient-specific diagnosis and treatment. The familiar cliche' applies, "We must treat the patient and not the disease."

This statement of traditional wisdom could not be more true of hypercholesterolemia. Routine prescription of a low cholesterol diet and not much else is woefully inadequate case management, and allows what is a reversible condition to progress to the stage of life threatening tissue degeneration.

The cholesterol axiom stands without foundation. The true causative factors of hypercholersterolemia can be reliably determined and effectively treated.

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