Pharma Versus Farmer
Food as Heart Medicine*

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Evaluating drugs is easy; assessing the healthfulness of food is not. For drugs, we have a proscribed process: determine the dose; screen for toxicity; and then randomly give the drug or its placebo to enough subjects for a sufficient length of time to acquire statistically meaningful endpoints. Deciphering good nutrition is far more complex. As omnivores, our food choices are endless. We select food by habit, taste, cost, and availability. Food healthfulness trails these selection factors and is generally poorly understood, even by the medical community. On average, medical students receive only 23.9 h of instruction on nutrition (1).

Drug trials are well designed. Baked and broiled fish reduce the incidence of heart failure; fried fish does the opposite (2). As with drugs, food combinations affect their biological impact. Endothelial function is decreased by olive oil alone, but not when consumed on a salad (3). Evaluating food requires long-term studies because diseases such as atherosclerosis take years to decades to become manifest. Unlike drug trials, food trials cannot be blinded, and adherence to an experimental diet is often problematic. Importantly, removal of a food or macronutrient from a diet to assess its potential harm requires adding back some other food to maintain energy intake. Without certainty of the healthfulness of the substituted food, such an assessment is meaningless.

Although factually correct, last year’s meta-analysis showing a neutral effect of dietary saturated fatty acids (SFA) on coronary heart disease (CHD) lacked insight into food substitution (4). Adding to the public’s confusion, a responding Time magazine cover story was headlined “Eat Butter. Scientists labeled fat the enemy. Why they were wrong” (5). In this issue of the Journal, Li et al. (6) clarify substitutions for SFA to reduce CHD.

Combining data from the large, observational Nurses’ Health and Health Professionals Follow-up Studies, these investigators report that replacing 5% of energy intake (about 100 kcal) from SFA with equivalent energy from polyunsaturated fatty acids (PUFA), monounsaturated fatty acids, or carbohydrates from whole grains significantly reduced CHD by between 9% and 25%. In contrast, replacing SFA with carbohydrates from refined starches or sugars (5% of energy intake) increased CHD risk by 10%, and substitution with trans fats (2% of energy intake) increased CHD risk by 20%. This clarification has important public health implications. We have been rightly demonizing saturated and trans fats, but ignoring the adverse impact of refined starches and added sugars on CHD. The current study also reports that neither low total fat nor low total carbohydrate diets were associated with reduced CHD risk. It is time to set aside the low-fat versus low-carbohydrate diet debate. Healthfulness clearly lies in the quality or type of both fat and carbohydrate.

The benefit of substituting PUFA for SFA is well supported, although the most convincing randomized controlled trial literature is fairly old. A good example is the Finnish Mental Hospital Study, which, using a

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crossover design, compared a usual diet served in 1 facility with a diet in which PUFA replaced SFA in another facility (8). Leaving aside issues of subject consent, diet adherence was ensured. The PUFA-substituted low-SFA diet significantly reduced serum cholesterol by 42 mg/dl and reduced nonfatal myocardial infarction plus CHD death by approximately one-half in both crossover periods. In contrast, a meta-analysis of 11 cohort studies in which SFA were replaced by carbohydrates showed no reduction in CHD (9).

Is a dietary carbohydrate as bad as a saturated fat? As with fats, the answer totally depends on the type of carbohydrate. Again, the current study provides useful clarification of carbohydrate type beyond the usual simple versus complex (saccharide chain length) or glycemic index (blood glucose rise) classifications. In this study, carbohydrates were divided into 3 categories: those from whole grains; those from refined starchy and sugars; and those from fruits, vegetables, and legumes. Fruits, vegetables, and legumes were not included in the analysis because their health benefit is thought to be independent of their carbohydrate content, a point I will discuss later. The nutrition facts box required on packaged food lists SFA and trans fat content (monounsaturated fatty acids and PUFA may also be listed), but it is more difficult to recognize true whole-grain foods, especially when faced with pervasive product mislabeling. True whole-grain foods should list whole-grain wheat or other whole grains as the first ingredient. Inclusion of bran is also a good sign, but terms such as refined grain, bleached grain, or even multigrain do not connote healthfulness. Importantly, true whole-grain breads, cereals, and pasta should minimally contain 3 g of fiber per serving.

Nutrition science is slowly realizing the adverse cardiovascular effects of refined starchy and added sugars. In a National Health and Nutrition Examination Survey study of 11,733 healthy subjects, daily consumption of >25% of energy intake from added sugar was associated with an almost 3-fold increase in cardiovascular disease mortality compared with that of subjects with <10% added sugar intake (10). Three 12-oz cans of most regular soft drinks provide >25% of daily energy intake from added sugar. A meta-analysis of 39 dietary trials recently showed that a high intake of added sugar is associated with increased low-density lipoprotein cholesterol, triglycerides, and blood pressure, the latter by 7/6 mm Hg (11).

One important limitation of the current study is that all SFA do not have the same association with CHD (4). The most common SFA, palmitic and stearic acids (found in meat and dairy products), are almost certainly associated with CHD. Less common SFA, such as margaric acid, are probably harmless. Even for SFA, it is misleading to make blanket categorizations. The other limitations of this study are inherently those of food versus drug research. The study is observational, probably confounded to some degree by unmeasured factors, and does not prove causation. The investigators excluded fruits, vegetables, and legumes because of their association with reduced CHD, without definitive evidence from randomized trials. Whereas randomized trials have recommended increased fruit and vegetable intake as part of overall dietary changes, none have dealt solely with this food category. The fructose in fruit differs from that in regular soft drinks only by quantity, although the latter is an important factor in hepatic conversion to glucose versus triglycerides. Clearly, fruit is more than fructose, and its fiber and micronutrients make a difference. Our current understanding of nutrition is not sufficient to assign benefit to the specific micronutrients that make fruits and vegetables so healthful. Alcohol, a simple sugar, was also excluded because of its widely recognized association with reduced CHD, especially as consumed in the Mediterranean diet. Lastly, even the most detailed, self-reported diet questionnaire is never as accurate as a good pill count.

What we are left with is a slightly clearer message about food as heart medicine. We in health care need to be better informed about nutrition and nutritional research and have a clearer public health message. The challenge will be to convince an increasingly wary public that we know what we are talking about.

**REFERENCES**


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