

The Retreat of the Diet-Heart Hypothesis

Uffe Ravnskov, M.D., Ph.D.

The original diet-heart hypothesis was overly optimistic. That's the word from Frank B. Hu and Walter C. Willett, well-known epidemiologists from Harvard University.¹

Over many years Willett and his group have published a large number of cohort diet studies of hundreds of thousands of healthy individuals. They have analyzed dietary differences in detail between those who have remained healthy during the observation period and those who have become sick or have died from various diseases, mainly coronary heart disease (CHD).

The results from these studies, together with an extensive review of all metabolic studies and clinical experiments, inspired them to present new dietary advice aimed at preventing CHD.

Hu and Willett say the interest should not focus solely on serum total cholesterol or low-density lipoprotein cholesterol (LDL-C), because the effects of diet on CHD can be mediated through multiple biological pathways. Moreover, use of a single intermediate end-point as a surrogate of CHD risk could be misleading.

More specific and firmer evidence on diet and CHD is now available. Hu and Willett list a number of other intermediate biological mechanisms that may influence risk of CHD, such as blood pressure, thrombotic tendency, cardiac rhythm, endothelial function, systemic inflammation, insulin sensitivity, oxidative stress, and homocysteine level.

Saturated fatty acids and trans-fatty acids raise LDL-C, whereas polyunsaturated fatty acids lower it. Therefore we are advised to replace saturated and trans-fat with non-hydrogenated unsaturated fat or omega-3 fatty acids, for instance by replacing red meat with chicken and fish. The authors add, however, that except for the trans family, all fatty acids—in particular the saturated ones—raise the “good” HDL cholesterol.

Although geographic and migration studies have shown the importance of environmental factors in the cause of CHD, they are seriously confounded by other factors for which it is difficult to adjust. According to Hu and Willett, prospective cohort studies are more reliable, but they are “surprisingly few.”

The largest and most detailed is their own study of 80,062 healthy nurses. Here, high consumption of trans-fatty acids, and to a lesser degree saturated fatty acids, was associated with an increased risk of CHD, whereas high consumption of mono- and polyunsaturated fatty acids was associated with a decreased risk. No association was seen between dietary cholesterol and CHD or stroke.

Admittedly, there is little support from the controlled, randomized dietary trials, say Hu and Willett. However, there is increasing evidence that omega-3 fatty acids may reduce the risk of CHD, most probably because of beneficial effects on cardiac rhythm and endothelial dysfunction and a decrease in thrombotic tendency.

Hu and Willett give a list of supposedly beneficial food items such as nuts, fish, fruits, and vegetables, particularly green leafy vegetables and whole grains. Warnings are given against red and processed meats, sweets and desserts, potatoes, French fries and refined grains. But, as they add, simply lowering the percentage of energy from total fat in the diets is unlikely to improve lipid profile or reduce CHD incidence. The same applies for treating obesity by diet; long-term clinical trials have provided no good evidence that reducing dietary fat per se leads to weight loss.

Comments

Although Hu and Willett warn against using surrogate outcomes such as lipid values as evidence, they do this themselves. Food items are frequently recommended not because their benefit has been proved in clinical trials but because they lower the bad or raise the good cholesterol. Their argument is that the evidence is strongest when results from different types of studies are consistent.

There is no such consistency, however: Lowering cholesterol by dietary means does not improve health. There isn't “little support” from the trials: there is none at all. Two meta-analyses of all controlled, randomized dietary trials, in which the only type of intervention was a lowering of dietary saturated fats, an increase of dietary polyunsaturated fats, or both, found that the total number of deaths was identical in the treatment and the control groups.^{2,4}

Also, Hu and Willett's warnings against emigration and geographic studies should have included all types of epidemiological studies, including their own cohort studies, because such studies are prey to bias from numerous factors that are difficult or impossible to control.

It is elementary that epidemiological evidence cannot be used to establish causality, but only to create hypotheses. For instance, the observation that heart patients have eaten less fruit, nuts, fish, and vegetables, and more bread and potatoes does not necessarily mean that such a diet increases heart disease, but may reflect a lower social position and a bad economy, thus automatically introducing many other factors with a detrimental influence on health. As Hu and Willett base their dietary advice mainly on these epidemiological studies, it has little credibility, in particular because their references are both incomplete and misleading.

Of the 11 cited studies, nine of which are cohort studies, only one found heart disease to be associated with a high intake of saturated fat. In addition to the 10 cited studies in which no association was found, there are 11 other studies, not mentioned by the authors, that were also nonsupportive.² In their own study, their most important support, the association between heart disease and saturated fat disappeared when other dietary factors were controlled.⁵ Neither do they mention that in three studies coronary patients had eaten significantly more polyunsaturated fat than those without coronary disease.²

There is much evidence to suggest that omega-3 fatty acids have a beneficial influence on cardiovascular disease. However, as the physiological effects of omega-6 fatty acids on the eicosanoid hormone system are the opposite of the omega-3 fatty acids, imbalance may occur not only by eating too little of omega-3, but also by eating too much of omega-6 fatty acids.

Unfortunately the dietary advice we have been given for many years has resulted in a gross excess intake of omega-6 fatty acids because current guidelines stress vegetable oils. These guidelines amount to a large-scale experiment using human beings as the test animals, because numerous studies have shown that an excess of vegetable oils rich in omega-6, especially if heated, are detrimental to the immune and reproductive system of experimental animals. This was also the reason that the initial advice, to eat as much polyunsaturated fats as possible, was changed and provided with an upper limit of 10 percent of the total calorie intake. However, the public has never been told of this limit or the reason for it.

The chief reason for Hu and Willett to present new dietary guidelines has most probably been the dismal failure of the present ones. Since the low-fat, high-carbohydrate message was introduced more than a decade ago there has been a concomitant and notable increase of obesity and type II diabetes in the United States. This is commented upon only briefly in Hu and Willett's conclusion and not mentioned in the abstract. Neither does it mention that the dietary trials have failed, that a low-fat diet doesn't work, nor that dietary cholesterol has no impor-

tance—findings that certainty would send shock waves through mainstream health-related organizations.

Hu and Willett should be credited for the first attempt by establishment researchers to question the diet-heart hypothesis. As in war, progress is proclaimed with pomp, while defeats are whispered if reported at all. However, the body of contradictory evidence is available.^{6,7}

Uffe Ravnskov, M.D., Ph.D., of Lund, Sweden, is an independent researcher (see www.ravnskov.nu/uffe). He is the spokesman for THINCS, The International Network of Cholesterol Skeptics (www.thinco.org) and author of *The Cholesterol Myths*. E-mail: uffe.ravnskov@swipnet.se

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