

RED MEAT AND OUR HEALTH: SEPARATING SCIENTIFIC FACT FROM POLITICS, EMOTION AND MISINFORMATON

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The 2010 Dietary Guidelines for Americans recommends restricting our intake of saturated fat to less than 7 percent of calories, and our cholesterol intake to less than 300 mg per day (less than two eggs). They promote the use of low-fat milk and lean meat, and the use of “meat substitutes” in school lunches. These recommendations are consistent with the official dietary policy that began in 1977 with the release of the first Dietary Goals for the United States by the United States Senate Select Committee on Nutrition and Human Needs. These guidelines were not justified by the then-available science. They were adopted despite the concerns of researchers and physicians. Subsequent research has disproven the hypothesis upon which they were based. They have failed to produce the promised benefits. Since animal products are a significant source of saturated fat and cholesterol, the official advice has been to limit the consumption of animal products in general and red meat in particular. At best animal products have been wrongly accused and unfairly impacted by public policy; at worst vast physical and fiscal harm has been done to the American public.

Introduction: A thorough discussion of diet, health and human nutrition is beyond the scope of this paper. The comprehensive review by Taubes (2008) is highly recommended. Rather, this will be a brief examination of the dietary cholesterol and saturated fat recommendations.

In 1977 the United States Senate Select Committee on Nutrition and Human Needs chose one side of an on-going scientific debate. They endorsed the unproven diet-heart hypothesis, which proposed that the excessive consumption of fat in our diets – particularly saturated fats – raises serum cholesterol levels and so causes atherosclerosis, heart disease, and untimely death (Taubes, 2008). That decision was antithetical to the then-mainstream paradigm of the fattening carbohydrate, since low fat diets are higher in carbohydrates by definition. Ultimately, the goal of all dietary policy became reducing heart disease, and what was good for the heart must be good for every other diet-related matter. Thus an unproven hypothesis became the unquestioningly accepted basis for dietary recommendations for over a generation. The 2010 Guidelines, the “federal government's evidence-based nutritional guidance to promote health, reduce

the risk of chronic diseases, and reduce the prevalence of overweight and obesity,” (USDA, 2011) continues to maintain this position. The USDA’s admission that despite their dietary advice, “more than one-third of children and more than two-thirds of adults in the United States are overweight or obese.” (USDA, 2011) suggests the need for a thorough re-evaluation of the diet-heart hypothesis. A brief examination of the effect of dietary cholesterol upon serum cholesterol levels, and the relationship between saturated fat and coronary heart disease will demonstrate that this hypothesis was not true and that advice to limit the consumption of animal products is groundless.

Discussion: At the time of the Committee’s decision there was a vigorous scientific debate about the diet-heart hypothesis. “Two strikingly polar attitudes persist on this subject, with much talk from each and little listening between.” (Blackburn, 1975). Three years later, the year after Dietary Goals was released, Thomas Dawber wrote: “It must still be admitted that the diet-heart relation is an unproved hypothesis that needs much more investigation.” (Dawber, 1978). Indeed, the Committee didn’t even know if their recommendations would work. The first entry on their list of “Important questions, which are currently being investigated” was “Does lowering the plasma cholesterol level through dietary modification prevent or delay heart disease in man?” (Senate Committee, 1977) Available research suggested it would not.

Two Columbia University biochemists had demonstrated in 1937 that dietary cholesterol has little or no influence on serum cholesterol (Rittenberg, Schoenheimer, 1937). This finding has never been refuted. For most individuals, the effect of following the recommendation would be “clinically meaningless.” (Howel et al., 1997). Nevertheless, we are still advised to eat less cholesterol because “telling people they should worry about cholesterol in their blood but not in their diet has been deemed too confusing” (Taubes, 2008). Lowering serum cholesterol by replacing saturated fat with polyunsaturated fats had produce mixed results. Such cholesterol lowering interventions occasionally reduced heart disease mortality, but they increased cancer mortality (Dayton et al., 1969), so there was no decrease in total mortality. More deaths were recorded in the intervention group of one study, but the results went unreported for 16 years (Franz et al., 1989), because “we didn’t like the way it turned out.” (Taubes, 2008). This relationship between low cholesterol and increased cancer mortality has been repeatedly observed (Feinleib, 1983).

Ironically Ancel Keys, the father of the diet-heart hypothesis, reported seven years after the Guidelines were released that neither high cholesterol nor saturated fat consumption predicts total mortality (Keys et al, 1984). Keys later recanted the idea that dietary cholesterol raises blood levels: “Cholesterol in food has no effect on cholesterol in blood and we’ve known that all along.” “I’ve come to think that cholesterol is not as important as we used to think it was,” he said, “Let’s reduce cholesterol by reasonable means, but let’s not get too excited about it.” (Boffey, 1987).

Just when the Committee was forming the guidelines that would shape the eating habits of every American, the first reports on Low Density Lipoprotein (LDL) cholesterol and High Density Lipoprotein (HDL) cholesterol were emerging from the Framingham,

San Francisco, Puerto Rico, Albany and Honolulu cohort studies. They demonstrated that: Total cholesterol does not predict future heart disease; LDL cholesterol is a “marginal risk factor;” HDL cholesterol is a 4-fold better predictor of risk than LDL cholesterol and the only reliable predictor of risk for men or women over 50. It was demonstrated that saturated fat raises HDL cholesterol while carbohydrates lower it (Castelli et al, 1977, Gordon et al, 1977). It was reported in 1981 that saturated fat and total fat were positively associated with longevity (Gordon et al, 1981, Feinleib, 1981). This information would not deter policy makers from labeling saturated fat “artery-clogging” and that carbohydrates were “heart-healthy diet food.” The 2010 Guidelines, still state that “Healthy diets are high in carbohydrates.” (USDA, 2010).

The basis for recommending low-fat and low-saturated fat diets has been further disproven by recent research. Meta-Analyses on “Reduced or modified dietary fat for preventing cardiovascular disease” found no effect on longevity, and no “significant effect on cardiovascular events.” (Hooper et al, 2001). An analysis of “Multiple risk factor interventions for primary prevention for coronary heart disease” demonstrated that “The pooled effects suggest multiple risk factor intervention has no effect on mortality.” (Ebrahim et al. 2006) The Women’s Health Initiative failed to prove several frequently-stated dietary myths, although policy hasn’t been affected. “The intervention did not reduce risk of CHD or stroke.” (Howard et al. 2006) “A low-fat dietary pattern did not result in a statistically significant reduction in the risk of invasive breast cancer...” (Prentice et al. 2006). “There is no evidence that a low-fat dietary pattern intervention reduces colorectal cancer risk...” (Beresford et al. 2006). “A low-fat dietary pattern among generally healthy postmenopausal women showed no evidence of reducing diabetes risk...” (Tinker et al. 2008). Prior to the release of the 2010 Guidelines, the FAO stated that “The available evidence from cohort and randomized controlled trials is unsatisfactory and unreliable to make judgment about and substantiate the effects of dietary fat on risk of CHD.” (FAO, 2010, Skeaff, Miller, 2009). And in 2010 “A meta-analysis of prospective epidemiologic studies showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD.” (Siri-Tarino et al 2010) Yet the recommendations to restrict total fat and saturated fat consumption continue.

Substantial evidence has accumulated that these recommendations are in fact harmful. “The low-fat, high-carbohydrate diet, promulgated vigorously ... by the USDA food pyramid, may well have played an unintended role in the current epidemics of obesity, lipid abnormalities, type II diabetes, and metabolic syndromes.” (Weinberg, 2004).

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