Controlling Dietary Total and Saturated Fat To Reduce Cardiovascular Disease:
A Classic Public Health Error

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Introduction

Several years prior to his famous Seven Countries Study, Ancel Keys had already begun to characterize fat as the dietary equivalent of cigarettes. It was the early 1950s, and he had no real evidence for this claim; nonetheless, he advised Americans to limit fat intake to 30% of total calories.\(^1\) Subsequently, the *Dietary Goals for America* of 1976 endorsed Keys’ 30% rule and added to it, limiting saturated fat to 10%. The current *Dietary Guidelines for America* restates this as follows:

Consume less than 10 percent of calories from saturated fatty acids … keep total fat intake between 20 to 35 percent of calories, with most fats coming from sources of polyunsaturated and monounsaturated fatty acids, such as fish, nuts, and vegetable oils. When selecting and preparing meat, poultry, dry beans, and milk or milk products, make choices that are lean, low-fat, or fat-free. Limit intake of fats and oils high in saturated and/or *trans* fatty acids, and choose products low in such fats and oils.\(^2\)

The public health theory that saturated fat and heart disease are inextricably connected has been communicated to marvelous effect, and today it is difficult even to *think* of saturated fat as anything but harmful. Yet, as we shall see, this uniformly accepted hypothesis has no reliable supporting evidence, and public policy based on it has arguably done more harm than good.

Origin of the Idea

In the decade after the end of World War II, the public health community in the United States focused increased attention on heart attacks and strokes. Arteriosclerosis had been discovered, and researchers simplistically assumed that the deposits of cholesterol and fat seen in sclerotic vessels were the residue of dietary cholesterol and fat. When the results of the Seven Countries Study and the Framingham cohort came in, all the factors seemed to be in place to justify assumption of causality.

The Data

Ancel Keys conducted the Seven Countries Study to prove what he already believed, that fat causes heart disease. In this cross population study, cohorts of men aged 40-59 were chosen in eighteen
areas of seven countries, and surveyed as to diet. Along with dietary recall surveys of all participants, a random sample of families in each geographical area had its meals subjected to direct chemical analysis. Mortality data from existent death certificates were compared with dietary patterns in an initial cross-sectional phase of the study. Subsequently, participants were followed until death, and the cause of their death recorded. The results were taken to show an association between dietary fat (and saturated fat) and mortality from heart disease. This study was seminal in the anti-fat movement.

In 1961, the famous Framingham Heart Study also reported an association between saturated fat intake and cardiovascular disease. Subsequently, other cohort studies were performed, and their outcomes were taken as supporting these results.

The Framingham study had additionally reported that high dietary intake of fat, and of saturated fat, was associated with high levels of serum cholesterol. This led directly to the cholesterol hypothesis of heart disease. Subsequently, a randomized trial, the NIH-sponsored Lipid Research Clinics Coronary Primary Prevention Trial, seemed to find that the cholesterol-lowering drug cholestyramine significantly reduced mortality. (This conclusion has since been challenged.) Based on these results, the NIH, along with the American Heart Association, made a questionable logical leap: assuming that if a cholesterol-lowering drug could reduce mortality, limiting dietary fat and saturated fat should do so as well.

Note the problematic nature of the supporting evidence for this conclusion: (a) A non-causal association had been found between total serum cholesterol and heart disease. However, we now know that breakdown into HDL, etc., is necessary to determine actual association. (More on this below.) (b) A non-causal association had been found between serum cholesterol and dietary intake of fat, saturated fat and cholesterol. However, we now know that while dietary cholesterol intake is associated with saturated fat intake, it does not affect serum cholesterol, except at extremes. We also know that dietary fat, per se, does not raise total cholesterol. (Some forms of fat, such as mono-unsaturated fat, may lower it.) (c) A causal (though statistically very tiny) reduction in mortality via use of the cholesterol-lowering drug cholestyramine had been demonstrated. However, drugs have numerous effects, and so do dietary constituents and drawing a conclusion about the effects of one based on the effects of the other is
inappropriate. Nonetheless, the NIH and the AHA did draw a conclusion, and initiated the hypothesis about dietary fat that now saturates (so to speak) our consciousness.

**Problems with the Data**

The Seven Countries study suffered from the inherent limitations of an ecologic design, as well as the fact that within-country population samples were chosen by convenience rather than randomization. Furthermore, while the predicted correlations were seen in six of the seven countries surveyed, no dose dependent effect of fat or saturated fat intake was seen.

Because Framingham was a cohort study, its results were inherently more reliable than the Seven Countries study (and validity was assumed to extend beyond the township.) However, confounding by interrelationship between studied variables (intake of fat, saturated fat and total calories, as well as cigarette smoking) could not be factored out.

Also, the biological plausibility needed to infer causation from association has faded with increasing knowledge. We now know that the cholesterol and fat in sclerotic vessels is produced locally via a type of inflammatory process, and that serum lipids play an irritant, not a depository role.

Furthermore, as already alluded to, levels of total cholesterol are no longer the point at issue. Rather, we are now concerned about ratios of HDL to LDL, as well as levels of triglycerides. Though it is now known to be true that reduced intake of (some forms of) saturated fat causally decreases total serum cholesterol as well as LDL cholesterol, it is also now known that when saturated fat is replaced by carbohydrate HDL levels are simultaneously reduced and triglycerides are raised. Thus, the net effect is neutral, or even negative.

Worse still, there may not even be any association between high saturated fat intake and heart disease. The numerous within-population ecologic studies as well as cohort and case control studies published in the last three decades have shown inconsistent results at best; in fact, according to some experts, the epidemiologic evidence is now significantly more negative than positive. If current data suggest anything at all about fat consumption, it is not that one should decrease either total fat intake or saturated fat, but rather that one should increase monounsaturated fat intake. But a type of circular
reasoning is widely employed; those studies finding an association are considered valid while the others are explained away. This fallacy of “presuming the conclusion” can be seen clearly in the so-called “French paradox.” As is well known, the French consume more saturated fat per person than Americans but suffer lower rates of CVD.  

(These data are derived from diet recall surveys and overall food consumption statistics on the one hand, and death certificates on the other.) Inventive explanations for this inconsistency have been found in wine polyphenols, a supposed general protective effect of alcohol and measurements of serving size (and total calories.) But the French findings are only a paradox if one assumes the conclusion; otherwise, they are a counterexample.

There are other counterexamples too. For example, in an RCT, the Women’s Health Initiative, reduction of total fat intake along with an increase in fruit and vegetable intake, did not have a significant effect on cardiovascular disease.  

Reaching for straws, proponents of fat reduction often state that, at the very least, reducing fat aids in weight reduction. In support of this, energy-density, time-energy displacement, caloric regulation and other theories are often cited. Yet, numerous ad libitum and metabolic ward studies have failed to find any difference in caloric consumption based on the relative proportion of fats, proteins and carbohydrates in foods, and over periods of up to a year, low-carb, high-protein and/or high fat diets have proven at least as effective, if not more effective, than the standard low-fat approach.

**The Growth and Harms of an Unfounded Public Policy**

By the late 1970s, an immense public health campaign had been undertaken to demonize fat and saturated fat. Organizations involved in this campaign included the CDC, the NIH, the AHA, the National Cholesterol Education Foundation (NCEP), the FDA (through food labeling) and the USDA (via the food pyramid). Many aspects of the food industry enthusiastically joined in, marketing an enormous variety of low fat foods. This campaign was aided by an emotional phenomenon that can be denoted “Health Puritanism,” the instinctive notion that if one likes to eat something it must be bad for you.
The anti-fat policy has been highly successful, at least superficially. According to data from NHANES I, II and III, over the thirty year period subsequent to 1970, average percent caloric intake from total fat and saturated fat decreased."17 Unfortunately, over the same time period, average caloric intake has also increased, and so has obesity. Heart disease has fallen over the same interval, but reductions in cigarette smoking and improvements in blood pressure control are likely supervening causes. It is, in fact, possible (if hypothetical) that the success of the policy has acted to increase heart disease rates: The recommended low fat diet is also a high carbohydrate diet, and high-carb diets might very well promote metabolic syndrome.18 Whether or not this is true, the rise in average caloric intake during the period of fat reduction has definitely been due to a rise in carbohydrate consumption.19

The anti-fat crusade also damaged the beef industry, after the earlier anti-cholesterol error savaged the egg industry, and the anti-salt crusade made millions eat bland food. The campaign has been so successful that it is difficult not to think of “red meat” as a vice, and therefore that to dislocate this portion of agriculture is no more ethically problematic than to impair sales of tobacco or liquor. But logical sense fails to support the emotion. Consider this: After broiling, the a top sirloin steak is about two-thirds protein and one-third fat; half of the fat is of the monounsaturated variety, and of the remainder, one-third is stearic acid, a type of saturated known to have no effect (or a beneficial effect) on cholesterol profile.20,21 There is not, and has never been any reason to demonize red meat – even assuming the saturated fat hypothesis were true.

Another unfortunate result of the anti-saturated-fat campaign was that it led directly to the popularization of margarine; trans-fats have far more plausible evidence for negative health effects than saturated fats ever did. In addition, if one sets aside the skewing perspective of Health Puritanism, it no longer seems a side-effect free intervention to shame people for eating foods they like. Finally, widespread dissemination of unfounded advice by public health authorities, continuing to this day, will ultimately damage the reputation of Public Health in general, a loss of reputation that might be felt keenly when a time comes that the public is asked to trust a correct recommendation.
REFERENCES

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