On Trial: Saturated Fat:
Proven Villain or Medical Myth?

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". . . for the great enemy of the truth is very often not the lie–deliberate, contrived, and dishonest–but the myth–persistent, persuasive, and unrealistic. Too often we hold fast to the clichés of our forebears. We subject all facts to a prefabricated set of interpretations. We enjoy the comfort of opinion without the discomfort of thought."

John F. Kennedy,
Commencement Address at Yale University,
1962

OPENING STATEMENT

It has been almost 40 years since the Senate Select Committee on Nutrition and Human Needs published its initial Dietary Goals for the United States in 1977. That mandate encouraged Americans to decrease consumption of total cholesterol and saturated fat, while increasing carbohydrate content to 55-60% of daily energy (caloric) intake.[1] The U.S. Department of Agriculture (USDA) reinforced this inherited dietary policy in its 1980 Dietary Guidelines for Americans, and in subsequent iterations.[2]

In response to these governmental directives to decrease consumption of cholesterol and saturated fat, the majority of Americans shifted their purchasing patterns in hopes of reducing the risk of coronary heart disease (CHD). The ever-innovative food industry followed suit by modifying its products to appease the newly "fat-phobic" American public, with a litany of 'low fat' and 'fat free' products. Our thinking at the time was quite simple—fat is bad for us, thus anything that is devoid of fat must be healthy. This mindset paid little regard to the growing consumption of processed foods and the artificial ingredients found in the vast majority of them.

This shift raises the concern that if Americans are not eating fat, they must be eating more of something else. And since fat tends to provide food with taste, sugar has quickly become the quintessential 'fat-free' additive to bolster palatable taste to new (and quite addicting) heights—with the added benefits of new food textures, longer shelf life, and improved portability.

Since 1971 the incidence of obesity has more than tripled–from 31 million people in 1971 to 111 million people in 2010. Even more sobering is the fact that 68.5% of Americans are currently either overweight or obese, including 31.8% of our children and adolescents.[3] Paralleling this trend is our incidence of insulin resistance and diabetes, which has more than quintupled from 4.2 million people in 1970 to 21.1 million people in 2010. This growth shows no sign of slowing, and is predicted to rise to 1 in 3 Americans being diabetic by the year 2050.[4] Looming above all these data is the fact that cardiovascular disease continues to reign as the most common cause of mortality in the United States.

Is it possible that the low-fat dietary directives and subsequent nutritional council recommendations provided to millions of patients for the past 40 years have been . . . wrong?

PRESENTATION OF THE EVIDENCE

EXHIBIT A: HISTORICAL PERSPECTIVE

Ever since the term atherosclerosis was adopted to describe the mysterious accumulation of waxy plaque found on the walls of arteries in 1904, scientists have worked to find its cause. Russian researcher Nikolai Anichkov demonstrated in 1913 that although feeding rabbits pure cholesterol (or cholesterin as it was called back then) induced arterial atherosclerosis, that outcome could not be replicated in animals that were not natural herbivores.[5] Although he did not experiment on humans, he is credited with laying the groundwork for what would eventually become the lipid hypothesis, which simply states that elevated cholesterol in the blood causes heart disease.

Efforts to expand Anichkov's research remained mostly dormant until post-World War II, when an American physiologist and nutritional scientist named Ancel Keys noticed that mortality from heart disease had decreased in zones where food rationing was in

place—while elsewhere in the industrialized world death rates from heart disease were increasing. He explored a potential diet-heart relationship prospectively in a cohort of middle-aged men and found that the higher their total serum cholesterol, the more likely they were to die from heart disease.[6]

Subsequently turning his focus to the causes of a rise in total serum cholesterol, Keys used data from the late 1940s on food intake and mortality to plot national fat intake against mortality rate from CHD for six countries (USA, Canada, England, Italy, Australia, and Japan). This simplistic but sobering graph displayed a nearly perfect upward curve, demonstrating that the more fat a country consumed, the higher its incidence of heart disease. This became the start of Keys' legacy, the formulation of the diet-heart hypothesis, which states that elevated intake of saturated fat causes high blood cholesterol, which causes heart disease.

His work was criticized and disregarded when he presented his graph at the 1955 conference of the World Health Organization (WHO) without a body of evidence to back up its correlation. His data were also challenged and ridiculed meticulously by a statistician and a New York State Commissioner of Health in a 1957 paper entitled Fat in the diet and mortality from heart disease: a methodological note.[7] They discovered that Keys actually had sufficient data for twenty-two countries that greatly impaired the trend found in his initially impressive six country analysis. In fact, by cherry-picking the data as Keys had done, they proved that the higher the saturated fat consumption, the lower the mortality rate of that country. They further noted that the dietary data, which came from the Food and Agriculture Organization (FAO), showed only how much food was available for consumption in each country, not how much food was actually consumed. Despite this damning critique, the American Heart Association published a 4-page report in 1961 that included Keys' recommendation that "the best scientific evidence of the time" strongly suggests that Americans would reduce their risk of heart disease by reducing saturated fat (primarily from milk and meat) in their diets. In 1967, Keys made the cover of TIME magazine and told TIME that the ideal heart-healthy diet should be almost 70% carbohydrate and only 15% fat.[8]

In the wake of the critique of his six country analysis, in 1958 Ancel Keys launched his famous observational study, the Seven Countries Study, which was published in 1980. Although the study showed a relationship of sorts between serum cholesterol and heart disease, the pattern did not appear to surface until a fairly high threshold. Keys summarized the findings as demonstrating that "at blood serum levels below 220 mg/dL or so, cholesterol is not a significant factor" for heart disease. Moreover, there was no correlation between myocardial infarction and diet within the countries he studied.[9]

EXHIBIT B: CONTRADICTORY EVIDENCE, PRIOR TO 1989 CONSENSUS

Although the work of Ancel Keys laid the foundation of our low-fat culture, there was (and continues to be) a plethora of data that challenge his observational data. Unpublished data from the Framingham Heart Study in 1968 found no association with the amount or type of fat consumed when comparing the diets of men with total serum cholesterols of >300 mg/dL to those with levels <170 mg/dL.10 Various epidemiologic evidence, including studies of Benedictine and Trappist monks, Navajo Indians, Irish immigrants to Boston, Swiss Alpine farmers, Masai, and other African pastoralists, reported no association between dietary saturated fat and heart disease.[11-14] The 1958 Western Electric Study found that not only was there no association between saturated fat intake and heart disease at 4-year follow up, but there were more coronary events in the low-fat intake group than in those with higher fat intake. In a 24-year follow up to this study in 1981, the authors found that the "amount of saturated fatty acids in the diet was not significantly associated with the risk of death from CHD."[15]

In the 1970s, the ability to fractionate blood cholesterol provided new lipid markers including triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C), from which low-density lipoprotein cholesterol (LDL-C) could be calculated. The National Institute of Health (NIH) funded studies to investigate these new lipid markers via five trials which came to the following conclusions:[16,17]

- High LDL-C is a "marginal risk factor"
- Low HDL-C is a 4-fold better predictor of risk than LDL-C and is the only reliable predictor of risk for men or women over the age of 50
- Consumption of saturated fat raises HDL-C
- Carbohydrates (specifically sugars and highly refined grains) lower HDL-C

- Saturated fat and total fat intake are negatively associated with the risk of myocardial infarction and are positively associated with longevity in the Framingham, Honolulu, and Puerto Rico cohorts.

The authors concluded that that which raises HDL-C should be considered to reduce the risk of cardiovascular disease.[18,19] Regardless of the surprising influence of HDL-C, these results became overshadowed by the subsequent findings of the Multiple Risk Factor Intervention Trial (MRFIT) in 1982 and the Lipid Research Clinics Primary Prevention Trial (LRCPPT) in 1984. The former trial randomized men with a high risk for CHD into a control group that was provided no advice vs. a group that was given medication for hypertension where appropriate, counseling on smoking cessation, and dietary advice to reduce saturated fat and cholesterol intake. After 7 years, the mortality rates of control and intervention groups were nearly identical.[20]

Subsequently, the LRCPPT randomized subjects into a control group that was committed to a low saturated fat diet, and an experimental group that was committed to a low saturated fat diet and placed on a bile acid sequestrant (Cholestyramine, the first anti-cholesterol agent on the market). The intervention group saw reductions in total serum cholesterol of 12.4% and LDL-C of 20.3%, with decreased mortality at 10 years.[21] Although the authors recognized the trial was not designed to assess directly whether lowering cholesterol by diet prevents CHD, *TIME* magazine erroneously reported "Sorry, it's true. Cholesterol really is a killer."[22] This was a drug trial and not a diet trial, yet Basil Rifkind, the NIH director of the trial, was quoted in *TIME* magazine stating, "It is now indisputable that lowering cholesterol with diet and drugs can actually cut the risk of developing heart disease and having a heart attack."[22]

With this final tipping point, the 1984 NIH consensus conference concluded that there is "no doubt" that a low-fat diet "will afford significant protection against coronary heart disease" to every American over the age of two.[23] This recommendation was fortified by the 1989 National Academy of Sciences report on Diet and Health, which stated that the "highest priority is given to reducing fat intake, because the scientific evidence concerning dietary fats and other lipids and human health is strongest and the likely impact on public health the greatest."[24]

EXHIBIT C: CONTRADICTORY EVIDENCE, POST-CONSENSUS (1990 ONWARD)

The Lifestyle Heart Trial in 1990 showed that the combination of smoking cessation, a low-fat (10% maximum of daily calories) vegetarian diet, no flour or sugar, stress management, and vigorous exercise showed regression of coronary atherosclerosis by 20% in one year based on pre- and post-study cardiac catheterization.[25] The principle author of this study, being a vegetarian and professionally known for his low-fat dietary bias, erroneously attributed the coronary plaque regression to the adoption of a very low-fat diet. However, there were so many other confounding variables in the experimental arm that one should more objectively conclude that coronary plaque regression may occur when subjects stop smoking, eliminate white flour and sugar, adopt a low-fat diet, exercise vigorously, and practice stress management. Patients in this study were basically adopting a vegetarian lifestyle with consumption of whole grains and a large amount of vegetables. Given the robust amount of existing data, it must be recognized that the elimination of white flour[26-29] and sugar,[30-32] and regular exercise[33] could just as easily have been the reason for plaque regression. Of course, the study could not adequately conclude which of the interventions was most prominent in causing regression of atherosclerosis.

The 2006 Women's Health study concluded that the low-fat dietary pattern did not reduce risk of CHD or stroke, did not result in a statistically significant reduction in risk of invasive breast cancer or colorectal cancer, and did not reduce the risk of developing diabetes.[34-37]

Two Cochrane Database meta-analyses dated 2001 and 2006 found that a low-fat diet had no effect on longevity and "no significant effect on cardiovascular events,"[38] or "no effect on mortality."[39] Following a 2010 meta-analysis showing no association between saturated fat consumption and CHD, the preponderance of data led the FAO/WHO to write an expert consultation background paper stating, "The experts agreed with the evidence summarized in two recent reports that there is no probable or convincing evidence for significant effects of total dietary fats on coronary heart disease or cancers."[40] Since then, another meta-analysis in March 2014 revealed that the current evidence does not support current cardiovascular guidelines that encourage low consumption of total saturated fats.[41]

EXHIBIT D: EFFECT OF DIETARY THERAPY ON LIPID MARKERS

The low saturated fat diet gained immense popularity over the past half-century due to its effect of

lowering total serum cholesterol and LDL-C , despite its often overlooked tendency to raise triglycerides (TG)[42,43] and lower HDL-C.[44,45] Although elevated LDL-C is a major cause of atherosclerosis,[46] our increasing prevalence of obesity and insulin resistance has led to a greater appreciation of atherogenic and diabetic dyslipidemia[47]—the triad of elevated TG, elevated small-dense LDL-C, and low HDL-C —which heightens risk for CHD.[48,49] This dyslipidemia pattern almost always has TG concentrations that are greater than 150 mg/dL, and often exceed 200 mg/dL.50 When TG is >100 mg/dL, the LDL particle number (LDL-P) and/or apolipoprotein B (apoB), and not LDL-C, are more accurately correlated with cardiovascular events.[51,52] This discordance between LDL-C and LDL-P (or apoB) and our understanding of atherogenic and diabetic dyslipidemia, makes historical data regarding saturated fat difficult to place into a modern context.

When compared to the low-fat diet and alternative popular diets, it is the low-carbohydrate dietary strategy that has led to greater weight loss,[53,54] significant increases in HDL-C with concomitant decreases in TG,[53,54] and noteworthy reductions in hemoglobin A1c.[54] Such findings should not be surprising as such results were evident in the data on the much earlier NIH-funded Framingham cohorts which demonstrated that the subjects who ate the most fat and least amount of refined grains and sugars experienced the greatest increase in HDL-C.[16-19]

Although the data on dietary intervention and more advanced lipid makers are scanty, there are a few short-term studies available. In one study, a low-fat (24% of calories from fat)/high-carbohydrate diet led to significantly higher levels of TG and small LDL particle concentrations as well as lower HDL-C levels when compared to a high-fat (46% of calories from fat)/low-carbohydrate diet[55]—with another study showing that dietary carbohydrates are the principal driver of atherogenic dyslipidemia.[56] When compared to a low fat diet, a high saturated fat diet (50% daily calories) led to a significant reduction in TG and VLDL triglycerides (with a decrease in VLDL size) with an increase in LDL particle size without an increase in LDL-C or LDL-P—all signs of improvement in insulin resistance.[57] Such findings are further corroborated when insulin sensitivity is taken into account.[58] These data should evoke at least some skepticism as to the effectiveness of the low-fat dietary strategy.

CLOSING STATEMENTS

The observational epidemiologic studies that have driven the diet-heart hypothesis have been riddled with conflicting information through the decades, causing much confusion and even requiring the Consensus Conference in 1984 to reach a verdict regarding the inconsistent data. The persistent, guideline-backed conviction that fat is harmful stems mostly from historical, observational epidemiologic data of populations that consumed little fat and (in some cases) had a lower incidence of CHD. In those studies, however, and in many subsequent ones, several other dietary components, including refined grain and sugar, were not accounted for. Nevertheless, since medication-based interventions for secondary prevention began in 1984, there has been strong and unarguable evidence that anti-cholesterol agents are efficacious. Unfortunately, diet and drug interventions are distinctly different and independent interventions, and the role of one cannot be uncritically extrapolated to presume the role of the other.

THIS AUTHOR'S VERDICT

The perceived association between consumption of saturated fat and the predilection for CHD is so ingrained in our culture and medical practice that the relationship has become sacrosanct. However despite guidelines from the USDA and AHA that support this association, we cannot ignore the growing prevalence of insulin resistance and metabolic syndrome following decades of policy-driven dietary directives that have promoted a low-fat, high carbohydrate diet. The health outcomes of our historical and conventional dietary guidelines as well as more recent data appear to favor the consumption of moderate amounts of saturated fat. This is especially true if the alternative is a low-fat diet in which saturated fat is not replaced by vegetables, fruits, and whole grains, but rather by refined carbohydrates and processed foods with added sugars that are permitted to use the misguided label of "fat free" and "low fat."

Our experience with "low-fat" diets, CHD, obesity, insulin resistance, and metabolic syndrome underscores the fact that the influence of a particular macronutrient on the risk of CHD cannot be completely explained by one macronutrient alone. Reductionism as an approach is a tool used by opportunists in nutritional science, and is a prevalent bane of the discipline; it confuses a public that seeks only to learn what constitutes healthy eating. Nutritional

science has become so wide-ranging, that we must abandon the simplistic view that all food items fall into the confining macronutrient categories of carbohydrate, fat, and protein. After all, saturated fat can include processed grain-based desserts as well as natural sources such as butter, milk, eggs, and unprocessed meats—whereas carbohydrates can include white flour and candy, as well as fresh vegetables and fruit.

The conventional wisdom that the universal diet for primary and secondary prevention of cardiovascular disease should be a low-saturated-fat, low-cholesterol diet is not supported by the preponderance of data found in clinical trials. Moreover, the data are clear that there is no one universal diet for all humankind, and dietary strategies must account for individual and cultural variability. Those with insulin resistance may flourish with a low-carbohydrate dietary strategy, whereas the insulin sensitive individual who is without metabolic derangement may thrive with a low-fat diet that is rich with whole grain carbohydrates and an abundance of fresh vegetables and fruits. Greater attention must be given to dietary patterns that focus on natural, minimally-processed foods that are mostly plant based, high in dietary fiber, and low (if any) added sugar—instead of demonizing one macronutrient and/or food additive.

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