The Clintons: Chelsea (Pregnant), Hillary (Unhealthy), and Bill (Healthy)

The Clinton family has been in the spotlight for decades, and much illumination has been on their health and diets. High-profile medical doctors have prescribed treatments for them ranging from heart surgery to vegan food plans, with outcomes visible to the public. The population's interpretation of their behaviors has worldwide impact, including influencing healthcare delivery, food buying habits, and the results of local and national US elections. Money sways politics and laws. Agribusinesses, including the meat, dairy, and egg industries, contribute more than $250 million annually to candidates and for lobbying efforts. Recent events surrounding the Clintons' health and diet have the potential for world change. PAGE 2

Clearing Up The Confusion Surrounding Saturated Fat
By Travis

In the February, May and August 2013, and February 2014 McDougall newsletters, I presented readers with articles addressing the dangers of low-carb and Paleo diets. Please take this opportunity to read these articles.

The ever continuing confusion surrounding diet and health escalated again recently with several reports downplaying the dangers of foods rich in saturated fat. These reports have ignored a large body of relevant evidence accumulated over a century from clinical, population and animal studies demonstrating the dangers of foods rich in saturated fat. These reports only benefit those who profit from promoting such foods, and have the potential to harm the average uniformed individual.

In this article I address the research which has been used to downplay the dangers of saturated fat, and summarize the relevant lines of evidence that have gone largely ignored. I demonstrate that the same research cited to downplay the dangers of saturated fat actually provides strong evidence that saturated fat increases the risk of death from heart disease, the number one cause of death in the world. In addition, I address the evidence of the adverse effects that low-carb, high-saturated fat diets have on heart health. PAGE 5

Featured Recipes

- Southwest Stew
- Lentil and Rice Loaf
- Creamy Mushroom Gravy
- Quinoa Curry Bowl
- Blueberry Muffins

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Evaluation of Chowdhury Meta-Analysis on the Association of Fatty Acids with Coronary Risk, Part 2

This is part 2 of my review of the Chowdhury meta-analysis study [1] that was published online at the Annals of Internal Medicine on March 18, 2014. The study effectively said that the current guidelines on saturated fatty acid (SFA) intake (<10% of calories from SFA) were not justified by the evidence and should be reconsidered. That led Mark Bittman, the NY Times Magazine's lead food columnist to write, Butter is Back, and said, "Julia Child, goddess of fat, is beaming somewhere. Butter is back, and when you're looking for a few chunks of pork for a stew, you can resume searching for the best pieces — the ones with the most fat."

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The Clintons: Chelsea (Pregnant), Hillary (Unhealthy), and Bill (Healthy)

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sways politics and laws. Agribusinesses, including the meat, dairy, and egg industries, contribute more than $250 million annually to candidates and for lobbying efforts. Recent events surrounding the Clintons' health and diet have the potential for world change.

Chelsea Clinton, at age 34, announced her pregnancy on Thursday April 17, 2014. Her personal diet since childhood has been vegan (a diet without meat, poultry, fish, and dairy), or nearly so. Chelsea's wedding on July 31, 2011 was well publicized as "vegan"; however, she says that since age 29 she has added meat back to her diet.

A celebrity pregnancy of this prominence should be making "nutrition and pregnancy" headlines worldwide, causing medical and dietetic professionals to ask, "Where should pregnant women get their protein, calcium, omega-3 fats, and other essential nutrients?" This question would lead them to discover that human history, backed by 100 years of consistent scientific research, provides convincing evidence of exactly where a pregnant woman should get her nutrients: from a diet based on starches, vegetables, and fruits, which happens to be a diet free of animal foods.

This type of diet has been shown to offer the best health outcomes for both mother and baby. By following what a century of science points to as ideal human nutrition during our productive years, we could eliminate the epidemic sufferings caused by obesity, gestational diabetes, and hypertension (eclampsia), and could substantially reduce birth defects and Cesarean births.

As Hillary Clinton considers another run for the presidency, few topics receive more scrutiny than her health; and her personal appearance often emerges as a topic of conversation. One observation has been, "She looks to have gained a significant amount of weight since 2008. She seems pale, tired, and yes, aged." Obviously the demands from her career have taken their toll, but some of the responsibility for her failed health must be laid at the feet of her medical advisors.

That credit primarily goes to Mark Hyman, MD. Their personal and professional relationship has been ongoing for as long as nine years, during which time her health and appearance have deteriorated. Contrast this with the positive objective changes accomplished by her husband during the past four years.

On April 11, 2014, a New York Timesarticle identified Dr. Hyman as Mr. Clinton's doctor: "Dr. Hyman was charged with helping the former president after a 2004 quadruple bypass surgery." Until this article appeared, I had never heard of Dr. Hyman's involvement in Mr. Clinton's personal health.

Mark Hyman, MD is another low-carb diet promoter in line with the authors of the Atkins Diet, Wheat Belly and Grain Brain. Dr. Hyman states, according to the New York Times, that, "It's hard being a vegan to eat enough good, quality protein and not have too much starch." He believes that saturated fat (meat, dairy, and eggs) is good for people, and he uses the one commonly cited, dairy industry-funded study to support his viewpoint. (See my March 2014 newsletter on "Dr. Lard.") Mark Hyman, MD is without traditional medical credibility and is a "supplement hawker."

Our 42nd president switched to a vegan diet only after his second brush with death at the hands of heart surgeons. He underwent bypass surgery on September 6, 2004, and an angioplasty with two stents placed was performed on February 11, 2010. Fortunately, he became a strict, very low-fat vegan in March of 2010, and as a direct result quickly lost 30 pounds, lowered his cholesterol, and became more vigorous and youthful in appearance.

I, Dr. John McDougall, consider any suggestion in the New York Times article that credits Dr. Hyman for turning Mr. Clinton's health around to be false. Undoubtedly, the person most responsible for his dietary conversion was Dean Ornish, MD (once a well-known medical advisor to Mr. Clinton). Mr. Clinton was influenced further by Caldwell Esselstyn's book, Reverse and

Prevent Heart Disease. I may have had a small influence on our former president's decision to eat healthy through a McDougall newsletter I sent to him in February of 2010. (I say this only because he used a statement that could be attributed to me, "…at Thanksgiving I had one bite of turkey.")

Most concerning is that Dr. Hyman is presently trying to dissuade our statesman from his highly successful vegan diet—the very diet that saved his life. The Times went on in its April 11, 2014 article to report: "One of the first things Dr. Hyman did was to wean Mr. Clinton off his previously prescribed vegan diet." Making dietary recommendations that threaten the life of a former president is indeed a serious matter.

To date, efforts to cause Bill Clinton to eat more meat, dairy, and eggs have been unsuccessful. While appearing on ABC's "Jimmy Kimmel Live" April 3, 2014, Mr. Clinton did not dispute the host's claim that he was a practicing vegan. I do not understand why Mr. Clinton has not set (as of April 30, 2014) the record straight about how he regained his lost health and appearance. Bill Clinton's vegan diet may be viewed as "political suicide," and his motives could theoretically be to protect Hilary Clinton's aspirations for the US presidency in 2016. (Remember, the dairy, beef, pork, and poultry industries speak loudly.)

Publically, Mr. Clinton also says that he has changed his diet in large part because he wants to know his grandchildren. Soon his wish will be fulfilled. Hopefully his wife and daughter, maybe via his model, will also make the connection between a health-promoting vegan diet and living a long, healthy, energetic, (and powerful) life.

Clearing Up The Confusion Surrounding Saturated Fat
By Travis

In 2010, Siri-Tarino and colleagues published a meta-analysis of prospective cohort studies evaluating the association between dietary saturated fat and cardiovascular disease in the American Journal of Clinical Nutrition.1 Based on the results of this meta-analysis, these researchers concluded that there was insufficient evidence from prospective cohort studies to conclude that dietary saturated fat

increases the risk of coronary heart disease. However, a number of prominent diet-heart researchers identified many serious flaws and omissions in this meta-analysis that cast doubt on the validity of these researchers conclusions.2 3 4 5 6

More recently, Chowdhury and colleagues published a separate meta-analysis in the Annals of Internal Medicine, and reached similar conclusions to that of Siri-Tarino and colleagues regarding the association between saturated fat and coronary heart disease.7 Unfortunately, this meta-analysis also failed to sufficiently address a number of important limitations that it shares with the meta-analysis by Siri-Tarino and colleagues. Furthermore, in this meta-analysis, although positively, but not significantly associated in the random-effects model, both dietary and total circulating concentrations of saturated fat were associated with a small, but statistically significant increased risk of coronary heart disease in the fixed effects model (RR=1.04 [95% CI, 1.01, 1.07] and RR=1.13 [95% CI, 1.03-1.25], respectively). These significant findings were however ignored in the conclusions of this study. Nevertheless, the media and proponents of popular Low-Carb and Paleo diets have repeatedly cited these meta-analyses as evidence to support a diet rich in saturated fat.

Saturated Fat and Coronary Heart Disease Mortality

In the editorial to the Siri-Tarino meta-analysis, Jeremiah Stamler noted that saturated fat intake was more strongly associated with fatal than non-fatal incidence of coronary heart disease. Stamler calculated that based on the 11 studies included in thRe meta-analysis which provided estimates specifically for fatal cases, saturated fat was associated with a 32% increased risk of death from coronary heart disease, when weighted by person-years of exposure.2 Siri-Tarino and colleagues noted this concern in a follow-up paper, but instead downplayed these findings by asserting that in their own analysis of only 7 studies, saturated fat intake was associated with only a borderline significant 18% increased risk of death from coronary heart disease, when using the random effects model (RR=1.18 [95% CI 0.99-1.42]).8 Similarly, in the more recent meta-analysis, Chowdhury and colleagues found that in their sub-analysis of only 9 studies, saturated fat intake was associated with a borderline significant 7% increased risk of death from coronary heart disease (RR=1.07 [95% CI, 1.00-1.13]).7

It can be deduced by the estimates and the sample size of these sub-analyses by both Siri-Tarino and Chowdhury, that only the studies that provided estimates specifically only for fatal cases were included. Therefore these sub-analyses excluded studies that provided estimates for fatal cases in additional to that of total incidence of coronary heart disease. In total, 14 prospective cohort studies provided estimates for death from coronary heart disease, of which 3 were not included in the original meta-analysis by Siri-Tarino and colleagues.1 9 10 11 12 13 14 15 16 17 18 19 20 21 22

The exclusion of several studies in these analyses warrants a reanalysis of the studies evaluating the association between saturated fat and the risk of death from coronary heart disease. I therefore performed a meta-analysis including all 14 studies for which estimates were available specifically for death from coronary heart disease. Similar to the methods of Siri-Tarino and colleagues, I chose to compare extreme quantiles of saturated fat intake where possible. However, for the studies which estimates were provided as either a 1% increase of

energy, or as a 1-unit increase, the estimates were transformed to represent roughly a 5% increase in energy from saturated fat, as this was similar to the difference for high vs low quantiles of intake in the other studies included in this meta-analysis. In order to ensure that the methods used for the statistical analysis were consistent with that used by Siri-Tarino and colleagues, I performed the meta-analysis in Review Manager (from The Cochrane Collaboration), and pooled the estimates using the random effects model for both within-study and between-study variation. Similarly, risk ratios and 95% confidence intervals were log transformed to derive the corresponding standard error for beta-coefficients by using Greenland's formula.23 Otherwise, the exact P-value was used where available to derive the corresponding standard error.

In a meta-analysis including 14 studies, dietary saturated fat intake was associated with a statistically highly significant 24% increased risk of death from coronary heart disease (Fig. 1). Similarly, for the 11 studies included in the Siri-Tarino meta-analysis, saturated fat was associated with a statistically highly significant 26% increased risk of death from coronary heart disease (RR=1.26 [95% CI, 1.14-1.40]).

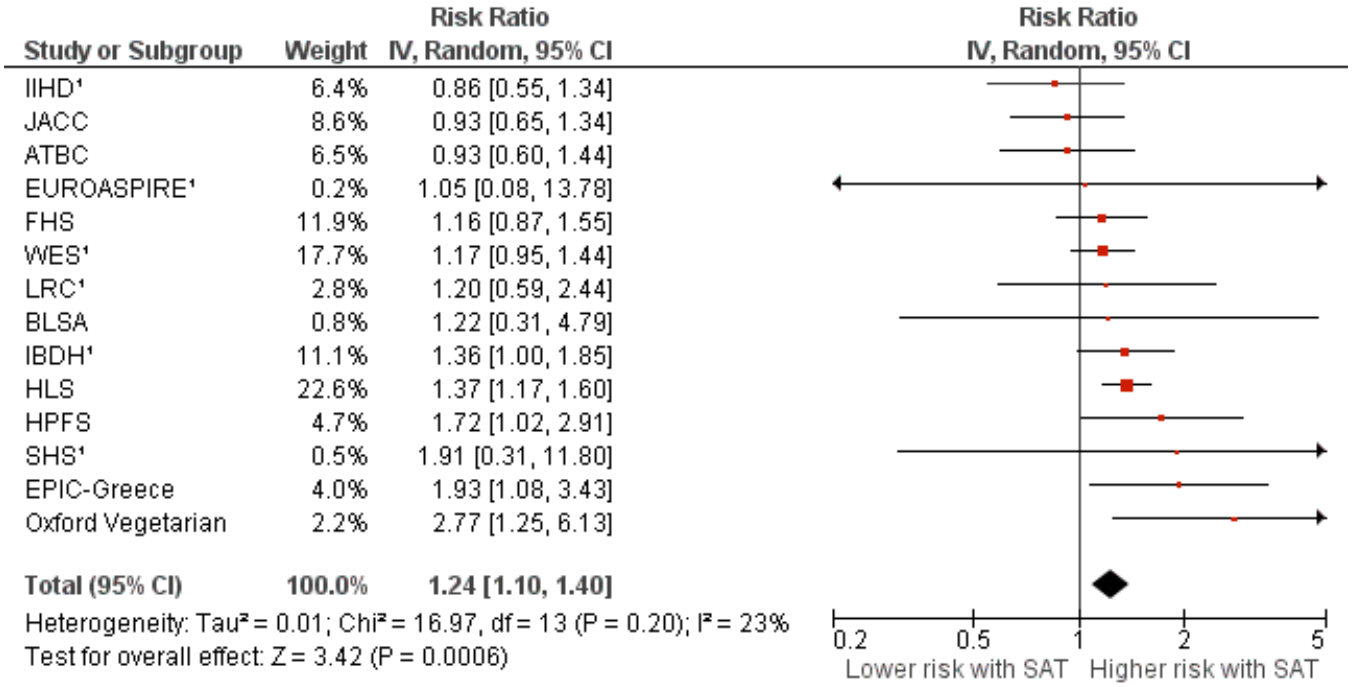


FIGURE 1. Risk ratios and 95% CIs for fully adjusted random-effects models examining associations between saturated fat intake in relation to coronary heart disease mortality. ¹Studies that included adjustments for serum or LDL cholesterol. SAT, saturated fat intake.

It is well established that saturated fat raises serum and LDL cholesterol, and that these blood lipids increase the risk of coronary heart disease.24 25 26 27 However, in this meta-analysis, almost 40% of the weight was derived from studies that controlled for either serum or LDL cholesterol. Therefore, the inclusion of these studies would be expected to have bias these

findings towards null.2 In a sub-analysis excluding the 6 studies that controlled for either serum or LDL cholesterol, saturated fat was associated with a statistically significant 30% increased risk of death from coronary heart disease (Fig. 2). Interestingly, in a sub-analysis including only the 6 studies which controlled for either serum or LDL cholesterol, saturated fat was associated with a statistically significant 18% increased risk of death from coronary heart disease (RR=1.18 [95% CI, 1.01-1.37]). This suggests that the adverse effects of saturated fat may extend beyond on simple measures of blood lipids.

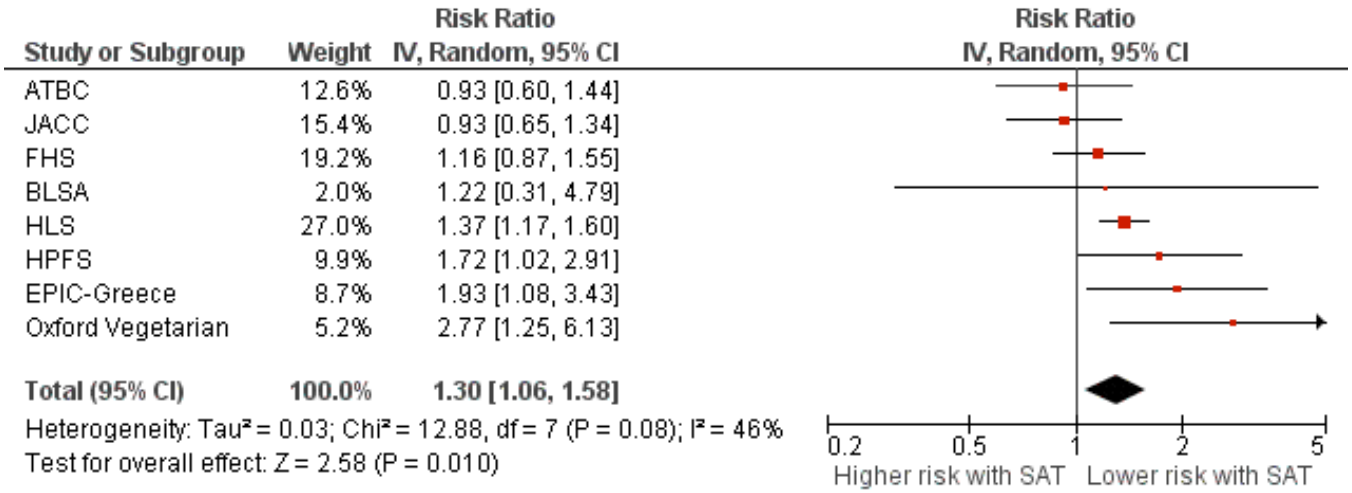


FIGURE 2. Risk ratios and 95% CIs for fully adjusted random-effects models examining associations between saturated fat intake in relation to coronary heart disease mortality. SAT, saturated fat intake.

As coronary heart disease is the leading cause of death in the world, naturally these findings should be a cause for concern.28 Nevertheless, both the Siri-Tarino and Chowdhury meta-analyses are widely cited by proponents of Low-Carb and Paleo diets as providing compelling evidence in favor of a diet rich in saturated fat. It is important to note, however, that in the studies included in this meta-analysis, the difference for high vs low intake of saturated fat only ranged between about 5% and 10% of energy. This suggests that individuals following popular variants of these diets which often emphasize far higher intakes of saturated fat than recommended levels may be at a much greater risk of death.

It is important to note that the influence that saturated fat has on the risk of disease is not primarily determined by intake per se, but by which foods saturated fat is substituted for. As the intake of dietary fiber was universally low among subjects in these studies, this suggests that subjects consuming diets lower in saturated fat were substituting saturated fat primarily with lean animal foods and heavily processed foods.29 As dietary fiber was associated with a decreased risk of death from coronary heart disease in a number of these studies, this suggests that compared to fiber-rich foods, foods rich in saturated fat may be associated with an even stronger risk of coronary heart disease death.29

Although in this meta-analysis, the Israeli Ischemic Heart Disease Study appeared the least favorable of the hypothesis that saturated fat increases the risk of death from coronary heart disease, it should be noted that not only were the estimates controlled for serum cholesterol, in this study, saturated fat as a percentage of fat was actually associated with a statistically significant increased risk of death from coronary heart disease. In addition, subjects who were classified as being most adherent to religious Orthodoxy, which is typically accompanied by fasting periods in which the consumption of meat and other foods rich in saturated animal fat are prohibited, experienced a significantly lower death rate of coronary heart disease.11 This observation is supported by several other studies which found that Orthodox fasting is associated with improved cardiovascular risk factors, including blood lipids.30

The findings from this meta-analysis are in agreement with the demonstrated unequivocal causal relationship between diets rich in cholesterol and saturated fat, and the development of atherosclerosis in nonhuman primates, among dozens of other animal species. It had also been demonstrated that such diets cause heart attacks, and even cardiovascular related deaths in nonhuman primates at a rate similar to that of high-risk populations living in developed nations.31

The findings from this meta-analysis are also in agreement with numerous longitudinal ecological studies. For example, intake of saturated fat explained about 88% of the variance in death from coronary heart disease between the 16 cohorts in the 25-year follow-up of the Seven Countries Study.32 Similar estimates were also found for foods rich in saturated fat, including butter, meat, and animal foods combined.33 Similarly, in 1989, Epstein examined the changes in death from coronary heart disease in 27 countries during the previous 10 to 25 years, and noted that:

> *"In almost all of the countries with major falls or rises in CHD mortality, there are, respectively, corresponding decreases or increases in animal fat consumption..."*

Epstein also noted that a number of other risk factors, such as smoking could not explain these findings, as the prevalence of smoking among women either remained largely unchanged or increased in most nations during this period, yet similar declines in death were often observed in both men and women.34 Epstein's findings are further supported by a number of studies that have incorporated the IMPACT CHD mortality model, which has been shown to adequately explain which risk factors and treatments that have contributed most significantly to the changes of rates of coronary heart disease mortality throughout most parts of the world.35

Dietary Patterns and Coronary Heart Disease Mortality

Dietary patterns characterized by high or low intakes of saturated fat may provide indirect evidence of the effect saturated fat has on the risk of death from coronary heart disease. For example, a recent meta-analysis of prospective cohort studies by Noto and colleagues found that both low-carbohydrate, and low-carbohydrate, high-protein diets, which highly correlated with saturated fat intake were associated with a statistically significant increased risk of death from all causes combined.36 Conversely, the findings for death from cardiovascular disease, although positive, were not statistically significant. However, several of the studies controlled

for saturated intake, and sub-analyses in several of the studies found that the excess risk of death was greater when either saturated fat intake was above the median, or when the diets were classified as being animal based.37 38 Similarly, a sub-analysis in one of the studies found that the association with death was stronger after excluding nonadequate dietary reporters.39

These sub-analyses would likely allow for a clearer interpretation of the effects of a carbohydrate restricted diet rich in saturated fat. Unfortunately, estimates based on these sub-analyses were not provided in the meta-analysis by Noto and colleagues. Therefore, I performed a meta-analysis based on the studies included in the meta-analysis by Noto and colleagues, while using the estimates for the sub-analyses described above where possible.37 38 39 40 41 For the statistical analysis I used the same methods described by Noto and colleagues.36

In this meta-analysis, a high low-carbohydrate score was associated with a statistically significant 15% increased risk of death from cardiovascular disease, for which only 2 of 4 cohorts did not control for saturated fat intake (Fig. 3). On the other hand, a high low-carbohydrate, high-protein score was associated with a statistically significant 100% increased risk of death from cardiovascular disease, for which 3 of 4 studies did not control for saturated fat intake (Fig. 4).

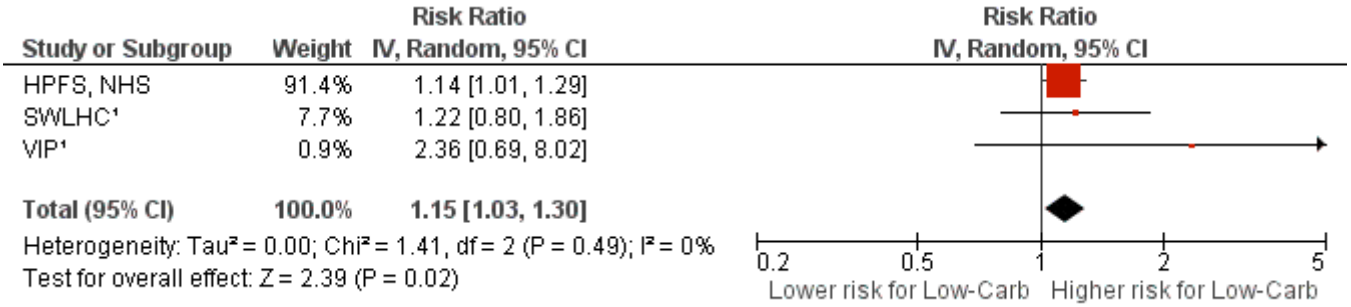


FIGURE 3. Risk ratios and 95% CIs for fully adjusted random-effects models examining associations between low-carbohydrate diets in relation to cardiovascular disease mortality. ¹Studies that included adjustments for saturated fat intake.

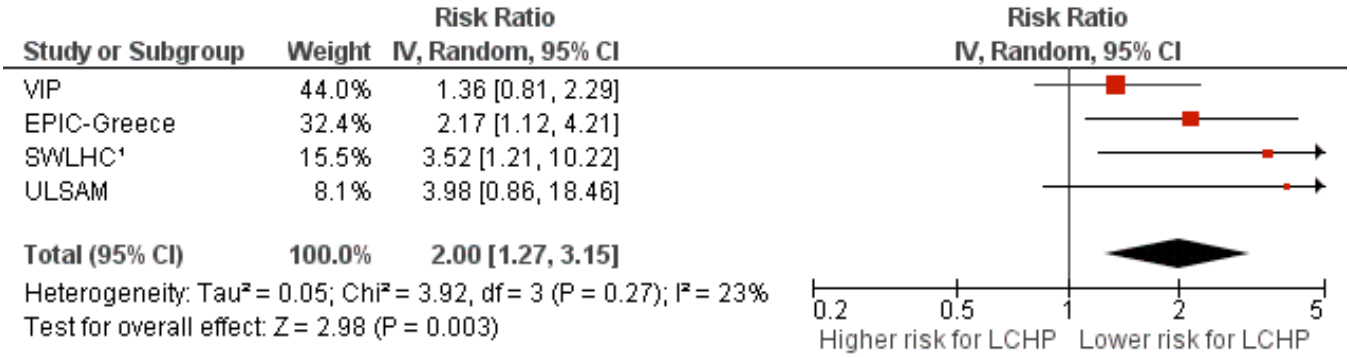


FIGURE 4. Risk ratios and 95% CIs for fully adjusted random-effects models examining associations between low-carbohydrate, high-protein diets in relation to cardiovascular disease mortality. [1]Studies that included adjustments for saturated fat intake. LCHP, low-carbohydrate, high-protein diet.

As coronary heart disease is the number one cause of cardiovascular death in the nations where these studies were carried out, this provides indirect evidence that diets rich in saturated fat, at least in the context of a carbohydrate restricted diet, increases the risk of coronary heart disease. Furthermore, the difference in intake of saturated fat between the low and high low-carbohydrate scores was generally smaller than the difference of intake between popular low-carbohydrate diets and recommended levels, suggesting that individuals who follow more extreme variants of these diets may be at an even greater risk of death. As reviewed previously, these findings may be explained, in part, by a number of adverse effects that carbohydrate restricted diets have been shown to exert on cardiovascular risk factors. For example, recent meta-analyses of randomized controlled trials have found that compared to diets rich in nutrient poor, low-fiber carbohydrates, carbohydrate restricted diets raise LDL cholesterol and impair flow-mediated dilatation.42

Findings from prospective cohort studies comparing vegetarians characterized by consuming moderately low saturated fat diets and health conscious omnivores may provide further indirect evidence of the adverse effects of saturated fat. I showed previously in a meta-analysis of 7 prospective cohort studies that compared to vegetarians, health conscious omnivores experienced a statistically highly significant 32% increased risk of death from coronary heart disease (Fig. 5).43 44 45 46 47

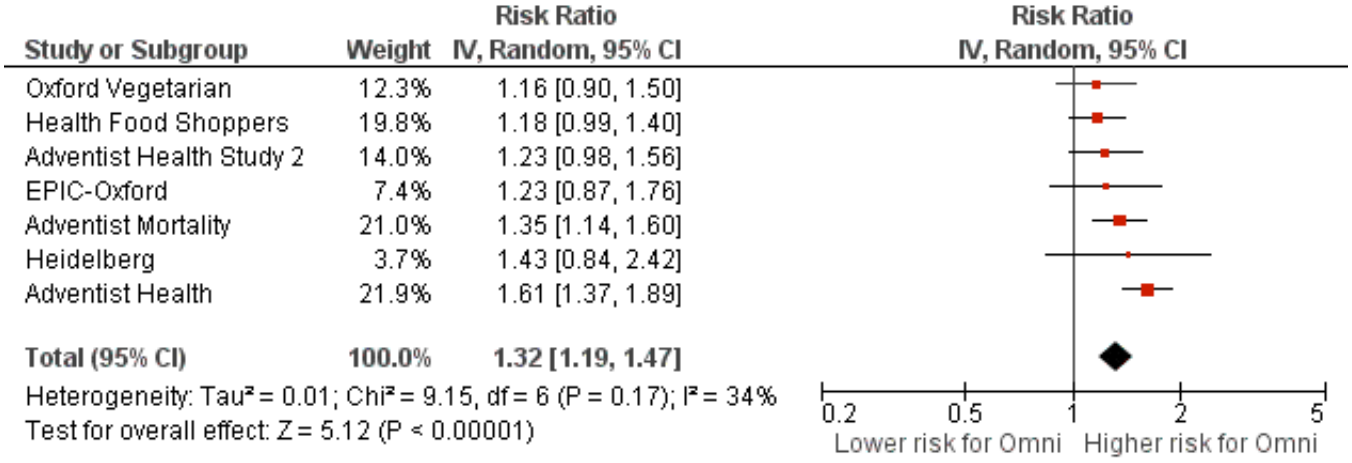


FIGURE 5. Risk ratios and 95% CIs for fully adjusted random-effects models examining associations between omnivorous diets in relation to coronary heart disease mortality.

It is important to note that the omnivores in these studies had a relatively low intake of meat, suggesting that individuals following popular meat based diets may be at a greater risk of death. This suggestion is supported by a recent meta-analysis of prospective cohort studies which found that an increment of 1 mg/day of heme iron, found only in animal tissue is

associated with a 27% increased risk of coronary heart disease.48 As reviewed previously, in these studies, the degree of reduction in risk of death from coronary heart disease observed in vegetarians in these studies was generally in proportion to the expected reduced risk based on the differences in levels of total and non-HDL cholesterol. This suggests that these results may, in part, be explained by differences in intake of saturated fat.

The findings of a pooled-analysis of 11 prospective cohort studies by Jakobsen and colleagues suggested that replacing saturated fat with polyunsaturated fat, but not monounsaturated fat or carbohydrate, was associated with a significantly decreased risk of death from coronary heart disease.49 However, the researchers pointed out that these findings should be interpreted with caution, as the main dietary source of monounsaturated fat in these cohorts was animal fat, whereas the quality of carbohydrate was not considered. In this pooled-analysis, dietary fiber intake was controlled for, essentially removing a primary benefit of replacing foods rich in saturated fat with carbohydrate. In a different pooled-analysis including virtually the same studies, an increment of 10 g/day of dietary fiber was associated with a 27% decreased risk of death from coronary heart disease.23 This suggests that replacing saturated fat with the equivalent energy from fiber-rich carbohydrate would likely be associated with a significantly reduced risk of death from coronary heart disease. This suggestion is supported by a different meta-analysis which found that an increment of about 2 servings a day of whole grains was associated with a 22% decreased risk of death from cardiovascular disease.50 Interestingly, even Siri-Tarino and colleagues concluded in a more recent paper that saturated fat should be replaced with polyunsaturated fat and whole grains in order to reduce the risk of cardiovascular disease.7

Saturated Fat is a Major Problem

The findings reviewed here support the hypothesis that saturated fat increases the risk of coronary heart disease mortality. Furthermore, as reviewed previously, evidence also suggests that the hazardous effects of diets rich in saturated fat are also applicable to diets rich in organic, grass-fed animal foods. However, saturated fat is only one of a number of problems as far as chronic diseases are concerned. The effect that a particular food has on the risk of coronary heart disease cannot be fully explained by saturated fat content alone, but rather by multiple nutrients that likely operate together in a complex manner to modify the risk of disease. Therefore, it may be more appropriate to focus attention on recommending healthy dietary patterns that are naturally low in saturated fat, while rich in dietary fiber and other beneficial nutrients; primarily, minimally processed, plant-based diets. Such a focus may be more effective to help lower the intake of saturated fat, while simultaneously improving overall dietary quality compared to the more contemporary reductionist approach of focusing on modifying single nutrients.

In forthcoming parts of this review, I will examine both the effects of dietary and total circulating concentrations of saturated fat on the risk of total incidence of coronary heart disease. In addition, I will examine a number of other important limitations of the studies included in these meta-analysis that may have bias these findings towards null.2 3 4 5 6

Study acronyms: ATBC, Alpha-Tocopherol Beta Carotene Study; BLSA, Baltimore Longitudinal Study of Aging; EPIC-Greece, European Prospective Investigation into Cancer Greece; EUROASPIRE, European Action on Secondary and Primary Prevention through intervention to reduce events; FHS, Framingham Heart Study; HLS, Health and Lifestyle Survey; HPFS, Health Professionals' Follow-Up Study; IBDH, Ireland-Boston Diet Heart Study; IIHD, Israeli Ischemic Heart Disease Study; JACC, Japan Collaborative Cohort Study; LRC, Lipid Research Clinics; MALMO, Malmo Diet and Cancer Study; NHS, Nurses' Health Study; SHS, Strong Heart Study; SWLHC, Swedish Women's Lifestyle and Health Cohort; ULSAM; Uppsala Longitudinal Study of Adult Men; VIP, Västerbotten Intervention Program; WES, Western Electric Study.

Featured Recipes

Cathy Fisher, is the creator of StraightUpFood.com, a blog that offers recipes and information on how to eat a health-promoting, plant-based (vegan) diet. Cathy's education and experience are grounded in her work with the McDougall Program (2006 to present) and TrueNorth Health Center (2010 to present), where she regularly teaches cooking classes to in-patient clients. Cathy graduated with her BA in Psychology, going on to earn a credential in Early Childhood Education and a certification in Nutrition Education. Cathy enjoys presenting to groups and giving people the practical skills they need to successfully shift to a healthier diet.

Southwest Stew

This chunky, satisfying stew is full of beans, greens, corn, mushrooms, bell pepper, and plenty of herbs and spices to please your taste buds and fill your belly. This recipe makes 12 cups of stew, perfect for leftovers throughout the week!

1 medium yellow onion, chopped
1 medium red bell pepper, chopped
6 medium white or cremini mushrooms, sliced
1 tablespoon chili powder
1 tablespoon dried oregano
2 teaspoons ground cumin
2 teaspoons granulated garlic
4½ cups water
½ to 1 can (6 ounces) tomato paste (see Notes below)
1 can (15 ounces) black beans, drained and rinsed (or 1½ cups)

1 can (15 ounces) pinto beans, drained and rinsed (or 1½ cups)
1 can (15 ounces) diced tomatoes (including juice)
1 can (15 ounces) corn, drained and rinsed (or 1½ cups)
5 cups chopped (into bite-size pieces) collard greens
2 cups loosely packed cilantro leaves (or parsley), coarsely chopped

1. Heat a large soup pot on high heat with 2 tablespoons water in the bottom. When the water begins to sputter, add the onion, bell pepper and mushrooms and cook stirring for 3 minutes, adding water as needed. Add the chili powder, oregano, cumin, and garlic, and cook for an additional 2 minutes, still adding a little water as needed.

2. Add the 4½ cups of water to the pot along with the tomato paste, and stir or whisk to blend in. Add the black beans, pinto beans, diced tomatoes, corn, and collard greens. Reduce heat to medium-low and cook covered for 15 to 20 minutes, stirring once or twice. Stir in the cilantro and serve as is or with ground pumpkin seeds and chopped cilantro (as shown above).

Preparation: 20 minutes
Cooking: 25 minutes
Serves: 8 (makes 12 cups)

Notes:

- Fresh garlic may also be used; substitute with 1½ tablespoons freshly minced (about 6 medium cloves).

- If you can't find collard greens, you may also use kale. If you use chard, add it in closer to the end of cooking since it cooks quickly.

I try to avoid using partial cans of ingredients, but for this recipe I like to use a half a can of tomato paste since it delivers a milder tomato flavor. But feel free to use the whole can if you like a richer, zingier tomato base.

Lentil and Rice Loaf
This flavorful loaf is perfect for anytime you want a hearty main dish. It can be made with fresh or dried herbs. Make two loaves while you're at it and freeze one for leftovers. This is delicious topped with Creamy Mushroom Gravy (below)! (Prepare your ingredients while the rice and lentils are cooking.)

1¾ cups water
½ cup brownish-green lentils
½ cup short-grain brown rice
2 teaspoons poultry seasoning
1 teaspoon granulated onion

1 medium onion, chopped
5 medium white or cremini mushrooms
1 large rib celery, chopped
1 tablespoon freshly minced garlic (about 5 medium cloves)

¾ cup quick rolled oats
1 can (6 ounces) tomato paste
1½ teaspoons freshly minced rosemary (or ½ teaspoon ground/dried)
1½ teaspoons freshly minced thyme leaf (or ½ teaspoon dried)
1 tablespoon minced fresh sage leaf (or 1 teaspoon dried/rubbed sage)
1 cup parsley leaves, chopped
½ cup chopped pecans or walnuts

1. In a medium sauce pan on high heat, combine water, lentils, rice, poultry seasoning, and granulated onion. Bring to a boil, then turn down to simmer and cook covered for 45 minutes. When done cooking, remove from the heat and let stand for 10 minutes with the lid still on.

2. Preheat oven to 350 degrees. Line a standard size loaf pan (9 by 5 by 3-inch) with parchment paper and set aside (or use a silicone loaf pan).

3. In a medium skillet on high heat, add 1 tablespoon of water. When the water begins to sputter, add the chopped onion, mushrooms, and celery, and cook stirring for about 3 minutes, adding water as needed. Add the garlic, and cook stirring for an

additional 2 minutes, until the vegetables have softened, adding water as needed. Remove from heat and set aside.

4. Using a large bowl, combine the oats, tomato paste, rosemary, thyme, sage, parsley, and nuts. When the cooked vegetables, and rice and lentils have cooled for about 10 minutes, add them to the bowl as well, and stir well until all ingredients are thoroughly mixed.

5. Place about half of the mixture into a food processor and pulse 5 to 6 times (so it gets blended slightly, but is still somewhat chunky). Spoon this into the loaf pan. Place the remaining mixture into the food processor and blend in the same way. Add to the loaf pan.

6. Press down firmly and into the corners. Shape the top flat or with a slight rise down the middle. Cover with a piece of aluminum foil and cook for 30 minutes. Remove the foil and cook for another 20 to 25 minutes until the top is browned. Remove and let cool at least 10 minutes before slicing and serving.

Preparation: 55 minutes (including cooking rice and lentils)
Cooking: 50 minutes (baking)
Makes: 1 standard loaf (about 10 ¾-inch slices)

Creamy Mushroom Gravy
This gravy is delicious with mashed potatoes or the Lentil and Rice Loaf. It's rich and flavorful even without soy sauce, flour or cornstarch.

2 cups water
¼ cup raw unsalted cashews (1½ ounces)
6 medium white or brown (cremini) mushrooms (6 ounces)
1½ cups chopped yellow onion (½ of a large onion)
1 small carrot, sliced
1 celery rib, sliced
2 teaspoons freshly minced garlic
1 teaspoon poultry seasoning
freshly ground black pepper

1. Place the water and cashews into a blender, and set aside.

2. In a medium saucepan on high heat, add 1 tablespoon of water, and when it starts to sputter, add the mushrooms, onion, carrot, and celery. Cook stirring for about 3 minutes until the vegetables soften, adding a little water as needed to prevent sticking. Add the garlic and poultry seasoning, and continue stirring for another 2 minutes (adding water as needed).

3. Add the cooked vegetables to the blender of water and cashews, and blend until smooth (add more water if you'd like a smoother consistency). Return the gravy to the saucepan (on medium heat) and stir briefly just to reheat, grinding a bit of black pepper in at the end. Serve immediately.

Preparation: 10 minutes
Cooking: 15 minutes
Makes: about 3 cups

Quinoa Curry Bowl
This quick and easy dish uses "stir fry" frozen vegetables, along with fresh ginger and garlic. Frozen veggies can be a healthy and delicious meal addition. Look for 100% frozen vegetables without any added salt or oil.

1½ cups water
¾ cup uncooked (dry) quinoa
1 teaspoon granulated onion
½ teaspoon curry powder
1 bag (16 ounces) frozen "stir fry" blend vegetables
1 tablespoon minced garlic (4 to 5 medium cloves)
1 teaspoon minced fresh ginger
1 teaspoon curry powder
2 tablespoons tahini (ground sesame paste)
½ of an avocado (optional)
sesame seeds to garnish (optional)

1. Stir the water, quinoa, granulated onion, and ½ teaspoon curry powder together in a medium saucepan, and bring to a boil. Reduce heat to a low simmer and cook covered with a tight-fitting lid for 15 minutes.

2. While the quinoa is cooking, place the frozen vegetables into a skillet or soup pot on medium-low heat covered so the vegetables can thaw and soften. Stir occasionally, adding a little water if needed. While the vegetables are warming, mince the garlic and ginger.

3. When the vegetables have thawed and softened (5 to 10 minutes), increase the heat to medium-high and stir in the garlic, ginger, and 1 teaspoon curry powder, and cook stirring for 1 to 2 minutes. Remove the pan from the heat and add the tahini, adding a little water as needed to mix thoroughly. Stir the quinoa into the vegetables. Serve as is or with diced avocado and/or sesame seeds on top.

Preparation/cooking: 15 minutes
Serves: 2 to 4

Notes:

- I use a "stir fry" blend with this recipe, but any 16-ounce bag of frozen vegetables may be used. Also, feel free to toss in any favorite fresh vegetables, too, such as zucchini, cabbage, greens (kale, chard, spinach), or green onions.

- Quinoa is sold as a dry grain, and comes in white/yellow, red, and black. Use any color you like or a mix. (Two-and-a half cups of cooked rice may also be used in place of the cooked quinoa.)

Blueberry Muffins

Lemon zest and cardamom give these Blueberry Muffins their dreamy flavor and aroma. Oat and millet flours produce a muffin that is hearty and filling, while dates and applesauce lend sweetness and moistness without the use of sugar and oil.

12 Medjool dates, pitted and chopped (about 8 ounces)
1 cup non-dairy milk
1½ cups old-fashioned rolled oats
3/4 cup millet (dry)
2 teaspoons baking powder
½ teaspoon ground cardamom
½ cup applesauce
1 teaspoon lemon zest, packed
1 cup fresh or frozen blueberries (if frozen, do not thaw first)
½ cup coarsely chopped walnuts (optional)

1. Preheat oven to 350. Place the chopped dates and the non-dairy milk into a small bowl and set aside for 15 to 20 minutes (so the dates may soften).

2. Grind the oats and millet into a flour in your blender (a high-speed blender will do a finer job) and place into a bowl. Add the baking powder and cardamom to this and stir with a fork.

3. Place the dates and non-dairy milk into the blender and blend until smooth. Add this date mixture to the bowl of dry ingredients along with the applesauce and lemon zest, and mix with a spoon until all the dry ingredients have disappeared.

4. Gently fold in the blueberries and chopped walnuts. Spoon the batter into a silicone muffin pan or metal muffin pan lined with parchment paper muffins liners, filling each muffin cup about 3/4 full.

5. Bake for 25 to 30 minutes. The muffins will be done when the tops have begun to brown and cracks appear, and when a toothpick inserted comes out pretty clean. (These are hearty muffins, so they will rise but not too much.) Let cool in the pan for at least 15 to 20 minutes before removing.

Preparation: 30 minutes
Cooking: 30 minutes
Makes: 12 muffins

Notes:

- Blueberries tend to sink into the batter during cooking, so I like to push a few blueberries into the top of each muffin just before baking (make sure they're pushed in though, or they will roll off during rising).

- If you've never used cardamom, I urge you to seek some out. Cardamom is wonderfully aromatic; in a pinch you can substitute with 1½ teaspoons cinnamon.

Evaluation of Chowdhury Meta-Analysis on the Association of Fatty Acids with Coronary Risk, Part 2

"There are three kinds of lies: *lies, damned lies and statistics.*"[1]

Fred Pollack
April 29, 2014

This is part 2 of my review of the Chowdhury meta-analysis study [1] that was published online at the Annals of Internal Medicine on March 18, 2014. The study effectively said that the current guidelines on saturated fatty acid (SFA) intake (<10% of calories from SFA) were not justified by the evidence and should be reconsidered. That led Mark Bittman, the NY Times Magazine's lead food columnist to write, Butter is Back, and said, *"Julia Child, goddess of fat, is beaming somewhere. Butter is back, and when you're looking for a few chunks of pork for a stew, you can resume searching for the best pieces — the ones with the most fat."*

About Fred

I live with my wife, Iris, in Miami Beach. We are both 65. I worked at Intel for 23 years, and retired in early 2001. For most of my last 8 years at Intel, I directed the planning for Intel's future microprocessors. In January 1993, I was named an Intel Fellow.

My wife and I have been eating a low-fat whole-food plant-based diet since February 2009.

[1] Attributed by Mark Twain in his autobiography to Disraeli. But the origin is more complex than that. See http://www.york.ac.uk/depts/maths/histstat/lies.htm

From my own extensive reading of the medical/nutritional research, I doubted Chowdhury's conclusion, and thus was compelled to do an in-depth review. And, the only way to do this is to read and analyze all 20 of the SFA intake studies that were used in the Chowdhury meta-analysis. Part 1 of my review, published in last month's McDougall Newsletter, looked at just 2 of the 20 studies - one that showed the most benefit to higher intakes of SFA (MALMO) and one that showed the most harm (Oxford Vegetarian). My analysis of MALMO study described the major flaws, which were pointed out by the authors themselves in the paper that Chowdhury referenced. And, the conclusions in the paper are quite different than what Chowdhury's analysis indicated.

Last month, my expectation was that I could complete and document my analyses of all 20 studies, provide a summary for the newsletter, which would have a link to download my detailed (and, as my wife says, esoteric and boring) analyses of all 20 papers. But there just wasn't enough time. So for that, you will have to wait for part 3, next month.

So what is this part about? Two related topics. One is how one country, Finland, which had the highest coronary heart disease mortality in the world in the early 1970's, reduced their rate by 80% over a 35 year period. The other is about a study in east Finland (part of the Chowdhury et al meta-analysis), the Kuopio Ischaemic Heart Risk Factor (KIHD) study. With the data that I uncovered, the KIHD paper (2005) [3] should not have passed the peer-review process and been published.

When does the sin of omission become a ***lie****?* I will show that there were very relevant blatant omissions in the KIHD paper. Does that make it a ***damned lie****?* When ***statistics*** are applied to give the illusion of truth, we end up with ***"Lies, Damned Lies, and Statistics"***.

A day's of research on the internet by Chowdhury et al should have uncovered the flaws in the KIHD study, and thus, excluded it from their meta-analysis. But of the 20 studies in the Chowdhury meta-analysis, the KIHD study was the only one marked by Chowdhury as "Estimates were available through correspondence with the study authors." The other 19 studies had sufficient data in their published papers for Chowdhury et al to construct a Relative Risk (RR) for increased SFA intake with a 95% confidence interval (CI), but not the KIHD study. They had to communicate with KIHD researchers to get the extra data. And, nothing was said about the flaws? Nothing? Really?

The next section, i.e. the good news, describes what happened in Finland over a 35-year period, which I found fascinating. And, it provides a story of SFA intake and societal change. That will then give me the context to discuss the KIHD study.

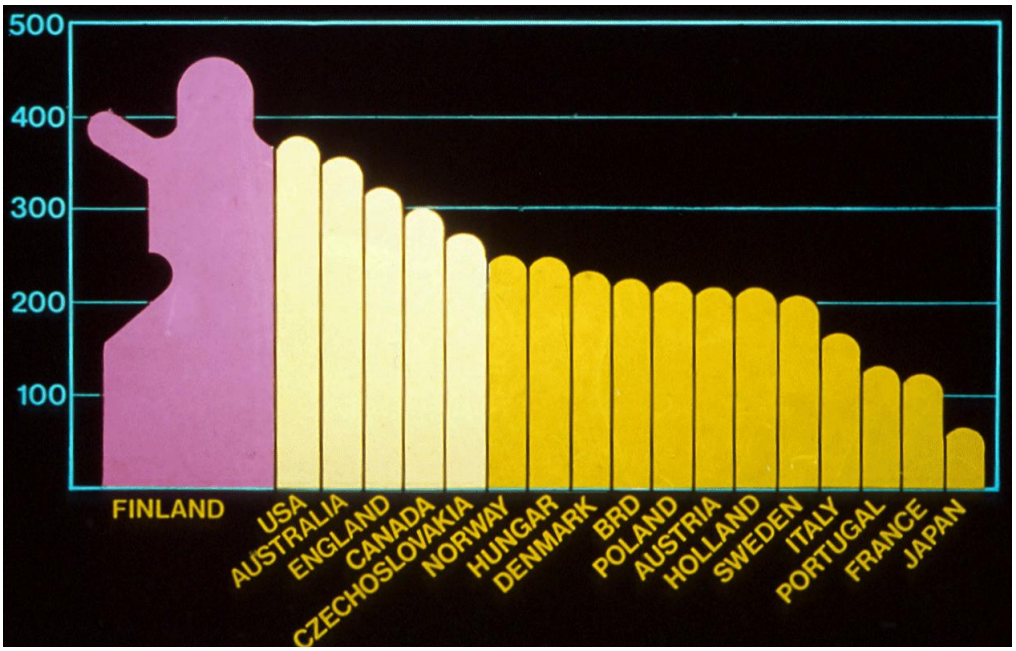
Since this is a long article for a newsletter, at the beginning of each section, I have the key messages. So just read those, if you want a quick read.

Finland (1972 to 2007)

Key Messages:
- In 1973, Finland had a highest country death rate for men from CHD.

- By 2007, the CHD death rate for men dropped by 80% - 3/4 due to a reduction in risk factors.
- The decrease in serum cholesterol was 2/3 of that reduction.
- ***Saturated fat (SFA) intake went from 22% of dietary energy intake to 13%***.
- From 1965 to 2005, butter consumption fell from 40lbs to 7lbs per person per year.

CHD Mortality per 100,000 men in 1973



In Finland, sample data was collected every 5 years, beginning in 1972. Beginning with the 1982 measurement, about 2-3 years after each measurement year, a summary article was often published in a medical journal [6, 7, 8, 9, 10]. The most recent one was published in 2009 [5], "Thirty-five-year trends in cardiovascular risk factors in Finland," and covers the period from 1972 to 2007. My summary draws from that paper, as well as a 2009 presentation [11] from Pekka Puska, Director General of Finland's National Institute for Health and Welfare (THL) and President of the World Health Federation. Below, all quotes are from the paper, and all graphs/charts are from the presentation (unless otherwise noted).

From the above chart of CHD mortality, clearly Finland had a problem in the early 1970's. The first risk factor surveys were carried out in East Finland (North Karelia and Kuopio provinces). "In 1972, a comprehensive community-based intervention programme, as a national pilot, was launched in North Karelia to reduce the burden of exceptionally high CHD morbidity and

mortality. . . .After the initial 5-year period of the North Karelia Project (1972–1977), national preventive activities were gradually implemented."

"A remarkable decline in serum cholesterol levels was observed between 1972 and 2007. Blood pressure declined among both men and women until 2002 but leveled off during the last 5 years. Prevalence of smoking decreased among men. . . .Body mass index (BMI) has continuously increased among men. . . .*Risk factor changes explained a 60% reduction in coronary mortality in middle-aged men while the observed reduction was* ***80%.***"

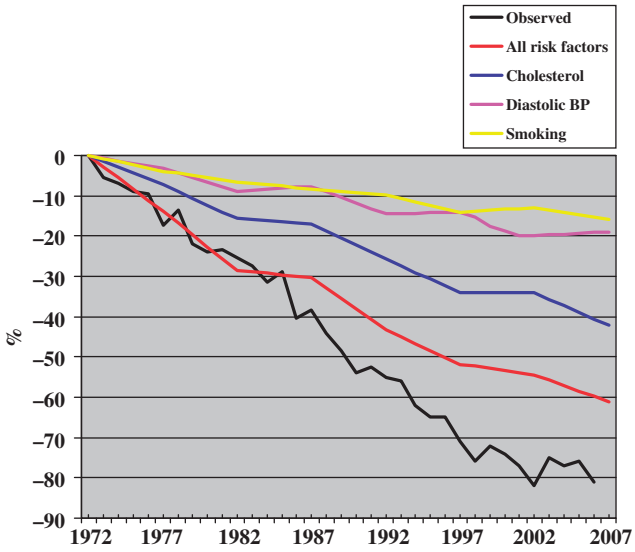


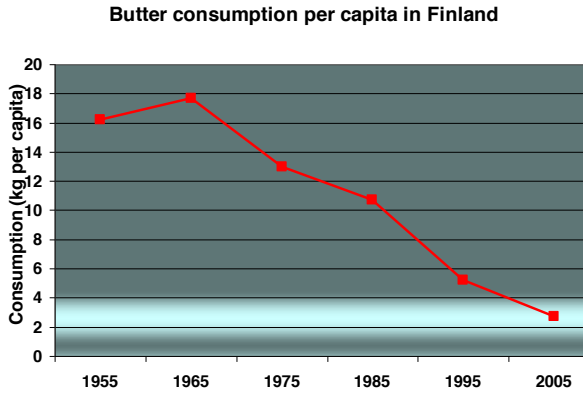
Figure 1 Observed and predicted decline in CHD mortality in men

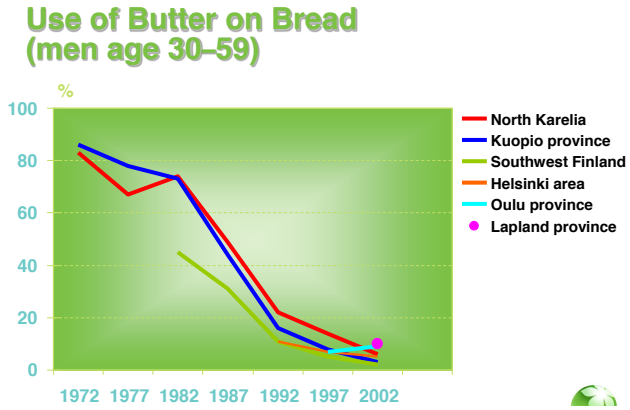
Figure 1 is from the paper. Note that the most significant component of the risk factors is the drop in total serum cholesterol, i.e. two-thirds of the 60% decline in risk factors.[2] "Since the mid-1980s, many new treatments and invasive procedures for coronary patients have become more common and can probably explain most of the remaining decline in observed CHD mortality. In Finland, the change of risk factors seems to be more important than in many other countries. Based on different models, the impact of risk factors on mortality varied from 60% in New Zealand to 44% in the USA, and the impact of treatment varied from 23% in Finland to 47% in the USA."

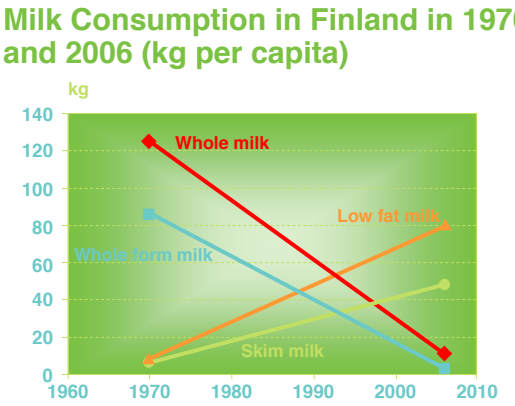
What was responsible for the drop in serum cholesterol? From the paper, "Saturated fatty acids play the key role in the regulation of serum cholesterol." And, ***"The total intake of***

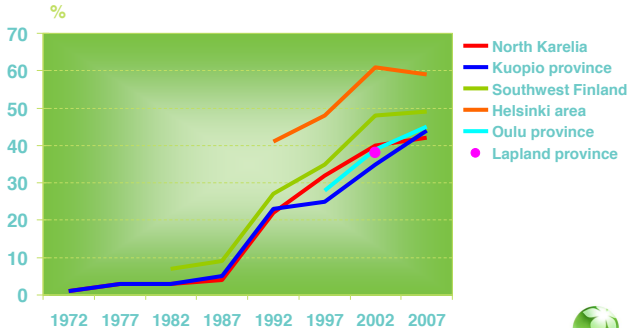
[2] As noted in the paper, "There was a levelling-off period in serum cholesterol level between 1997 and 2002. This was parallel with the changes in saturated and polyunsaturated fat intake."

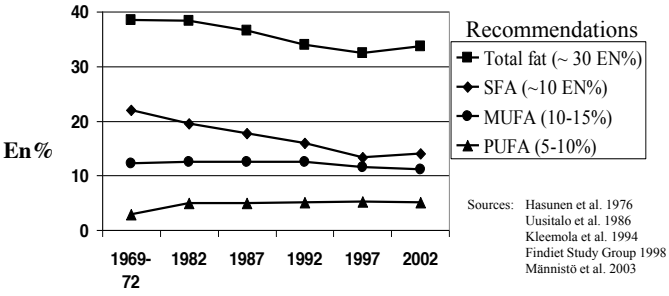
saturated fat has declined in Finland from 22% of energy intake to 13% between 1972

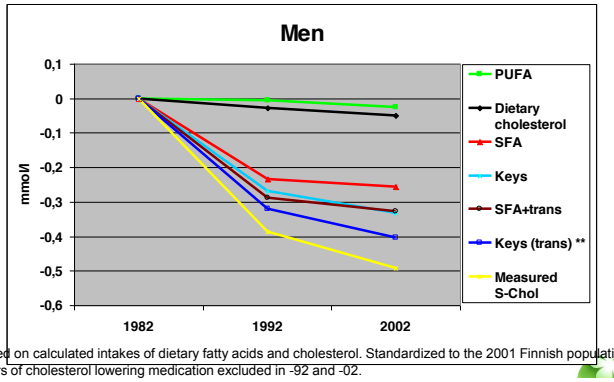












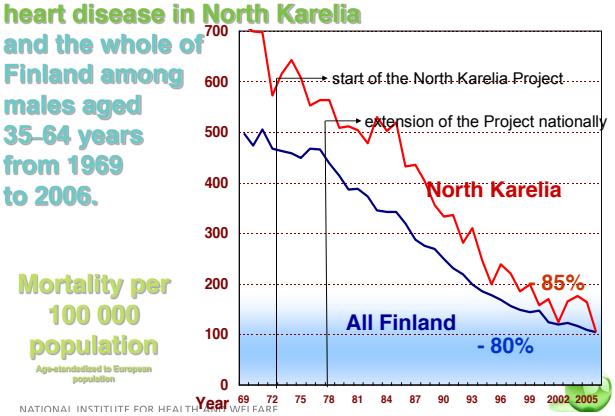
* Based on calculated intakes of dietary fatty acids and cholesterol. Standardized to the 2001 Finnish population. Users of cholesterol lowering medication excluded in -92 and -02.

and 2007."

"During the past 30 years, the greatest change in health behavior in Finland has indisputably been the changes in diet, especially in the type and amount of fat and intake of fresh vegetables and fruit. In the early 1970s, Finland was a country with much dairy farming. Butter

and milk production was subsidized and all vegetable oil was imported. The domestic vegetable oil industry was developed in the late 1970s and the popularity of vegetable oil grew remarkably in the 1980s. People were 'educated' in the effects of the types of fat and so could avoid fatty milk products and spreads with high saturated fat content as well as food products high in cholesterol."

Age-adjusted mortality rates of coronary heart disease in North Karelia and the whole of Finland among males aged 35–64 years from 1969 to 2006.



Mortality Changes in Finland from 1969–71 to 2006 (Men 35–64 Years, Age Adjusted)

	Rate (per 100.000) 1969–71	2006	Change from 1969–71 to 2006
All causes	1328	583	- 56%
All cardiovascular	680	172	- 75%
Coronary heart disease	489	103	- 79%
All cancers	262	124	- 53%

Finland Has Shown

- Prevention of cardiovascular diseases is possible and pays off
- Population based prevention is the most cost effective and sustainable public health approach to CVD control
- Prevention calls for simple changes in some lifestyles (individual, family, community, national and global level action)
- Influencing diet and especially quality of fat is a key issue
- Many results of prevention occur surprisingly quickly (CVD, diabetes) and also at relatively late age
- Comprehensive action, broad collaboration with dedicated leadership and strong government policy support

To summarize, the CHD death rate in Finland over a 35 year period dropped 80%. Three-fourths of this (60%) was explainable by a reduction in risk factors. About two-thirds of that was due to the major drop in serum cholesterol, and that was due principally to the drop in SFA intake from 22% of energy intake (i.e. calories) to 13%.

Kuopio Ischaemic Heart Risk Factor (KIHD) Study

Key Messages

- 1551 middle-aged men from East Finland recruited from 1984-1989. Ended in Dec-2001.
- Studied PUFA intake effect on CVD death.
- Data (Food, lifestyle, blood-work, etc) collected ***only at beginning*** of study.
- Failed to disclose dramatic changes in Finnish diet during the study period in the paper.
- Failed to disclose major confounders (Trans-fats, mercury, excess body iron).
- When I say, ***"Failed"***, I mean that they knew, and *blatantly omitted* relevant data in the paper.
- Insufficient data in the paper to assess SFA intake - why Chowdhury et al had to contact researchers.
- One-day's internet research on the KIHD study would have been sufficient for any one to reject this study from a meta-analysis. Why didn't Chowdhury et al?

This 2005 paper [5], cited by Chowdhury, is titled, "Prediction of Cardiovascular Mortality in Middle-aged Men by Dietary and Serum Linoleic and Polyunsaturated Fatty Acids." Thus, as

the title implies, the paper did not present any data on the risk of CHD with increased SFA intake. Rather, it presented data on the reduced risk of CHD with increasing PUFA (and Linoleic) intake. To get the SFA-intake/CHD-death relationship, Chowdhury et al had to request that data from the authors of the paper.

Before getting into my criticism of the authors, w.r.t. their sin of omissions, I'll present a quick summary of the study. Any quotes are taken directly from the paper.

"The study population comprised a random age-stratified sample of 2682 men living in eastern Finland who were 42, 48, 54, or 60 years old at baseline between 1984 and 1989. . . . all men with a history of CVD, diabetes, or cancer at baseline (n = 1123) were excluded. Men with missing data for both dietary and serum fatty acids were also excluded, leaving 1551 men for the analyses."

Dietary intake was based on 4-day food records (3 workdays and 1 weekend day). The consumption of foods was assessed at the time of blood sampling at baseline. Other measurements (e.g. blood pressure, weight, height), lifestyle (e.g. leisure time physical activity), and history (e.g. medical history, medications) were also determined.

"All deaths that occurred between study entry (March 1984–December 1989) and December 2001 were included." The median follow-up was 14.6 years. 78 men died of CVD (which included CHD and stroke). 225 men died from other causes.

Some info on fat intake, as % of Energy intake, for the entire cohort, as Mean (SD): Total fat, 39.7 (5.8); SFA, 19.4% (4.0); MUFA, 12.4 (2.2); PUFA, 4.5 (1.7). Note that the SFA intake is very high.

The paper presented 4 forms of multivariate analysis. Models 3 and 4 were over-adjustments, in my view. The analysis was done in thirds (tertiles). With Model 2,[3] men with a PUFA intake in the upper third were up to 54% less likely to die of CVD than those with an intake in the lower third (RR, 0.46 [95% CI, 0.24-0.86]).

Although the paper does not provide a similar analysis for SFA intake, it does provide results for dietary PUFA/SFA ratio, i.e. the higher the ratio, the lower risk of CVD. For model 2, men with a ratio in the upper third were up to 40% less likely to die of CVD than those with an intake in the lower third (RR, 0.60 [95% CI, 0.48-0.92]). But as PUFA intake goes up, does SFA intake go down?

---

[3] "model 2, adjusted for age, year of examination, smoking, alcohol consumption, adult socioeconomic status and moderate to vigorous leisure-time physical activity. model 3, adjusted for model 2 and plasma lipid-standardized alpha-tocopherol levels, plasma ascorbic acid, and dietary total energy and energy-adjusted saturated fat and fiber intake; and model 4, adjusted for model 3 and low-density lipoprotein cholesterol concentrations, systolic blood pressure, blood pressure medication, family history of ischemic heart disease, C-reactive protein concentrations, fasting concentrations of insulin and nonesterified fatty acids, and body mass index."

Can we learn anything about SFA intake from this study, as represented by the paper. Quantitatively, no. But we can qualitatively based on some statements in the paper:

> "Dietary PUFA and SAFA intake were inversely correlated (r = –0.34)."
>
> "*The inverse correlations of dietary PUFA and SAFA intake indicate that the apparent benefit of PUFA intake probably comes about in part through substitution of PUFA for SAFA intake (in these men, by substitution of margarine for butter).* This, coupled with the inverse association of the dietary PUFA/SAFA ratio with CVD mortality, ***provides support for increasing PUFA intake at the expense of SAFA intake in the primary prevention of CVD*** and underscores the importance of dietary fat quality over quantity. These findings agree with those from the Western Electric Study, in which coronary death was the outcome, and the Nurses Health Study, in which myocardial infarction was the outcome."

In other words, nothing surprising in this study, but they could have done a better job in showing data that directly supports the above statement in ***Bold***.

The Problems with the KIHD Study [published in 2005]

Key Messages
- Based on the major changes in the Finnish diet during the 14.6yr study period, using measurements taken at the beginning to predict CVD deaths is absurd, and the paper failed to disclose this.
- There were 3 well-documented confounders (trans-fats, mercury, and iron) that had to be known to the authors, but were never mentioned in the KIHD paper.

The dietary intake and blood work were done only at the beginning of this 14.6 year study, approximately from 1986 through 2001. What did the authors of the paper say about this (emphasis is mine):

> "The correlation of serum linoleic acid proportions measured 4 years later with dietary linoleic acid intake at baseline in 895 men participating in the KIHD Study 4-year follow-up was 0.36 (data not shown). Serum esterified fatty acid proportions are thus a good measure of habitual dietary fat composition. Saturated fat intake in Finland has decreased since the mid-1980s [**ref to a 1996 paper**] when the KIHD Study began, but our data indicate that the relative ranking of these men with respect to dietary fat quality ***may*** *be stabile, at least during the first 4 years of follow-up.*"

The 1996 paper that they referenced is the 20yr Finland follow-up study (1972-1992) [8]. Yet from the Thirty-Five year follow-up study discussed in the previous section, data was gathered every 5 years, and the authors would have access to the data through 2002 well before they published their 2005 paper. Also, a 25yr follow-up (1972-1997) was published in 2000 [10], never mentioned or referenced in the KIHD paper (published in 2005).

Thus, given what was going on Finland, the authors' various associations (e.g. U. of Kuopio and Finland's Research Institute of Public Health), the authors must have known that diet and

serum cholesterol levels had major changes between 1987 to 1997 not just in Finland as a whole, but specifically in East Finland. While it is possible that their study population did not change their diet, it seems highly unlikely. At the very least, they should have communicated this in the paper, and specifically made reference to the data that indicated the major changes in East Finland.

For example, from the 25yr paper [10], in Kuopio province, mean serum cholesterol dropped by almost 10% between 1987 and 1997 (240 to 218 mg/dl). Perhaps, more importantly, the percent of men with cholesterol above 250 mg/dl dropped from 38% to 18%.

If the authors had cited this data, would the peer reviewers have approved the article for publication? Perhaps, but they might have insisted that a summary statement like the following be included: Given the major changes that took place in the Finnish diet between 1987 to 2002, the results reported in this study have no credibility.

Why did Chowdhury et al include this study in their meta-analysis? When they contacted the authors of the 2005 paper were the problems with the study communicated?

2. **Other Confounders, undoubtedly known, but Not Mentioned**.

In looking at some related research from Finland, I came across three major confounders that were not even mentioned in the KIHD 2005 paper: trans-fats, mercury, and iron.

Key Messages:
- **Trans-fats.** In a large SW Finland study, men in the highest quintile of Trans-Fat intake had a **38% higher risk of CHD death** than men in the lowest quintile.
- **Mercury.** From the same population of men in the KIHD study, men in the upper third of mercury hair content, had a **68% greater risk of CVD death** than men who had a lower content.
- **Iron.** From the same population of men in the KIHD study, men in the highest third of body iron stores had a **2.9-fold risk of Acute Myocardial Infarction (AMI)** compared with men in the lowest third of body iron stores.
- **No mention in the KIHD 2005 paper of Trans-Fats, Mercury, and Iron.** Why? The Mercury, Iron, and KIHD papers shared an author. The Trans-Fat paper was a huge study (21,930 men), started at the same time as the KIHD study.

Trans-Fats

A different Finish study looked at the “Intake of Fatty Acids and Risk of Coronary Heart Disease in a Cohort of Finnish Men” [12]. This was just a 6 year study, but involved 21,930 men from Southwestern Finland - vs. Eastern Finland, the area of KIHD study. This study consisted of all smokers (at least 5 cigarettes per day), aged 50-69, free from heart disease and diabetes. The original intent of this double-blind/placebo controlled study was to determine whether supplements of Alpha-Tocopherol or Beta-Carotene (**ATBC**) would provide a reduction in lung-cancer incidence. It didn’t. But there was so much data collected in this study that it could also be used to analyze other things.

Food intake was determined at the beginning of the study, and data was presented for both coronary events and coronary death. Since the KIHD just looked at death, just that data will be presented. In this excerpt from table 2 of the study, the data is for quintiles of trans-fat intake (each quintile is defined by the mean of the intake). Thus, **men in the 5th quintile of Trans-Fat intake had a ~38% higher risk of CHD death than ones in the 1st quintile.**[4]

trans-Fatty acids			
Intake (g)	Cases/ person-years	Age-adjusted RR	Multivariate RR
1.3	109/25,070	1.00	1.00
1.7	122/25,756	1.05 (0.81–1.36)	1.05 (0.81–1.36)
2.0	136/26,112	1.14 (0.88–1.47)	1.12 (0.87–1.45)
2.7	111/26,265	0.92 (0.71–1.20)	0.90 (0.69–1.18)
5.6	157/26,186	1.38 (1.08–1.76)	1.39 (1.09–1.78)

Could the intakes of trans-fats also been a factor in the KIHD study? Table 4 on the ATBC study provides an additional clue. This table gives us the intake and measurements by quintile of Trans-fat intake. A few things to note:

- Q5 had less than 15% of the butter intake as any of the other 4 quintiles.
- Q5 had 3-9 times the margarine intake as the other quintiles (i.e. 9X Q1)
- Q5 had the least saturated fat intake (i.e. 40% less than the average intake of Q1-Q4) and the highest PUFA intake (i.e. 2.2x the average of Q1-Q4). This should have been a protective effect, but was evidently trumped by the Trans-fat intake.

TABLE 4. Relation of energy-adjusted *trans*-fatty acid intake to selected coronary heart disease risk factors, as well as intake of nutrients and foods at baseline,* Finnish Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study, 1985–1988

Quintile of *trans*-fatty acid intake	Median daily intake of *trans*-fatty acids (g)	Medians of							
		Age (years)	Smoking (years)	No. of cigarettes/ day	Body mass index (kg/m²)	Serum cholesterol (mmol/liter)	HDL cholesterol† (mmol/liter)	Systolic BP† (mmHg)	Diastolic BP (mmHg)
1	1.3	56.4	36.8	19.6	26.2	6.1	1.1	140	88
2	1.7	56.6	36.5	19.6	25.8	6.2	1.1	140	88
3	2.0	57.0	37.1	19.8	25.6	6.3	1.1	139	87
4	2.7	56.9	36.9	19.6	25.6	6.2	1.1	139	87
5	5.6	56.2	36.9	20.0	26.0	6.0	1.1	140	88

	% of group		Median daily intake of							
	Education (>11 years)	Physical activity (>2 times per week)	Energy (kcal)	Triglycerides (g)	Saturated fatty acids (g)	*cis*-Mono-unsaturated fatty acids (g)	Poly-unsaturated fatty acids (g)	Linoleic acid (g)	Linolenic acid (g)	Omega-3 fish fatty acids (g)
1	11.1	20.5	2,657	88.1	43.9	29.1	8.9	6.4	1.5	0.4
2	9.7	17.6	2,794	101.2	53.4	31.7	8.6	6.1	1.4	0.4
3	8.8	17.3	2,844	110.2	60.1	33.4	8.6	6.0	1.3	0.4
4	10.9	17.9	2,770	101.0	57.8	33.5	10.1	7.4	1.5	0.4
5	13.6	19.6	2,673	100.2	38.1	30.3	19.7	16.6	2.2	0.4

	Median daily intake of									
	Cholesterol (mg)	Dietary fiber (g)	Alcohol (g)	Beta-carotene (mg)	Vitamin C (mg)	Vitamin E (mg)	Butter (g)	Margarine (g)	Red meat (g)	Fish (g)
1	517	24.5	16.9	1.75	102	9.7	35.7	5.4	60.4	34.2
2	574	25.0	12.6	1.75	101	9.5	44.2	8.9	63.4	32.8
3	599	24.4	10.0	1.71	96	9.4	51.2	10.2	62.9	31.3
4	584	23.3	9.6	1.70	93	10.6	43.9	17.3	60.7	30.6
5	465	24.6	10.9	1.67	96	17.8	5.1	49.4	56.3	32.2

* Directly age standardized to distribution of whole cohort.
† HDL cholesterol, high density lipoprotein cholesterol; BP, blood pressure.

[4] Age-adjusted column adjusted for age (5-year category) and treatment group. The Multivariate column was adjusted for smoking, body mass index, blood pressure, intakes of energy, alcohol, and fiber (quintiles), education (<7, 7-11, >11 years), and physical activity (<1,1-2, >2 times per week). Inclusion of fiber may be an over-adjustment, e.g. see MALMO study. But in this case, there is not a significant difference.

Recall the following quote from the KIHD study, "*The inverse correlations of dietary PUFA and SAFA intake indicate that the apparent benefit of PUFA intake probably comes about in part through substitution of PUFA for SAFA intake (in these men, by substitution of margarine for butter).*" I think it is reasonable to assume that the margarine in SW Finland is the same as the margarine in East Finland. Thus, if the KIHD paper is right about the substitution of margarine for butter, then it is likely that Trans-fats were a factor in the KIHD study. And, the Food surveys for both studies (KIHD and ATBC) occurred in the same time-frame (late 1980's).

We further know from the overall Finland data, that butter consumption plummeted between 1985 and 2005. This further suggests that trans-fat intake was likely a factor in the KIHD study, and KIHD paper makes no mention of this. WHY?

What did Chowdhury et al know about this? The ATBC paper is one of the 20 in their meta-analysis. (And, I'll get to that one in Part 3, next month.) But you can see why from the above data on SFA intake being the lowest in the highest Trans-fat quintile that the ATBC paper is not a good study for Chowdhury to include. And, did Chowdhury et al ask the KIHD researchers about the possible problem in their study w.r.t. trans-fat intake, when they were contacted for the SFA data?

Mercury

The ATBC paper provides a clue that mercury could be another confounder in the KIHD study. Here is the quote from the ATBC paper, "Among Finnish men living in the eastern lake area, a high intake of nonfatty freshwater fish and the consequent accumulation of mercury in the body were associated with an excess risk of myocardial infarction as well as death from coronary heart disease, cardiovascular disease, and all causes combined [13, JT Salonen et al 1995 paper]."

Rather than use the 1995 paper, I found a 2005 paper [14]. JT Salonen was one of the authors on both of these Mercury papers. JT Salonen was also one of the authors on the KIHD 2005 paper. So all is connected.

First lets compare the study groups in the KIHD 2005 paper to the Mercury 2005 one. Both start with exactly the same study population from East Finland. Both start with exactly the same number of men, 2,682.

- The Mercury study excluded men with a history of CHD or stroke, and of those remaining, DHA+DPA concentrations were available for 1842 men for the CVD death analysis. "Risk of all-cause mortality was estimated excluding only men without data on serum DHA+DPA concentrations; thus, the analyses for all-cause mortality included 2480 men."
- The KIHD study, "all men with a history of CVD, ***diabetes, or cancer*** at baseline (n = 1123) were excluded. Men with missing data for both dietary and serum fatty acids were also excluded, leaving 1551 men for the analyses." Thus, it seems that the men in the KIHD study are a subset of the 1842 men in the Mercury study.

One other minor difference. The KIHD study ended 1-year earlier (December 2001 vs December 2002). 78 CVD deaths in the KIHD study and 91 in the Mercury one (explainable by the difference in study sizes).

Men in the upper third of mercury hair content (>=2.03ug/g) had about a 68% greater risk of CVD death than men who had a lower content (i.e. the bottom 2 tertiles combined).[5]

Thus, Mercury hair content was a more significant factor in CVD death in the Kuopio Ischaemic Heart Study that any factor reported in the KIHD 2005 paper. Yet, the KIHD 2005 paper never even mentioned mercury as a factor. Why?

What is going on in East Finland with mercury, you might ask. The lakes in East Finland are contaminated with very high levels of mercury. There are a lot of large fish (e.g. pike) taken from these lakes and eaten. I suspect that the source of the lake mercury is from the air pollution of various coal-fired power plants, but I was unable to confirm this. But I was able to confirm that the mercury content of Finnish men is significantly higher than those in Sweden.

It is also interesting that the large fresh water fish, e.g. pike, are very low in all types of fat (i.e. saturated as well as DHA, DPA, and EPA). For example, looking at USDA database, 100g of raw northern pike contains just 0.60g of total fat (88mg of DHA+DPA). In comparison, 100g of raw Atlantic farmed salmon contains 13.4g of total fat (1,400mg of DHA+DPA).

Now an interesting thought experiment, combining all that I have written so far, in Part 2. Imagine a East Finnish man, in the early1990's, eating a lot of fresh-water fish from his local lakes, and pan-frying his fish in margarine. He has a low intake of SFA due to low-fat content of the lake fish, and his choice of margarine (vs butter) increases his heart-healthy PUFA, while reducing his SFA intake. His cardiologist is so happy with him. However, the combination of the trans-fat in the margarine and mercury in the lake fish might just kill him. And, if not, suppose, he is using an cast-*iron* pan to cook his fish. Read on, it gets even worse.

Iron

The previous subsection discussed a 2005 Mercury paper. That mercury paper never mentioned iron, but it did contain a reference to a 1992 paper by JT Salonen et al, titled "High stored iron levels are associated with excess risk of myocardial infarction in eastern Finnish men." [15] That was a controversial paper. So, using a different measuring technique, JT Salonen et al published a 1998 paper, with the same conclusion [16]. Title of the 1998 paper, "Association Between Body Iron Stores and the Risk of Acute Myocardial Infarction in Men."

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[5] Model 4 which adjusted for age, examination year, serum HDL and LDL cholesterol, family history of ischemic heart disease, systolic blood pressure, BMI, maximal oxygen uptake, urinary excretion of nicotine metabolites, serum selenium, serum DHA+DPA, and intake of alcohol, saturated fatty acids, fiber, and vitamins C and E. More precisely, an RR of 1.68 (95%CI, 1.15 to 2.44). With less adjustment, i.e. just age and examination years, the RR was 1.53 (95% CI, 1.08 - 2.18).

First, the 1992 study, which I'll refer to as K-Iron 1992. Similar to the other KIHD studies, i.e. it starts with 2,682 men, and then eliminates some for various reasons. This study eliminated those with some signs of ischemic heart disease, leaving 2005 men. Of these, 1931 had available blood work. In this study, in addition to data on serum cholesterol levels (including LDL and HDL), serum ferritin and blood hemoglobin concentrations were important. Ferritin level is a measure of stored iron.

The mean follow-up in this study was short - just 3 years. The mean serum ferritin concentration in the subjects was 166 ug/L, higher than adult men in most previous studies, eg one study in the state of Washington, the average was 94 ug/L. The median value in adult men is 69-149 ug/L. 25% of men in the K-Iron 1992 study had levels above 200 ug/L, and 6% above 400 ug/L.

From the paper, after "adjusting for age, examination year, cigarette pack-years, ischemic ECG in exercise test, maximal oxygen uptake,systolic blood pressure, blood glucose, serum copper, blood leukocyte count,and serum high density lipoprotein cholesterol,apolipoprotein B,and triglyceride concentrations, ***men with serum ferritin >=200g/l had a 2.2-fold (95%CI,1.2-4.0; p<0.01) risk factor-adjusted risk of acute myocardial infarction compared with men with a lower serum ferritin***. An elevated serum ferritin was a strong risk factor for acute myocardial infarction in all multivariate models. This association was stronger in men with serum low density lipoprotein cholesterol concentration of 5.0 mmol/l (193 mg/dl) or more than in others."

As the authors note, "The present data provide the first empirical evidence in humans of the role of high stored iron measured as elevated serum ferritin concentration as a risk factor for ischemic heart disease. . . . A major proportion of our subjects had serum ferritin levels that put them at increased risk of AMI."

And most interesting is their hypothesis at the end of the article, "Our findings do not undermine the role of LDL cholesterol in the etiology of CHD but rather help to explain why high serum LDL cholesterol concentration is more predictive of CHD in some individuals and populations than in others.The observed synergistic association of serum ferritin and serum LDL cholesterol concentration with the risk of AMI fits into the theory that iron overload would elevate the risk of AMI by promoting the oxidation of LDL."

Now for the 1998 study, which I'll refer to a K-Iron 1998, "Association Between Body Iron Stores and the Risk of Acute Myocardial Infarction in Men". The cases and controls for this study were nested from the KIHD cohort of 1931 men (as described in K-Iron 1992). The measure for iron body iron stores in this study was the concentration ratio of serum transferrin receptor to serum ferritin (TfR/ferritin), a state-of-the-art measurement of body iron stores. The measurements were carried out in 99 men who had an AMI during the 6.4 years of follow-up and 98 control men (matched for age, examination year, and residence). With this measurement a low ratio of TfR/ferritin indicates a high iron store.

Adjusting for the other strongest risk factors for AMI and indicators of inflammation and alcohol intake, ***men in the lowest third of the TfR/ferritin ratio (i.e. a high-level of body iron store)***

had a 2.9-fold (95% CI, 1.3 to 6.6, P=.011) risk of AMI compared with men in the highest third. "These data show an association between increased body iron stores and excess risk of AMI, confirming previous epidemiological findings," i.e., confirming the findings of K-Iron 1992.

To consider the synergism of iron stores and LDL cholesterol, the TfR/ferritin ratio was analyzed for men in 2 groups, below and above the LDL median level, which was 4.2 mmol/L (162mg/dl). After adjustment, in the high LDL cholesterol group, men in the lowest third of TfR/ferritin (i.e. those with a high iron store) had a 4.20-fold risk of AMI (P=0.0226) vs. men in the highest third (those with a low iron store). In men with low cholesterol, and the ratio was 2.43 (P=.148).

The paper also states: "We recently conducted another study in the KIHD cohort, in which we analyzed the association of voluntary blood donation (loss of iron >200 mg per donation) and AMI risk. In a multivariate model adjusted for the main coronary risk factors, blood donors had a relative AMI risk of 0.14 (95% CI, 0.02 to 0.97; P=.047) compared with nondonors. These results provide support for the iron-CHD hypothesis from an additional perspective."

So why the high iron content? The human body has no significant way of ridding itself of excess iron (other than bleeding). Instead, our intestines have a mechanism for controlling the absorption of iron - more absorbed when we need it, and less when we don't. There are 2 forms of dietary iron, heme and non-heme. Animals have both. Plants have just non-heme. Our intestines do a good job of controlling the amount of non-heme iron that is absorbed. But that does not work well for heme iron.[6] Sources of heme iron (highest to lowest) include clams, oysters, organ meats, beef, poultry, and fish. The presence of heme iron in our intestines increases the absorption of non-heme iron. A diet high in beef (about 60% heme, 40% non-heme) increases absorption of the non-heme portion even if the body already has plenty of iron stores. Alcohol also increases iron absorption. And, cooking in an iron skillet/pot adds iron to the food. Coincidentally, a very popular brand of iron cookware comes from Finland. To quote from that website: "The Sarpaneva pot is a classic piece of Finnish cookware design, so famous that it once graced one of Finland's postage stamps. Inspired by his blacksmith grandfather, Timo Sarpaneva designed the ***cast-iron*** Sarpaneva pot in 1960."

Summary

Enough criticizing the authors of the KIHD study. OK, so they stubbed a toe on the way to winning the marathon. Big deal. Everyone screws up from time to time.

The big important story is the amazing accomplishment of Finnish people (led by the medical community with the support of government) to transform their diet, e.g. dropping saturated fat intake from 22% of calories to 13%, and thereby reducing CHD death by 80% over a 35 year period.

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[6] For a good overview of this, see Dr. Greger's video, http://nutritionfacts.org/video/risk-associated-with-iron-supplements/

And, that's not all that Finland has done. You may have read how Finland transformed its educational system over this same period of time. In the 1970's, Finland's student achievement was low, but they steadily upgraded their education system, and reached the top about 5 years ago. And they took a wide achievement gap between rich and poor, and reduced it until it's smaller than nearly all other wealthy nations.

Will our government, industry, and media leaders learn from the Finnish experience? Will we hold them accountable? Or, will we just pass the butter. . . .

Related Information

Travis, who Dr. McDougall has featured in previous newsletters, wrote an excellent critique of both the Chowdhury et al article and Siri-Tarino one (Clearing up the Confustion Surrounding Saturated Fat). If you have any doubts about the medical/nutrition research that supports a low-fat whole-food plant-based (LF-WFPB) diet, simply read all posts on his blog site, and follow the links to the research he references.

Jeff Novick is always bombarded with emails whenever a new article comes out like the Chowdhury one, or the Siri-Tarino one. Check out his many comments on this thread in the forum:
http://www.drmcdougall.com/forums/viewtopic.php?f=22&t=15506

The go-to website for debunking all the low-carb, Paleo, etc folk is Plant Positive's website:
http://www.plantpositive.com
His newest series is Drivers of the Herd.

This recent Dr. Greger short video describes well what a low-carb diet does:
http://nutritionfacts.org/video/low-carb-diets-and-coronary-blood-flow/
Below the video, you will see "Sources Cited". Click on it to expose the links to all the medical/nutritional research referenced in the video.

Fun short video on Wheat Belly & Grain Brain, by Vegsource:
https://www.youtube.com/watch?v=DemGcWQAnpw

And, Dr. McDougall's Feb 2014 video on Wheat-Belly/Grain-Brain/low-carb:
http://www.drmcdougall.com/health/education/videos/free-electures/dr-mcdougall-criticizes-low-carb-diets/

For someone who is just looking for a relatively short intro, I put together a youtube playlist about 15 months ago. It consists of 6 2012 Tedx talks, each is about 17 minutes, from 6 different doctors: Esselstyn, Campbell, Barnard, McDougall, Lisle, and Ornish.
http://www.youtube.com/playlist?list=PL_fyN-QMR_RfvtJ3Hd45P1gnPwXdLfcJh

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