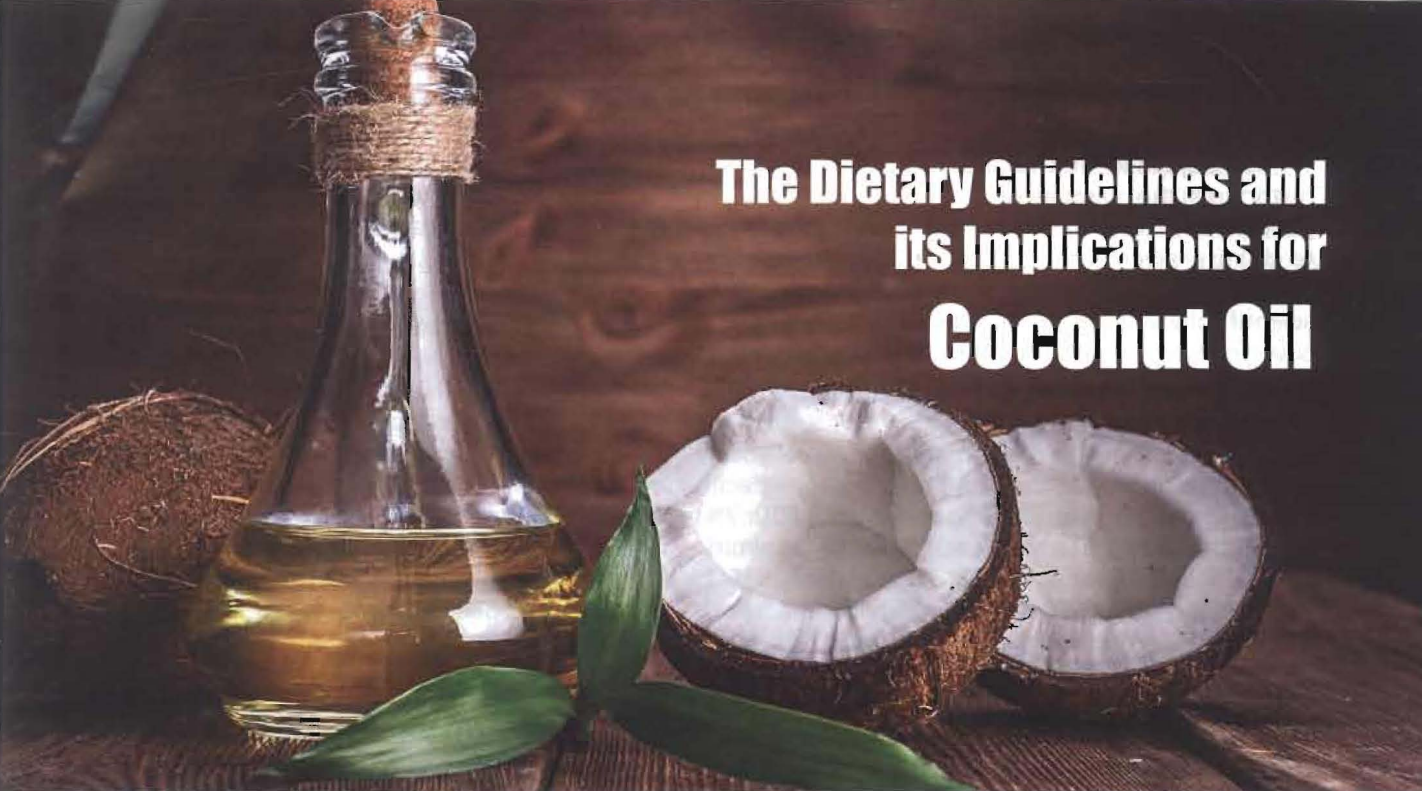


The Dietary Guidelines and its Implications for Coconut Oil



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The dietary advice that is generally followed nationally and internationally closely follows the Dietary Guidelines for Americans which was first published in 1980 and which has been through eight editions. All of the editions of the Dietary Guidelines recommend a diet that is low in fat, and most editions recommend the replacement of saturated fat with polyunsaturated fat. This recommendation is based on the saturated fat-cholesterol-heart disease hypothesis that was first proposed by Ancel Keys in the 1950s. Coconut oil was labeled as unhealthy because of its high saturated fat composition. However, this label is unwarranted. Re-analysis of the work that Keys undertook reveals that he used some inappropriate assumptions that invalidate his hypothesis. Keys undertook a large controlled feeding study, called the Minnesota Coronary Survey (MCS), to prove his hypothesis but he did not publish the results of this work. A recent re-analysis of this work has shown that his results do not support his hypothesis. Further, historical documentary evidence has revealed the significant involvement of the American sugar industry in influencing dietary policy by blaming saturated fat for heart disease. Populations that have adhered

to the low-saturated fat dietary recommendation have become significantly overweight and obese. In contrast, populations that continue to follow their traditional diet which includes coconut have not had high rates of obesity. The Keys hypothesis needs to be abandoned.

Introduction

In 1980, the U.S. Departments of Agriculture, and Health and Human Services published the first edition of the Dietary Guidelines for Americans to serve as the basis for U.S. federal food and nutrition education programs. Despite almost four decades and eight editions of the Dietary Guidelines, heart disease and obesity have remained major health concerns in the US. Cancer has crept up in the list of non-communicable diseases (NCI 2017) and Alzheimer's disease, which is still untreatable, is already exacting significant financial and social costs (Alzheimer's Association 2017). These non-communicable diseases have a dietary link. All editions of the Dietary Guidelines contain warnings against saturated fat, and all (except two editions) warn against coconut oil.

The objective of this article is to recount the history of the campaign against saturated fat in general and coconut oil in particular. It also seeks to discuss, given what is known about the science, whether these dietary recommendations were scientifically valid in the first place. This review will cover mainly the issues related to saturated fat and coconut oil and will only briefly include the issue of cholesterol where this is essential.

Coconut oil has the following approximate fatty acid composition (carbon number: %): caproic acid, C6: <1%; caprylic acid, C8: 7%; capric acid, C10: 7%; lauric acid, C12: 49%; myristic acid, C14: 18%; palmitic acid, C16: 8%; stearic acid: C18: 3%; oleic acid, C18:1: 8%; linoleic acid, C18:2 (ω -6): 2%; linolenic acid: C18:3 (ω -3): <1% (Codex Alimentarius 2015). Since C6, C8, C10, and C12 are classified as medium-chain fatty acids (MCFA) and C14, C16, and C18 are long-chain fatty acids (LCFA), the total proportion of MCFA is about 63% and LCFA is about 29%. Unsaturated fatty acids make up about 8%. Therefore, coconut oil is considered as a medium-chain saturated fat (Bach & Babayan 1982).

Ansel Keys and the Saturated Fat-Heart Disease Hypothesis

The campaign against saturated fat began with Ansel Keys, who was one of the most influential personalities in nutritional science during its foundational years from the 1950s to the 1980s. A very prolific researcher, he was able to develop wide scientific collaborations. His most influential paper was his 1986 "Seven Countries Study," an epidemiological study involving 15 cohort populations comprising 11,579 healthy men aged 40 to 59 years old in seven countries who were followed over 15 years (Keys *et al.* 1986). This was a pioneering multi-country, multi-year epidemiological study which had a great influence on nutritional science. In the said paper, he concluded that: "Death rates were related positively to average percentage of dietary energy from saturated fatty acids."

This paper capped a 35-year effort to link saturated fat, cholesterol and heart disease, an idea that Keys first mentioned in a conference paper that he delivered in South Africa (Keys 1955). Two years later, Keys formalized his saturated fat-heart disease hypothesis and advocated for a limit to be placed on saturated fat in the diet (Keys *et al.* 1957a). Briefly, this hypothesis claims that dietary saturated fats and cholesterol both raise serum cholesterol, and high serum cholesterol causes atherosclerosis that

increases the risk of coronary heart disease (Figure 1). In effect, saturated fat and cholesterol were identified as major causes of heart disease.

However, a re-evaluation of Keys' work has exposed several mistakes that formed part of the basis for his hypothesis. Keys' early experiments on coconut oil used hydrogenated coconut oil (Keys *et al.* 1957a, 1957b). It had already been reported three years earlier that partially hydrogenated fat which produces trans-fat resulted in the formation of atheromas in rabbits (Kritshevsky *et al.* 1954). In 1957, it was reported that trans-fats had been found in the arteries of humans who consumed hydrogenated vegetable oils (Johnston 1957), and later research showed that trans-fats raise serum cholesterol (Mensink & Katan 1990; Matthan *et al.* 2000). Keys' use of hydrogenated coconut oil may explain his results and his consequent bias against coconut oil.

Yerushalmy & Hilleboe (1957), in an immediate critique of Keys' 1957 papers, wrote: "In the proposition considered in this paper – the suggested association between fat in the diet and heart disease mortality – the examination of all available basic data and the tests for specificity show that the association lacks validity. Consequently, the apparent association in itself cannot serve as supporting evidence for the theory that dietary fat plays a role in heart disease mortality." Keys quickly responded to this critique in an editorial writing that: "This is not a test to prove causality which is seldom accessible to critical test by epidemiology but simply a way to decide whether the hypothesis is consistent with the distribution of the disease." (Keys 1957c).

Keys acknowledged the importance of obtaining comprehensive information regarding the distribution of a disease (in this case, heart disease) and this may have been the motivation for his Seven Countries Study. This study collected data on diet and lifestyle, but the subjects were predominantly Caucasian and middle-aged males. The countries that were included were the following: Finland, Greece, Italy, Japan, Netherlands, USA, and Yugoslavia (Keys *et al.* 1986). The fats consumed in these countries would have been mainly animal fat, which are long-chain fats. None of these countries consume coconut oil as a major component of their diet.

As early as 1965, the metabolic differences among the saturated fatty acids were already known. In a paper entitled, "Quantitative effects of dietary fat on serum cholesterol in man," Hegsted and co-workers (1965) identified myristic acid (C14) and palmitic acid

(C16), both LCFA, as primarily responsible for serum cholesterol. Nevertheless, Keys' warning against all types of saturated fat, coconut oil included, remained.

The Dietary Guidelines for Americans, 1980 to 2015

First published in 1980, the Dietary Guidelines for Americans has gone through eight editions. All of them, without exception, include Keys' warning to limit the consumption of saturated fat, regardless of type. Beginning with the 1995 edition, an upper limit of 10% of calories was recommended for saturated fat. Avoidance of coconut oil is specifically mentioned in all, except the 1995 and 2005 editions. Two important comments should be made here. First, although the metabolic and physiological differences between MCFA and LCFA have been known since the 1950s (Bach & Babayan 1982), the guidelines do not acknowledge this difference and have consistently considered them as one group. Second, because one needs to maintain 30-35% of calories from fat in one's diet, the recommendation to keep saturated fat below 10% meant an increase in consumption of unsaturated fat.

- An analysis of the content of the 2015 edition of the Dietary Guidelines shows a bias of recommendations against saturated fat in favor of soybean oil (an omega-6 oil), with a weak warning against refined sugar, in particular high-fructose corn syrup (HFCS)
- The warning against saturated fat is mentioned at least 54 times, without distinguishing medium-chain from long-chain fat.
- In contrast, the warning on trans-fats, which have been known for over two decades to cause coronary heart disease (Kris-Etherton 2010), is mentioned only 11 times and hydrogenated oil only nine times;
- Soy products are mentioned 59 times as a beneficial food item, including seven times for soybean oil. There is no warning against consumption of an excess of omega-6 fats.
- High-fructose corn syrup (HFCS), which is a major driver of obesity in the US (Bray *et al.* 2004), is mentioned only twice.

So what has been the result of the Dietary Guidelines for Americans? In 2010, 30 years after the first edition of the Dietary Guidelines, the Americans were overweight (37%), obese (35%) and extremely obese (6%). Since the early 1960s, the prevalence

of overweight, obese, and extremely obese rose from 48% to 78% in American adults (NIDDK 2012). It is noteworthy that there was a sharp increase in obesity beginning in 1980, the year the first Dietary Guidelines was published. Is this merely coincidental? Might there be a link between the advice being given by the Dietary Guidelines and this rise in the trend of overweight, obese, and extremely obese?

Americans in general have been obediently following the nutrition advice given in the Dietary Guidelines: since 1980, consumption of fats dropped from 45% to 34% while carbohydrate consumption increased from 39% to 51% of total caloric intake. The conclusion from the results is clear: adherence to recommendations to reduce fat and increase polyunsaturated fat consumption coincided with (or resulted in) a substantial increase in obesity (Cohen *et al.* 2015).

Soybean Oil and American Health

One constant warning of the Dietary Guidelines is to replace saturated fat with polyunsaturated fat. Soybean oil, a polyunsaturated fat, currently accounts for approximately 50% of vegetable oil consumption in the US, while coconut oil accounts for less than 3% (USDA 2014, Index Mundi 2016).

The saturated fat in the American diet comes mostly from meat and meat products, such as milk, cheese, and butter. Red meat accounts for 58% while fish accounts for only 10% of protein consumed in the US (Daniel *et al.* 2011). The large consumption of soybean oil (an omega-6 oil) and the relatively low consumption of fish (the major source of omega-3 oil) give an omega-6 to omega-3 ratio of about 15:1. Such a high omega-6 to omega-3 ratio has been associated with cardiovascular disease, cancer, and chronic inflammatory and autoimmune diseases. The ideal omega-6 to omega-3 ratio is about 4:1 (Simopoulos 2002, 2008, 2010).

A polyunsaturated fat (e.g., soybean oil) and a predominantly medium-chain saturated fat (e.g., coconut oil) would be expected to show different metabolic behaviors. Two studies comparing soybean oil and coconut oil show better health outcomes for coconut oil. A 12-week randomized, double-blind clinical trial involving 40 abdominally-obese (waist circumference, WC > 88 cm) women aged 20-40 years was conducted to determine how soybean oil and coconut oil would affect lipid parameters (HDL, LDL:HDL ratio, TC), WC, and BMI. The protocol also included 50 min of walking four days a week. Soybean oil caused an increase in TC and LDL, and

a decrease in HDL resulting in an increased LDL:HDL ratio. Coconut oil in comparison gave higher HDL and a lower LDL:HDL ratio. Both oils showed reductions in BMI, but only coconut oil gave a reduction in WC. The study concluded that dietetic supplementation with coconut oil does not cause dyslipidemia and may promote a reduction in abdominal obesity (Assunção *et al.* 2009).

Another study entitled “Soybean oil is more obesogenic and diabetogenic than coconut oil and fructose in mouse: potential role for the liver” reveals why soybean oil is an unhealthy oil when it is a major part of the diet (Deol *et al.* 2015). This study showed that soybean oil caused a general dysregulation of the genes of a major liver enzyme, cytochrome P450. Other genes involved in obesity, diabetes, inflammation, mitochondrial function, and cancer were also upregulated by the soybean oil diet. This study provides a direct causal link between soybean oil and obesity, diabetes, inflammation, and possibly cancer. Coconut oil in comparison did not give such effects.

High Refined Sugar and High PUFA Cause Various Diseases

Carbohydrates make up part of a normal diet. However, industrially refined sugars, especially in high amounts may lead to diabetes, obesity and various inflammatory diseases. It is well known that hyperglycemia, or high blood glucose, increases the risk of diabetes. However, the other common sugar (fructose) may be as harmful as glucose, if not more so. In processed foods, especially in soft drinks and other beverages, fructose is commonly introduced as high-fructose corn syrup (HFCS) because it is cheaper and easier to process than regular sugar (sucrose). Goran and co-workers (2012) found that the prevalence of diabetes was 20% higher in countries where HFCS is readily available as compared to others where HFCS is not as available. Whereas glucose is removed from the bloodstream by insulin, fructose flows through the blood stream until it reaches the liver where it is converted into fat and increases the secretion of very low density lipoprotein (VLDL). Fructose also decreases glucose tolerance, and raises the levels of insulin (hyperinsulinemia) and uric acid (hyperuricemia) (Mayes 1993; Sun & Empie 2012). Fructose has been found to be much more susceptible to autoxidation than glucose or sucrose. This autoxidation is promoted by phosphate and generates free radicals that can in turn oxidize PUFA and LDL (Lawrence *et al.* 2008).

In addition, elevated levels of glucose and fructose in the blood stream are harmful because these compounds react chemically with proteins to form adducts called advanced glycation end-products (AGE) (de Vos *et al.* 2016). On the other hand, when polyunsaturated fatty acids (PUFA) are heated, they are oxidized and form degradation products, such as malondialdehyde, which also react with proteins to form AGE.

AGE-formation alters the structure of proteins which may prevent proteins from functioning properly. If the protein is an enzyme, the reactions that it promotes may be blocked; if the protein is a receptor, this may prevent important processes from occurring. AGE provides a causative link between high sugar and high polyunsaturated fatty acid (PUFA) oils in the diet to various metabolic diseases, such as diabetes (Goldin *et al.* 2006), cardiovascular disease (Hegab *et al.* 2012), and Alzheimer’s disease (Ko *et al.* 2015).

Sugar Industry Tries to Hide the Truth, Blames Saturated Fat

Early warnings linking sucrose with coronary heart disease (CHD) began to emerge in the 1950s. John Yudkin, the founding professor of the Department of Nutrition, Queen Elizabeth College, University of London, was a contemporary of Ancel Keys who had a similar interest in determining whether diet had any influence on heart disease. However, Yudkin found the epidemiological link with sugar, not fat and claimed that: “There has never been any direct evidence for the hypothesis that fat consumption has anything to do with causing the disease; indeed, recent evidence points increasingly against it. For example, studies in East Africa have shown that cardiac ischaemia is rare both in the Samburu and in the Masai, who consume very large amounts of fat, almost all of it from meat and milk... On the other hand, there is now quite impressive evidence that a high intake of sugar (sucrose) may be an important factor in producing coronary disease. Firstly, epidemiological studies in which populations are compared show a rather better relationship of the incidence of the disease with sugar consumption than with fat consumption. Secondly, the increasing incidence of the disease that is found in many countries has followed an increasing consumption of sugar rather than of fat” (Yudkin 1965).

Keys (1971) was vigorous in his defense of sugar: “The widely publicized theory that sucrose in the diet is a major factor in the development of coronary

heart disease has been examined. The theory is not supported by acceptable clinical, epidemiological, theoretical or experimental evidence.”

Keys and other leading American nutritionists maintained their attack on saturated fat. In 1961, Keys co-authored a report of the American Heart Association with Harvard professor Frederick Stare which recommended “reasonable substitution of polyunsaturated for saturated fats as a possible means of preventing atherosclerosis and decreasing the risk of heart attacks and strokes” (Page *et al.* 1961).

In 1967, Stare, along with two other Harvard professors, Robert McGandy and Mark Hegsted, wrote two influential nutrition reviews that blamed saturated fats and cholesterol for heart disease concluding that “on the basis of epidemiologic, experimental and clinical evidence, that a lowering of the proportion of dietary saturated fatty acids, increasing the proportion of polyunsaturated acids and reducing the level of dietary cholesterol are the dietary changes most likely to be of benefit” (McGandy *et al.* 1967b). Intriguingly, they ended the review with this defense: “Since diets low in fat and high in sugar are rarely taken, we conclude that the practical significance of differences in dietary carbohydrate is minimal in comparison to those related to dietary fat and cholesterol.” (McGandy *et al.* 1967b). This may have been true in 1967, but is no longer true today when the amount of sugar may be as high as 25 percent of calories.

Recently uncovered industry documents reveal that the attacks on saturated fat and the promotion of sugar were part of a concerted campaign that was funded by the Sugar Research Foundation (SRF). As early as 1954, the SRF had identified a strategic opportunity for the sugar industry to increase market share by promoting a low fat (high sugar) diet and in 1965, it started strategically funding research projects by influential individuals. In 1967, the SRF paid \$6,500 for two key review papers by McGandy and colleagues (1967a, 1967b). This campaign continues to the present time with the promulgation of pro-sugar policies by various international agencies (Kearns *et al.* 2016). In all of these policies, the recommendations have been always the same: take a low-fat diet and replace saturated fat with polyunsaturated fat. This is a dietary battle that continues to this day.

The influence that the sugar industry wields over American and global nutrition policy can be

partly traced to the experts whom the industry supported. Fredrick Stare was one of the most influential American nutritionists. He founded the Department of Nutrition at the Harvard School of Public Health (HSPH) in 1942 and served as Chairman until he retired in 1976, a period of 34 years. He was the founding editor of *Nutrition Reviews*, wrote an American nationally syndicated column for many years entitled “Food and Your Health,” and published several popular books on nutrition. Stare was also a member of the scientific advisory committee of the SRF (Hegsted 2004; Kearns *et al.* 2016). Mark Hegsted, professor of nutrition at the HSPH, exercised strong influence on the US Food and Nutrition Board and the American Heart Association. He served on the editorial board of the most influential nutrition journals: *Journal of Lipid Research*, *Nutrition Reviews*, *American Journal of Clinical Nutrition*, and *Journal of Nutrition* and helped draft the first edition of *Dietary Guidelines for Americans* (Dwyer *et al.* 2010; Scrimshaw 2014). A significant part of American nutrition policy was shaped by Stare and Hegsted, and their students (Hegsted 2004; Dwyer 2010; Scrimshaw 2014).

Correlation is Not Proof

Epidemiological data, such as those obtained from the Seven Countries Study and its follow-up studies, provided correlations between a meat diet (which contains long-chain fat) and serum cholesterol. Serum cholesterol is then used as a surrogate indicator for heart disease. However, there are at least two correlations being applied, both of which do not provide unique proof for cause and effect. For example:

- Meat contains long-chain animal fat, but it contains many other components that are known to be harmful. If meat is fried in polyunsaturated oil, malondialdehyde and other degradation products may be produced which lead to the formation of AGE and free radicals (de Vos *et al.* 2016). The cholesterol present in the meat may oxidize and form oxidized cholesterol which has been shown to be harmful (Ng *et al.* 2008). Red meat has been found to produce trimethylamine (TMA) in the gut through the agency of gut microbiota. TMA in turn is converted into trimethylamine oxide (TMAO) in the liver and TMAO has been shown to cause atherosclerosis, which is linked to heart disease (Liu *et al.* 2015). Thus, the simple correlation between animal fat and heart disease does not prove causation and is not valid.

- Serum cholesterol itself is not a valid indicator of risk of heart disease. Interestingly, this is a result that Keys himself showed in his 1952 paper where he showed that there was a natural tendency in healthy men for cholesterol levels to increase with age. His own data showed that at age 20, healthy males have a serum cholesterol level of around 190 mg/dL, and this increases to over 260 mg/dL at age 70 (Keys 1952). However, he never referred to this work in his later studies. Later, Keys' failed MCS experiment showed that serum cholesterol levels do not predict heart disease.

Unfavorable Results were Withheld from Publication

Proof requires evidence of causality and Keys was aware of this. Since the Seven Countries Study was only an observational study, Keys wanted to do another study where he could carefully control the diet of the test subjects. In 1967, Keys and Ivan Frantz, Jr. undertook a project entitled "Effect of a Dietary Change on Human Cardiovascular Disease," also called the "Minnesota Coronary Survey" (MCS). This study was funded by the US National Heart, Lung and Blood Institute and was undertaken from 1968 to 1973. MCS was meant to be a landmark study because of its experimental design: the large number of subjects (n=9,423, male and female, age 20-97); the length of the feeding study (five years); the high level of dietary control; and the double-blind randomized protocol. MCS used residents in a nursing home and patients in six state mental hospitals in Minnesota. This enabled the study to carefully control and document the food that was actually consumed. The MCS study sought to apply the equation that Keys had first proposed in 1957 that correlated saturated fat with high serum cholesterol and then with heart disease (Keys *et al.* 1957a; Keys & Parlin 1966). Keys' fat diet-cholesterol-heart disease hypothesis had never been causally demonstrated in a randomized controlled trial and the MCS study was meant to prove this hypothesis. This study was conducted at the same time that Keys was coordinating the Seven Countries Study and would have provided powerful validation of the saturated fat-cholesterol-heart disease hypothesis.

Unfortunately, Keys himself did not publish the full results of this study and it remained hidden until Ramsden and co-workers (2016) obtained the raw data from this study over 40 years after it was conducted and subjected it to full analysis (O'Connor 2016). The analysis of MCS data was performed by

Ramsden and co-workers (2016) and are summarized as follows:

- The group that consumed the high linoleic acid diet showed a significant reduction in serum cholesterol compared with those on the saturated fat group. However, there was no difference in mortality among the groups.
- There was a higher risk of death in subjects who showed reduction in serum cholesterol level (22% increase in risk for each 30 mg/dL reduction in serum cholesterol).
- The main conclusions were that a high linoleic acid diet effectively lowers serum cholesterol but this increases the risk of CHD.

A partial release of the results of MCS study was made in a 1989 paper in the journal *Arteriosclerosis* with Frantz as lead author. This paper made the modest conclusion that: "For the entire study population, no differences between the treatment (high linoleic acid group) and control (high saturated fat group) were observed for cardiovascular events, cardiovascular deaths, or total mortality." (Frantz *et al.* 1989).

The results of the MCS study did not give the expected results and directly contradicted the conclusions of the Seven Countries Study which Keys had published a few years earlier in 1986. Although Keys was a co-proponent of the MCS study, his name did not appear as a co-author in the *Arteriosclerosis* paper; he was not even mentioned in the Acknowledgment. This might also explain why it was published in a journal of more limited circulation which gave it less exposure. It is clear that a wider distribution of the results of the *Arteriosclerosis* paper, with Keys properly included as co-author, would have been fatal to the saturated fat-cholesterol-heart disease hypothesis.

Influence on WHO Policy

The World Health Organization has adopted the saturated fat-cholesterol-heart disease hypothesis. For example, the WHO Healthy Diet Fact sheet No. 394 reads: "For adults. A healthy diet contains... less than 30% of total energy intake from fats. Unsaturated fats (e.g. found in fish, avocado, nuts, sunflower, canola and olive oils) are preferable to saturated fats (e.g. found in fatty meat, butter, palm and coconut oil, cream, cheese, ghee and lard) ..." (WHO 2015).

This WHO report was taken from a publication of the Food and Agriculture Organization, *Fats and*



Fatty Acids in Human Nutrition Report of an Expert Consultation.

“The Expert Consultation recognizes that grouping of fatty acids into these three broad groups (SFA, MUFA and PUFA) is based on chemical classifications, but it is clear that individual fatty acids within these groups have distinct biological properties. However, most of the epidemiological evidence reviewed by the experts uses broad groupings, which makes it difficult to distinguish and disentangle the effects of individual fatty acids.

“SFA refers to the major SFA in our diet, namely C14, C16, C18, except in the case of milk and coconut oil where SFA range from C4 to C18” (FAO 2008).

The experts ignored the distinction between medium-chain and long-chain fat. This distinction is central to the understanding of the health effects of coconut oil, which is made up of about 63% medium-chain fat. The failure to recognize this difference makes this document’s conclusions regarding coconut oil incorrect.

The Role of Coconut in Traditional Healthy Diets

There is abundant evidence that people who abandon their traditional coconut diets in favor of the American diet have become overweight or obese. WHO (2003) reported that Pacific islanders “were 2.2 times more likely to be obese and 2.4 times more likely to be diabetic if they consumed fat from imported foods rather than from traditional fat sources. The most commonly consumed imported foods providing fats were identified as oil, margarine, butter, meat and chicken, tinned meat and tinned fish.” Traditional fat sources in the Pacific islands are coconut, fish and pork.

A 1999 study among American Samoans showed that a shift to a modern diet increased

their carbohydrate and protein consumption and decreased their overall fat, in particular, saturated fat. This shift was identified as the cause of their increased incidence of obesity and cardiovascular disease (Galanis *et al.* 1999).

Evidence from Polynesia and the Philippines show that there is no link between coconut oil consumption and cardiovascular disease. In the Polynesian islands of Pukapuka and Tokelau, Prior (1981) reported that: “Vascular disease is uncommon in both populations (Pukapuka and Tokelau) and there is no evidence of the high saturated fat intake having a harmful effect in these populations.” Likewise, a population-wide study by Florentino & Aguinaldo (1987) in the Philippines showed that: “High coconut oil intake is not consistent with high CVD mortality rate.” They then concluded that: “These observations do not seem to corroborate the contention that coconut oil as naturally ingested in the diet together with other fat sources increases the risk of CVD.”

Conclusions and the Way Forward

Ansel Keys’ landmark Seven Countries Study became the basis for the recommendation of the Dietary Guidelines for Americans to consume a low-fat diet and to replace saturated fat by unsaturated fat. This is currently being put to question. Further, Keys’ study covered primarily animal fat which is mainly long-chain fat and is not applicable to a predominantly medium-chain fat, such as coconut oil. Therefore, the basis for Keys’ inclusion of coconut oil is incorrect.

Historical documentary evidence of the significant influence of the American sugar industry and a detailed analysis of published and unpublished research on dietary fat show that the current dietary recommendations for a low-fat diet and replacement of saturated fat with polyunsaturated fat are wrong and heavily influenced. Populations that have followed these recommendations have become significantly overweight and obese. Coconut oil has been labeled as unhealthy because of its high saturated fat composition. However, populations that consume significant amounts of coconut do not have high rates of obesity and heart disease. The Keys saturated fat-cholesterol-heart disease hypothesis has been shown to be in error in numerous studies, and significantly, in a study which Keys himself did not publish. This hypothesis should be abandoned. ■

Source: http://coconutresearchcenter.org/hwnl_15-4.htm