Healthy Ways Newsletter

The cutting edge source of information on coconut, fats and oils, diet, and nutrition.

E-Edition, Volume 15-4

Published September, 2018

Piccadilly Books, Ltd. www.piccadillybooks.com

For a free subscription click here

Bruce Fife, N.D. Publisher www.coconutresearchcenter.org

Print-Friendly Version

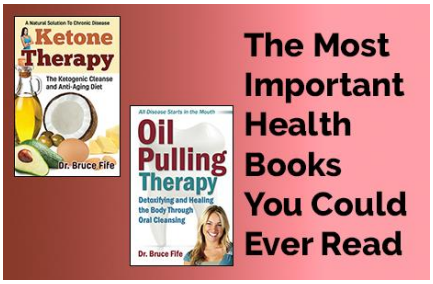
Stories in this issue

Is Coconut Oil A Poison?

"Coconut oil is pure poison," so claims Karin Michels, PhD, a Harvard professor during a lecture at the University of Freiburg, Germany. Is Dr. Michels correct? Is coconut oil poisonous? Let's look at the science. read more

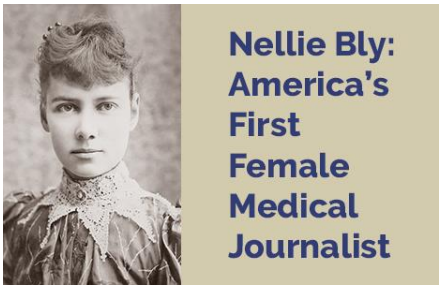
The Secret of Oil Pulling?

There have been many theories proposed to explain the incredible healing effects of oil pulling, but what exactly is it that makes oil pulling so effective? read more



The Most Important Health Books You Could Ever Read

There are thousands of health books covering a wide variety of topics ranging from diet and nutrition to exercise and physical therapies. Out of all of these books, which two or three books are the most useful? read more



Nellie Bly: America's First Female Medical Journalist

Nellie Bly could be called America's first female medical investigative journalist. She is best known for her undercover exposé of the infamous Blackwell's Island Insane Asylum. read more

The Dietary Guidelines and Its Implications for Coconut Oil

The last article in a five-part series by Dr. Fabian Dayrit. The government's dietary guidelines still recommend limiting saturated fat consumption. New research has proven there is no risk to eating saturated fats and in particular coconut oil. read more

Is Coconut Oil A Poison

"Coconut oil is pure poison," claims Karin Michels, PhD, a part-time professor at Harvard TH Chan School of Public Health. Her comments given in a talk at the University of Freiburg, Germany, sparked a media frenzy with headlines such as, "Coconut Oil is Pure Poison Harvard Professor Claims" appearing in newspapers and on the internet.

What makes Dr. Michels an authority on coconut oil? She is not a physician, or a nutritionist, or even a biologist. Her PhD is in biostatistics. Her specialty is statistics—manipulating numbers—not the study of diet or fats and oils. From her profile on the Harvard website, it appears she has never published any studies on saturated fat, let alone on coconut oil. Her comments were not based on any of her own published research, but were simply her *opinion* based on *old, outdated theories* about saturated fats.

Michels calls coconut oil "pure poison," saying it was "one of the worst foods you can eat" because it is full of saturated fat, and "saturated fatty acids can clog your arteries." She adds that "there is no study that proves significant health benefits of coconut oil."

Dr. Michels makes three general claims: 1) saturated fats cause heart disease, 2) coconut oil is a poison, and 3) there are no studies that show any health benefits to coconut oil. Let's look at what the science actually says about each of these statements.

Saturated Fats Cause Heart Disease

There has never been a study published that has been able to show that saturated fats or coconut oil cause heart disease. The *diet-heart disease hypothesis* that has been popular for the past 6 decades basically states that heart disease is caused by high cholesterol. Many studies have shown that some saturated fats can raise blood cholesterol, and therefore it has be assumed that eating too much saturated fat can promote or even cause heart disease. Researchers have been trying to prove this hypothesis for over a half a century without success. In fact, many studies have seriously challenged this hypothesis and serious researchers have now moved on to studying new, more likely, causes for heart disease.

Cholesterol is no longer considered the evil villain as it was once portrayed. There are many types of cholesterol, some good and some potentially harmful. Saturated fats, and in particular coconut oil, have been shown to raise HDL, the good cholesterol, that has been shown to protect against heart disease. The ratio of total cholesterol to HDL cholesterol is considered one of the most accurate and reliable indicators of heart disease risk. Coconut oil raises HDL, which lowers the cholesterol ratio, thus lowering the risk of heart disease. [1]

It is apparent that Dr. Michels has not kept up with the current science on coconut oil or fats and oils in general. Earlier this year researchers at the University of Cambridge School of Clinical Medicine published a study on the relationship between coconut oil and heart disease risk. The researchers compared the effects of coconut oil with butter and olive oil. Butter was chosen to represent a commonly used highly saturated animal fat and extra virgin olive oil was chosen as it is generally regarded as one of, if not the healthiest of fats. The study involved 96 participants who were assigned to consume 50 mg (about 3 tablespoons) of one of each of the three oils daily for 4 weeks as a part of their ordinary diet. The researchers found that coconut oil dramatically raises the protective HDL cholesterol without affecting the LDL or so-called bad cholesterol. Coconut oil lowered the

cholesterol ratio, and the risk of heart disease, more than either of the other two fats, indicating that it is even more heart-friendly than extra virgin olive oil. [2]

In recent years numerous studies have exonerated saturated fat as a cause of heart disease and put to rest the outdated diet-heart disease hypothesis. Last year the *Lancet*, one of the most prestigious medical journals in the world, published a study involving a team of 37 researchers from 18 countries. They gathered data on 135,000 subjects to evaluate heart disease risk in relation to fat intake. They discovered that fat consumption protected against heart disease and increased lifespan. Those people who cut back on fats, including saturated fat, had far shorter lives than those who ate coconut oil, butter, cheese, and meats. Consuming high levels of all fats, cut early death rates by up to 23 percent. The researchers stated that they found no correlation between saturated fat consumption and cardiovascular disease and that current dietary restrictions on saturated fat should be revised. [3]

This isn't the only study in recent years that has called for a revision on the recommendation to restrict saturated fats. A study published in the *American Journal of Clinical Nutrition* a year earlier investigated whether dietary saturated fat was associated with ischemic heart disease. The study involved 35,597 participants. The researchers also concluded that high saturated fat intake was not associated with increased risk of ischemic heart disease. [4]

In 2010 a groundbreaking study was published clearly showing that saturated fats do not cause heart disease. The study published in the *American Journal of Clinical Nutrition* analyzed all the previous studies with data for dietary saturated fat intakes and the risk of cardiovascular disease. This meta-analysis combined the data from 21 previously published studies, involving over 347,000 subjects. The study showed that there was no connection between saturated fat consumption and heart disease. Those people who ate the greatest amount of saturated fat where no more likely to suffer a heart attack or stroke than those who ate the least. No matter how much saturated fat one ate, the incidence of heart disease was not affected. This was the most complete review of the medical research on saturated fat ever done up to this time. [5]

Four years later, a different group of researchers from the University of Cambridge published another meta-analysis. This time the researchers combined the data from 72 previously published studies involving more than 600,000 participants from 18 countries. The researchers basically combined all the highest quality studies on fats and diet that had been done for the past several decades and analyzed them together. The results confirmed the previous meta-analysis—there is no connection between saturated fat intake and heart disease. [6]

The studies are clear, neither saturated fat nor coconut oil cause or even promote heart disease. Because they raise good HDL cholesterol and lower the cholesterol ratio, if anything, they help to protect against it.

Coconut Oil is a Poison

Dr. Michels calls coconut oil "pure poison." She claims it is not just a poison, but a "pure" poison; the connotation is, that it is extremely dangerous at even the smallest dosage. What is a poison? According to the *English Oxford Living Dictionary*, poison is defined as, "A substance that is capable of causing the illness or death of a living organism when introduced or absorbed." Does coconut oil fit this definition? Not hardly.

Coconut oil has been a major part of the diet of millions of people for thousands of years. In all that time it has never been known to cause any illness or kill anyone. On the contrary, there are many plants that are poisonous such as hemlock, belladonna (deadly nightshade), and death cap mushrooms. Consuming any of them, even in small amounts, will bring about sudden illness and quick death. Coconut oil, on the other hand, can be consumed daily in relatively large quantities without any ill effect. I know some people who consume as much as 12 tablespoons (180 ml) a day and are in excellent health.

According the United States Food and Drug Administration (FDA) coconut oil is perfectly harmless. It is included among the FDA's exclusive GRAS (Generally Regarded as Safe) list of food substances. To be included on this list requires rigorous testing to confirm that the item is safe. Coconut oil is given a GRAS

classification of "1," which is the highest or safest category within the GRAS list. According to the FDA this means that all available studies and historical data have shown that there is "no evidence" that shows or even "suggests" that coconut oil is harmful in any way. [7]

It is ironic that Dr. Michels calls coconut oil a poison, because it has proven to be not only harmless, but highly effective in saving the lives of people who have ingested actual poisons. The medical literature has described numerous instances in which coconut oil has been used in hospital settings as an antidote to otherwise fatal poisonings. For instance, the use of coconut oil has become a routine practice in some hospitals in the treatment of aluminum phosphide poisoning. [8] Aluminum phosphide is a common poison used for rodent control. There is no other known antidote and poisonings are almost always fatal unless treated with coconut oil.

Using coconut oil to nullify the effects of poisons is not that unusual. Researchers have known for many years about the detoxifying properties of coconut oil. Numerous animal studies have shown that coconut oil blocks the deleterious effects of a number of different chemical toxins. Coconut oil has been shown to alleviate the effects of at least 36 known toxins ranging from industrial solvents to aflatoxin. [9]

Calling coconut oil a pure poison only illustrates Dr. Michels' lack of knowledge about coconut oil, which makes anything she says about it totally unreliable.

There Are No Studies That Show Any Health Benefits to Coconut Oil

One of the most common arguments given in an attempt to discredit coconut oil is to claim that there is no evidence proving coconut oil has any health benefits. When a doctor or professor makes this statement, he or she is inferring that there are no studies to support the use of coconut oil as a healthy fat. They are counting on the listener to take their word on this simply because they are considered an expert. In reality, what they are doing is exposing their own ignorance and lack of knowledge on the subject.

When someone makes this type of statement it means they have not bothered to make even the slightest effort to find the facts. If they had, they would have found an abundance of information and research on coconut oil describing its many health benefits. Currently, there are over 10,000 studies on coconut oil listed in the medical literature. Most of these studies can be easily accessed on the internet.

If you go to my website, www.coconutresearchcenter.org and look under the heading "Medical Research," you will find a listing of hundreds of studies. Here you will find references to an abundance of published studies showing the therapeutic or beneficial effects of coconut oil on cardiovascular health, immune function, cancer, diabetes, liver and kidney health, digestive function, weight management, and much more. To say that there is no evidence for the health benefits of coconut oil is totally wrong and indicates that the speaker is either woefully ignorant, too lazy to do any research, or lying.

If you want to know the truth about saturated fats and coconut oil you should not listen to professors who have no idea what they are talking about, instead listen to researchers who have actually researched the topic. One of the reasons why Dr. Michels' comments received such notoriety is because of her association with Harvard. Being a Harvard professor gives a person some air of authority. However, there are other Harvard professors who are far more qualified than Dr. Michels on this subject, who have studied and published works on the health effects of coconut oil. One group of Harvard researchers that includes George L. Blackburn, MD, PhD, Edward Mascioli, MD, and Vigan K. Babyan, PhD state, "Coconut oil has an important medical role to play in nutrition, metabolism, and health care. Indeed, properly formulated and utilized, coconut oil may be the preferred vegetable oil in our diet and the special hospital foods used promoting patient recovery." These researchers made this statement after having spent years studying the health effects of coconut oil and other fats. Their comments hold far more authority than a biostatistician who apparently has never even bothered to do even an internet search on the subject.

References

Assuncao, ML, et al. Effects of dietary coconut oil on the biochemical and anthropometric profiles of women presenting abdominal obesity. *Lipids* 2009;44:593-601.

Khaw, KT, et al. Randomised trial of coconut oil, olive oil or butter on blood lipids and other cardiovascular risk factors in healthy men and women. *BMJ Open* 2018;8:e020167.

Dehghan, M, et al. Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. *Lancet* 2017;390:2050-2062.

Praagman, J, et al. The association between dietary saturated fatty acids and ischemic heart disease depends on the type and source of fatty acid in the European Prospective Investigation into Cancer and Nutrition-Netherlands cohort. *Am J Clin Nutr* 2016;103:356-365.

Siri-Tarino, PW, et al. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. *Am J Clin Nutr* 2010;91:535-546.

Chowdhury, R, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med* 2014;160:398-406.

https://www.fda.gov/Food/IngredientsPackagingLabeling/GRAS/SCOGS/default.htm.

Singh Baiwa, SJ, et al. Management of celphos poisoning with a novel intervention: a ray of hope in the darkest of clouds. *Anesth Essays Res* 2010;4:20-24.

Fife, B. *Ketone Therapy: The Ketogenic Cleanse and Anti-Aging Diet*. Piccadilly Books, Ltd.: Colorado Springs, CO; 2017.

The Secret of Oil Pulling

There have been many theories proposed to explain the incredible healing effects of oil pulling, but what exactly is it that makes oil pulling so effective?

If you do oil pulling daily, you are aware of how well it works to whiten your teeth, freshen your breath, and improve the general health of your teeth and gums. If you are unfamiliar with oil pulling, basically it is swishing vegetable oil (I recommend coconut oil), in your mouth like a mouthwash for several minutes and then spitting it out. Although very simple, the results can be incredible. Below are some of the comments people have made after they began oil pulling.

"I had what was the onset of an abscess," says Becki. "A 20-year-old crown needed to be replaced. I was going out of the country in a few days and couldn't get in to my dentist. I had oil pulled some in the past and had heard outrageous stories that oil pulling could actually help with cavities, mouth pain, tooth pain, etc. I was worried of having a miserable time on my vacation so, desperately, I started pulling with coconut oil two times per day. By the day I boarded my plane I had no evidence of the abscess. Still pulling, still no abscess, still haven't had the crown replaced! It's been three years! I'm a believer!"

"I have been oil pulling once a day for nearly a year and a half, and according to my hygienist, my teeth are in the best condition that they have ever been in," says Gregg. "Two years ago, two different dentists recommended that a couple of my molars either be pulled or root canalled. One dentist was able to take his probe and go to the end of the root on one of the teeth. Since oil pulling and cleaning up my diet, I still have all of my teeth, no root canals, and no pain."

"I swear by oil pulling," says C.J. "Under the watch of a dentist it has remineralized cracked, transparent teeth. I can't see those cracks anymore. Glass-like edges are fully remineralized into white teeth. Remineralizing was visible in two weeks of daily oil pulling and near done after one month. Stopped periodontal disease with gum pockets as deep as 5mm, completely closed, healthy in three days to one week; infections and sensitivities gone. Four subsequent checkups with no new cavities, no dental work needed, had not experienced that in over 40 years. Whitened teeth, despite still drinking lots of tea and some coffee."

How does oil pulling bring about its incredible cleansing and healing action? Some sources online claim that the remarkable cleansing effects of oil pulling are due to saponification—the process of turning oil into soap. Is oil pulling simply a process of washing the mouth with soap?

Soap is formed by mixing a fat or oil with a strong alkali. Lye, a very strong and caustic alkali, is used in soap making. It has been theorized that the vegetable oil used in oil pulling combines with bicarbonate in saliva creating a crude, mild soap. The swishing action not only creates the soap but scours the mouth clean at the same time. If that were the case, then any soap would work as well as oil pulling does, including the strong detergents contained in toothpastes and mouthwashes (i.e., sodium benzoate, sodium lauryl sulfate, etc.). However, there is no toothpaste or mouthwash that has the same cleansing or healing power as oil pulling.

Another problem with the saponification theory is that the average pH of saliva is 6.7—mildly acidic, not alkaline. Consequently, in the vast majority of cases soap would never be able to form. So the saponification theory doesn't look too promising. Despite these discrepancies, the saponification theory seems to be a popular explanation as to why oil pulling works so well.

To determine if this theory has any validity, I decided to put it to the test. I collected samples of both coconut and sesame oils after 15 minutes of oil pulling. The water (saliva) in the samples was evaporated off. During

this process it was obvious that saponification did not occur as the oil and saliva readily separated, with the oil floating on top. If the oil-saliva mixture had saponified the mixture would not have separated. Visually the saponification theory appears to have flunked the test.

One final step, however, would determine conclusively if any saponification actually occurred. Unlike fats and oils, soap is non-combustible—it will not burn. This characteristic is used in the design of fire extinguishers used on oil and grease fires. Oils burn at much hotter temperatures than ordinary combustibles and require special extinguishers. These fire extinguishers contain chemicals that convert the burning oil into soap, thus killing the fire.

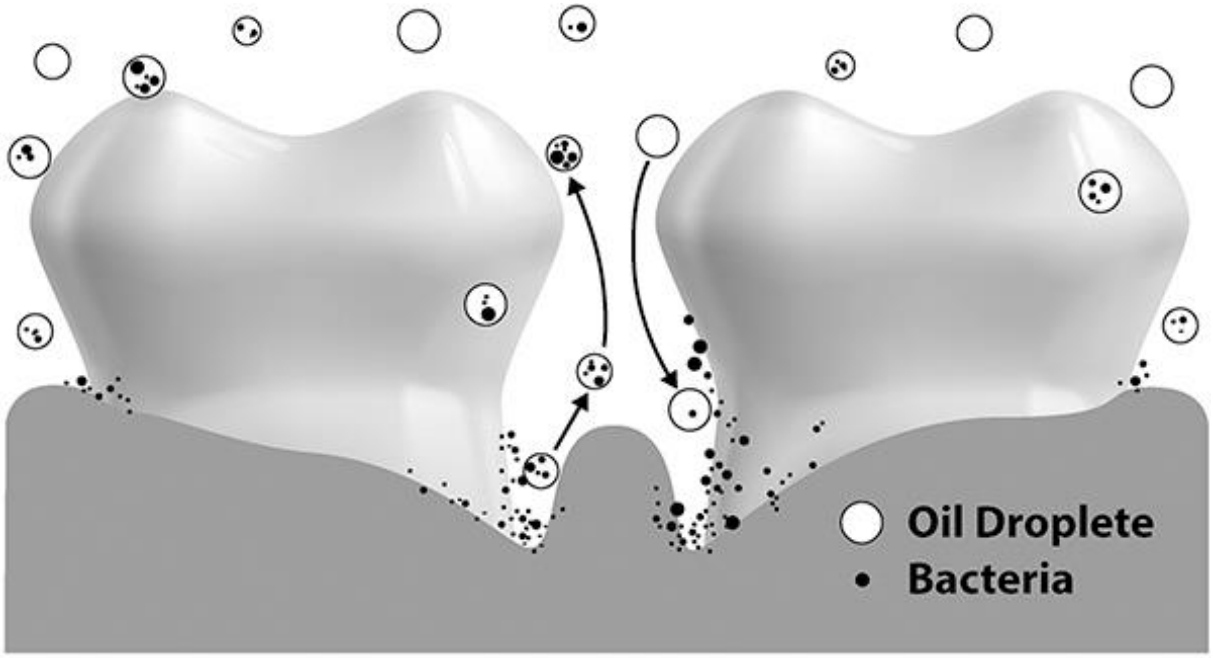
After removing the water/saliva from the two samples, what remained was either soap or oil. In both cases the samples were readily flammable, proving beyond any doubt that they had not been converted into soap. The saponification theory has been proven false.

If oil pulling does not involve saponification, what makes it such an effective oral cleanser? This is a topic I discuss in detail in my book *Oil Pulling Therapy: Detoxifying and Healing the Body Through Oral Cleansing.*

The mechanism is very simple. Oil pulling works by removing disease-causing microorganisms and toxins in the mouth that cause poor dental health. How does the oil work its magic? There is nothing magic about it; it is simple chemistry. Most of the microorganisms that inhabit the mouth consist of a single cell. These cells are covered with a lipid or fatty membrane, which is basically the cell's skin. Even the membranes surrounding our own cells are composed predominately of fat.

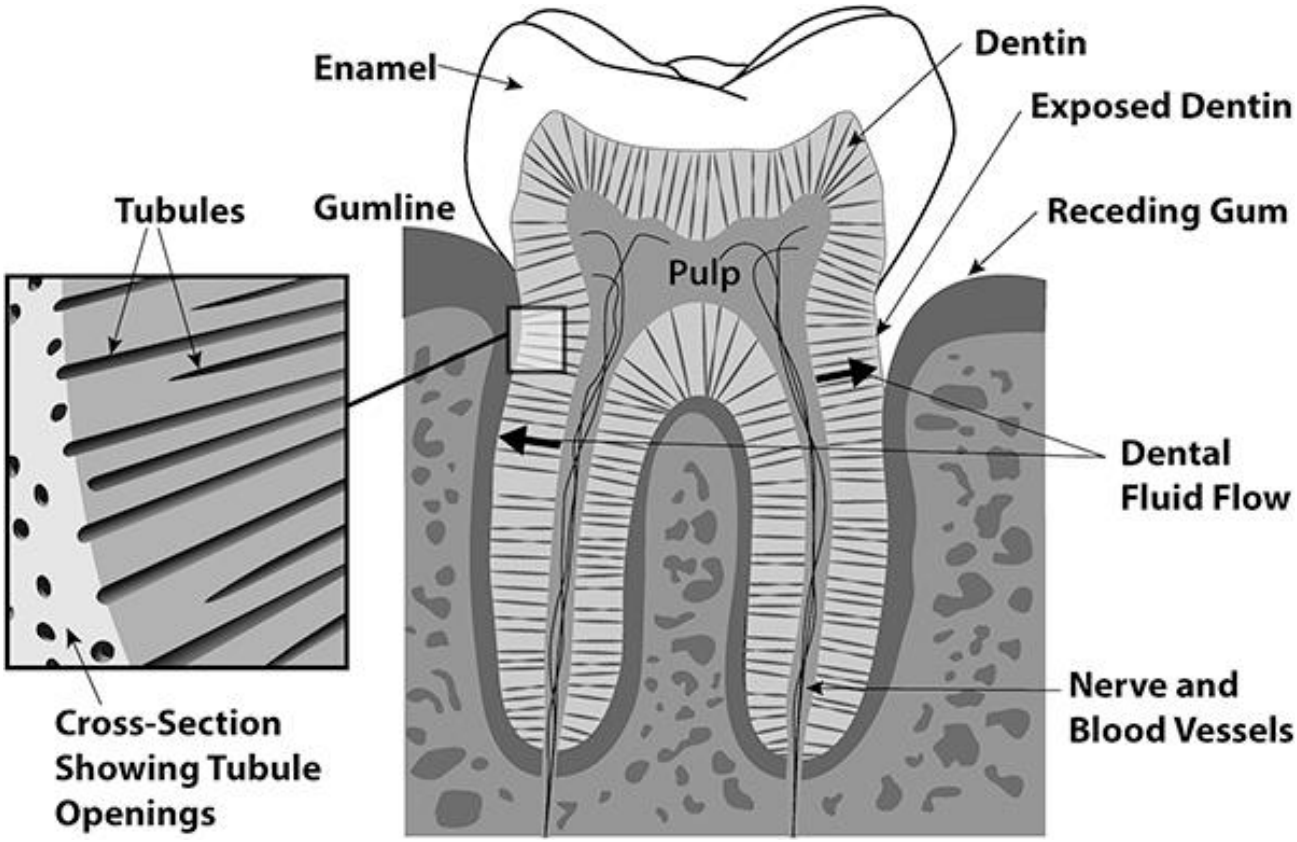
When you mix oil (fat) and water together, what happens? They separate. Oil and water do not mix. But when you add two oils together, what happens? They combine. They are attracted to each other like magnets. *This is the secret to oil pulling*. When you put oil into your mouth, the fatty membranes of the microorganisms are attracted to it. As you swish the oil around your teeth and gums, microbes are picked up as though they are being drawn to a powerful magnet. Bacteria hiding under crevices in the gums and in pores and tubules within the teeth are sucked out of their hiding places and held firmly in the solution. The longer you push and pull the oil though your teeth and gums, the more microbes are pulled free from their hiding places and absorbed into the oil. After 15-20 minutes the solution is filled with bacteria, viruses, and other organisms. This is why you want to spit it out rather than swallow it.

Food particles that get trapped between the teeth are also worked free. Much of it is also attracted to the oil, and if not, it is attracted to the saliva (water based) and still pulled out. So oil pulling literally "pulls" microbes and food particles (their food source) out of your mouth. The addition of saliva also helps to fight certain microbes and balances pH. Thus, you remove disease-causing substances and increase healing substances every time you pull.



Caption: As the oil is worked in the mouth, the swishing action emulsifies the oil with saliva, breaking the oil into a multitude of tiny droplets. As these droplets circulate around the mouth and teeth they absorb bacteria and other microbes. The droplets are small enough to reach deep down into periodontal pockets sucking up the bacteria that tend to hide there beyond the reach of tooth brushes, floss, and mouthwashes. Mouthwashes, which are water soluble, are unable to absorb microbes or pull them out of these deep pockets. This is one of the reasons why oil pulling is so effective at pulling infection out from around affected teeth.

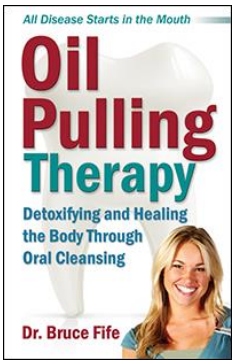
Our teeth contain millions of tiny canals or tubules that allow nutrients to flow from the bloodstream into the root of the tooth and out through the surface of the tooth and into the mouth. The normal, healthy flow of this fluid is from the inside of the tooth to the outside. This natural flow prevents bacteria from penetrating into the tubules and infecting the tooth. However, the consumption of excess sugar can cause the flow of this dental fluid to stop or even reverse, drawing bacteria into the tooth. Eating a diet high in sugar creates an environment that allows bacteria to colonize the dental tubules leading to tooth decay.



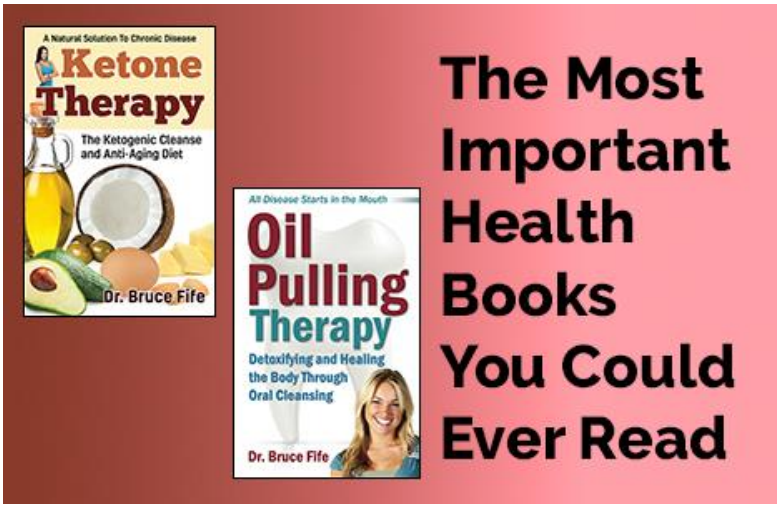
Caption: Dental fluid normally flows from the pulp outward distributing nutrients and minerals throughout the tooth. This outward flow prevents microorganisms from infiltrating the inside of the tooth. However, excessive sugar consumption can stop or reverse dental fluid flow, drawing bacteria inside of the tooth where they can colonize the tubules, causing decay. The sucking and swishing action of oil pulling reinforces the natural outward flow of dental fluid, drawing bacteria out of the tubules, preventing and stopping dental decay.

The sucking and swishing action of oil pulling creates a sucking force on and around the teeth, much like sucking on a straw. Bacteria hiding deep within periodontal pockets and within the tubules inside the tooth itself are drawn out. The flow of dental fluid through the tubules flushes bacteria out of these tiny canals where they are absorbed into the oil droplets and eventually are expelled from the mouth. In this manner, potential cavity-causing bacteria are removed from the inside of the teeth, as well as from deep periodontal pockets.

No amount of brushing, dental floss, antiseptic mouthwash, fluoride, oral irrigation using water-jets, or professional deep cleaning can remove the bacteria that penetrate into the tubule system of a tooth. The only way to remove this type of intruder is by increasing the outward flow of dental fluid and the only way to do that is through a proper diet and oil pulling. This is why oil pulling has proven to be so successful in helping people overcome dental problems when standard dental hygiene and professional care have failed.



Oil Pulling Therapy
by Dr. Bruce Fife
Available from Piccadilly Books, Ltd.
for more information or to order click here



The Most Important Health Books You Could Ever Read

There are thousands of health books covering a wide variety of topics ranging from diet and nutrition to exercise and physical therapies. Many of these books provide valuable information that can help improve overall health or aid those with specific health problems. Out of all these books, which ones provide the greatest overall benefit?

In my opinion there are two books that offer the potential for the greatest overall benefit to your health. They are *Oil Pulling Therapy* and *Ketone Therapy*. These two books contain information found in no other source.

Oil Pulling Therapy describes what I believe to be the simplest, yet most powerful natural form of oral cleansing and detoxification known. It describes a method of oral cleansing that has proven to be superior to tooth brushing and even the most potent antibacterial mouthwashes at reducing dental plaque, tooth decay, bad breath, and gum disease. Oil pulling can pull out infections and promote healing that is impossible with brushing and mouthwash or even routine dental work. It will often solve problems that were destined for major dental surgery and permanent tooth loss. It is inexpensive, simple to do, and far less traumatic than getting dental work done.

Oil pulling isn't just for the mouth, but affects the health of the entire body. The book explains in detail how and why oral health influences our entire bodies and can be the root cause of numerous health issues seemingly unrelated to oral health, such as arthritis, Alzheimer's, and diabetes, and even affect the growth and development of unborn infants. Adding oil pulling to your regular oral hygiene routine could not only protect you from a multitude of health issues, but improve or completely remove many of them. I don't know of any other method or non-dietary therapy that has such broad healing power.

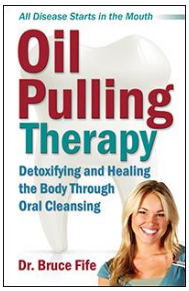
The second book *Ketone Therapy* is diet based. This book describes what is the most effective dietary approach for improving overall health. Ketone therapy can improve and balance all of the health markers that doctors routinely measure when evaluating a person's health status such as blood pressure, blood sugar and insulin levels, cholesterol and triglyceride levels, systemic inflammation, and body mass index.

Ketone therapy has proven to be far more effective at improving health parameters than any other diet. In addition, it can be used to prevent, reverse, and even heal cancer, atherosclerosis, obesity, diabetes, hormonal imbalances, glaucoma, chronic fatigue, inflammatory bowel disease and other digestive disorders, and a multitude of brain disorders including Alzheimer's, Parkinson's, ALS, autism, epilepsy, multiple sclerosis, stroke, and traumatic brain injury. No other diet in existence can do even a fraction of this.

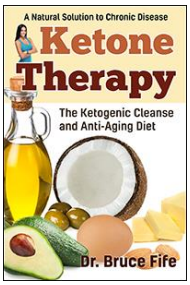
While there have been many books written about the ketogenic diet, most of which focus on weight loss, this book goes beyond the simple mechanics of the diet and its effect on weight. It focuses on how to use ketones, whether from the diet, coconut oil, or supplements for therapeutic purposes. It also dispels many of the myths and misconceptions about the ketogenic diet found on the Internet and in many other ketogenic books.

While not among my two top selections, but definitely high up on my list is *The Coconut Oil Miracle*. This book dispels the myths about coconut oil and explains why saturated fats, and particularly coconut oil can be used to improve your health and your life. It provides the reader with guidance in making wise dietary choices, especially when it comes to fats and oils.

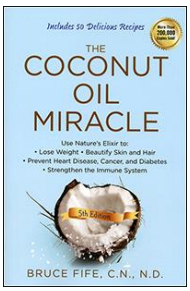
Another book I would highly recommend is *Dr. Fife's Keto Cookery*. This should be a companion volume to *Ketone Therapy* or any other book on the ketogenic diet. This book provides nearly 450 ketogenic recipes to help to help people be successful on a ketogenic diet. Unlike many of the recipes that are promoted as being ketogenic on the Internet and in ketogenic cookbooks, these recipes are truly ketogenic. Other so-called ketogenic recipes are not actually ketogenic. They may be low-carb, but not ketogenic, some are actually anti-ketogenic and will kick you out of ketosis. If you are going on a ketogenic diet to achieve any of the benefits I've outlined above, you won't get the full benefit unless your meals are actually ketogenic. That's why this book is so important. It is the only book, to my knowledge and I have looked at many, in which all of the recipes are ketogenic.



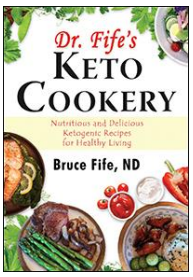
Oil Pulling Therapy
by Dr. Bruce Fife
Available from Piccadilly Books, Ltd.
for more information or to order click here



Ketone Therapy
by Dr. Bruce Fife
Available from Piccadilly Books, Ltd.
for more information or to order click here.



The Coconut Oil Miracle
by Dr. Bruce Fife
Available from Piccadilly Books, Ltd.
for more information or to order click here

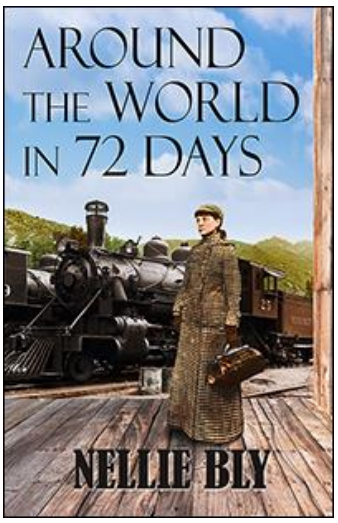


Dr. Fife's Keto Cookery
by Dr. Bruce Fife
Available from Piccadilly Books, Ltd.
for more information or to order click here

Nellie Bly: America's First Female Investigative Journalist

Nelly Bly's was one of America's most famous female journalists of the 19th century. She was a pioneer in the field of investigative journalism and was widely known for her writings about domestic servitude, political corruption, religious sects, baby-selling rings, swindlers, women's suffrage, and worker's strikes. She was a social crusader inasmuch as her editors allowed her to be. Bly was a mold-breaker in every sense of the word; she went places and did things that women had never done before, including her record breaking race around the world.

Inspired by Jules Verne's bestselling novel *Around the World in 80 Days*, Bly set off to follow the footsteps of Verne's fictional hero Phileas Fogg and circle the globe. Sponsored by her employer the *New York World* newspaper, she wrote articles chronicling her adventures. For a young woman to make such a journey alone in the 1880s was unheard of. Learning of Bly's around the world attempt, a competing newspaper the *Cosmopolitan*, sponsored its own reporter to beat the times of both Fogg and Bly. The race was on. Departing on the same day, Bly traveled eastward while her adversary went westward. Bly took advantage of all forms of transportation available to her in the 1880s—train, ship, carriage, rickshaw, sampan, catamaran, and even a donkey.



Around The World In 72 Days
By Nellie Bly
Available from Piccadilly Books, Ltd.
for information or to order click here

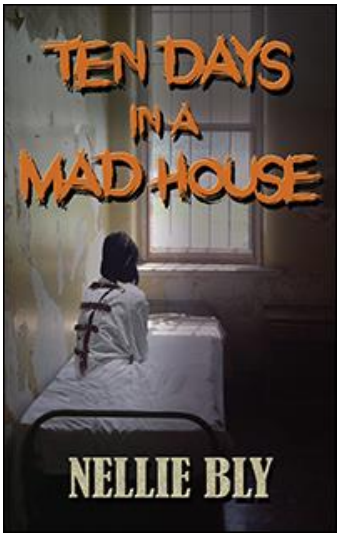
She traveled from New York to Europe, the Middle East, Southeast Asia, China, Japan, and across the American frontier completing the race in just 72 days beating her rival and establishing a world's record. She later wrote a book chronicling her adventure, titled *Around the World in 72 Days*, which became a bestseller.

She often undertook investigative assignments that were part dare and part circulation booster. On another occasion, she posed as an unwed mother to unmask the baby-buying trade. Working for a time as a foreign correspondent in Mexico, she risked imprisonment for writing critical articles of the government, prompting her to flee the country. Later in her career, she served as a war correspondent during World War I and wrote stories about the Eastern front, some of which described the role of women in combat.

Probably her most daring assignment was to get committed to an insane asylum in order to investigate rumors of brutality and neglect. Bly feigned lunacy to get herself committed to the infamous Blackwell's Island Insane Asylum in New York City. The asylum was underfunded, grossly overcrowded, and plagued with scandal. In the 1880s the mentally insane were not treated as patients with an illness, but as dangerous lunatics that had to be controlled by force, confined to cells, and kept out of the public eye. The insane ward where the patients were housed was referred to as the madhouse.

Once committed, she found it near impossible to get out. “From the moment I entered the insane ward on the Island,” writes Bly, “I made no attempt to keep up the assumed role of insanity. I talked and acted just as I do in ordinary life. Yet strange to say, the more sanely I talked and acted, the crazier I was thought to be by all....” Now trapped, Bly was tormented with rotted food, cruel attendants, and cramped and diseased conditions. After talking with other patients she became convinced many were just as sane as she was. In fact, the staff of the asylum, some of which were convicts from the nearby prison, was more frightening than the inmates.

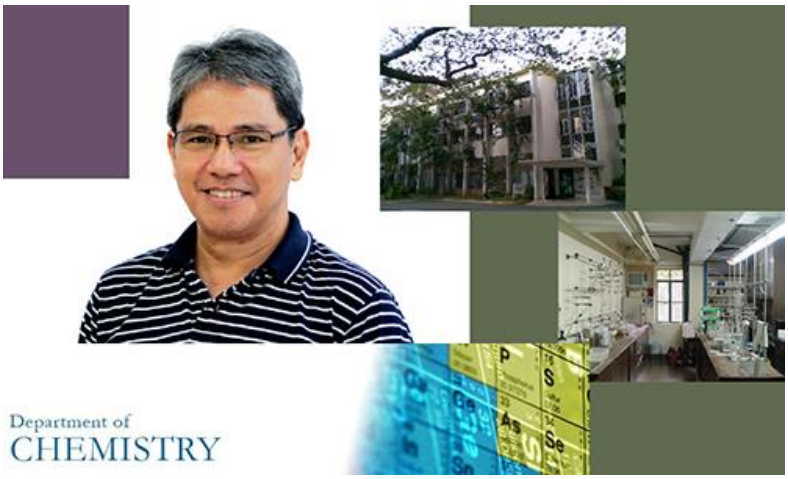
She was unable to convince the doctors she was not a lunatic and to let her free. Only with the aid of her editor was she able to escape and write about her harrowing experience trapped inside a madhouse. She later wrote a bestselling book recounting her experience titled *Ten Days in A Madhouse.*



Ten Days In A Madhouse
By Nellie Bly
Available from Piccadilly Books, Ltd.
for information or to order click here

The book provides an intriguing view into the way the mentally ill were treated a century ago. When it was released in 1887, it created quite a sensation, which led to a grand jury investigation and sweeping reform in the treatment of the mentally ill.

Anyone who is interested in mental health, psychology, the history of medicine, or the accomplishments of women throughout history will find this book fascinating.



The Dietary Guidelines and its Implications for Coconut Oil

Philippine Journal of Science
146 (3): 305-314, September 2017
ISSN 0031 - 7683
Date Received: 03 Apr 2017

Fabian M. Dayrit*

*Corresponding author: fdayrit@ateneo.edu
Chair, Scientific Advisory Committee on Health,
Asian and Pacific Coconut Community
Department of Chemistry, Ateneo de Manila University,
Katipunan Ave., Loyola Heights, Quezon City, Philippines

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.

Key words: Ancel Keys, coconut oil, dietary guidelines, dietary fat-heart disease hypothesis

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 essay 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).

Ancel Keys and the Saturated Fat-Heart Disease Hypothesis

The campaign against saturated fat began with Ancel 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.

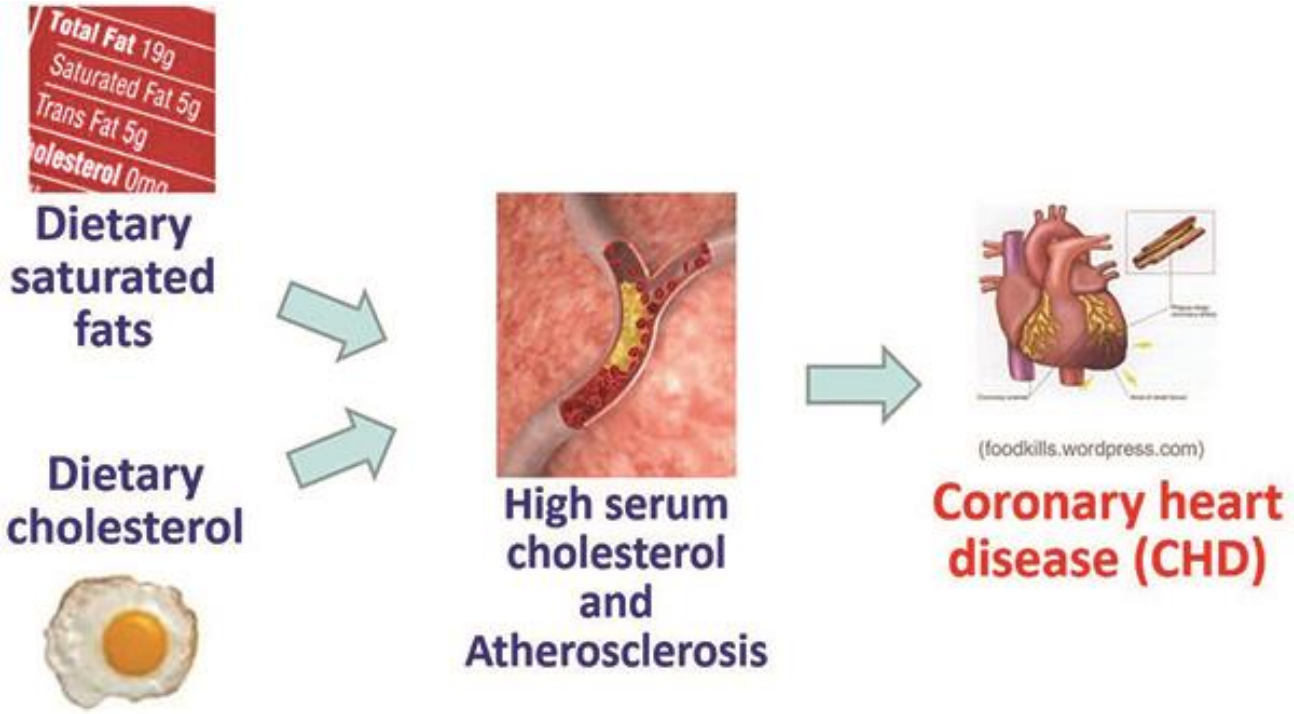


Figure 1. Ancel Keys proposed the saturated fat-cholesterol-heart disease hypothesis in 1957 to explain the correlations that he found between saturated fat, dietary cholesterol, and coronary heart disease.

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 (Figure 2) (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).

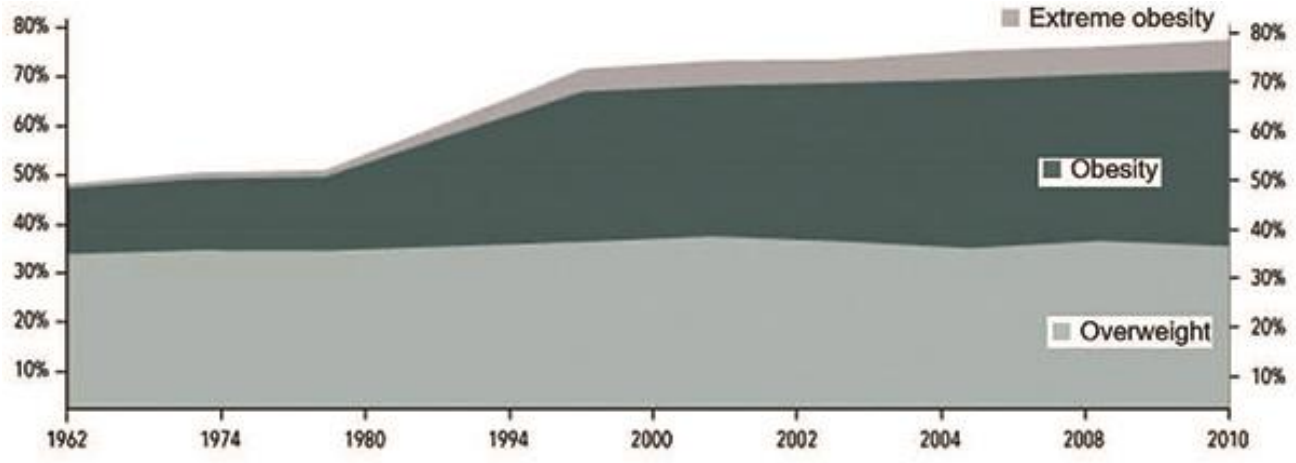


Figure 2. Trends in overweight, obese and extremely obese among American adults, 1962–2010 (NIDDK 2012).

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) (Figure 3).

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).

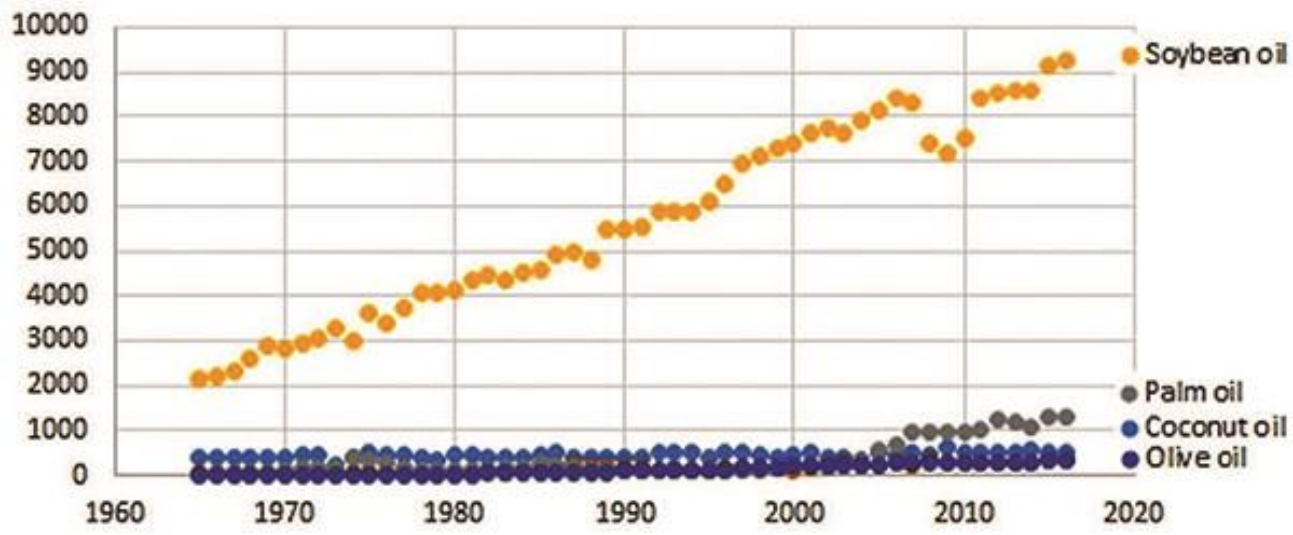


Figure 3. Vegetable oil consumption in US, 1965–2016 (data from Index Mundi 2016).

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 (Figure 4). 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.

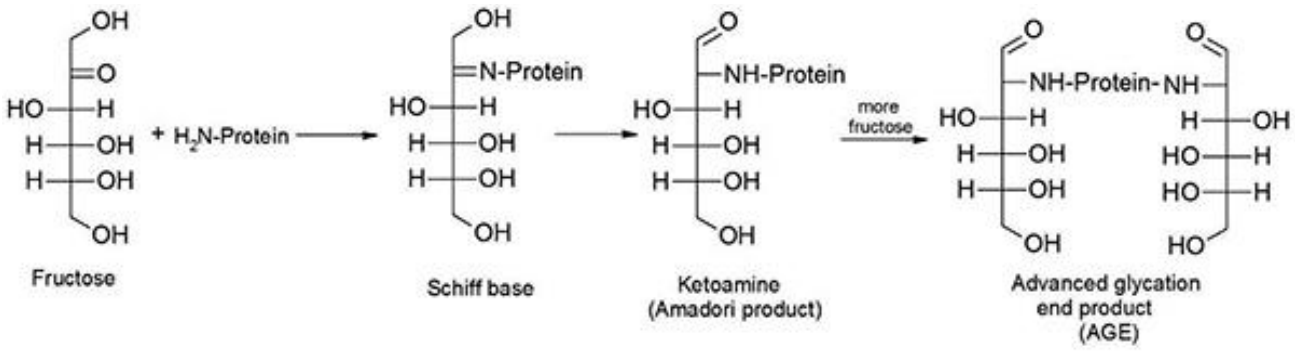


Figure 4. Advanced glycation end-products (AGE) are adducts of proteins with sugars, such as fructose and glucose. Oxidation degradation products from PUFAs (malondialdehyde) also form AGE. AGE provides a mechanistic link between high sugar and high PUFA oils in the diet to various metabolic diseases

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 (see below) 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. However, on page 9 of this publication, the following limitation was stated:

"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

Ancel 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.

REFERENCES

ALZHEIMER'S ASSOCIATION. 2017. Alzheimer's disease facts and figures. Retrieved from http://www.alz.org/documents_custom/2017-facts-and-figures.pdf on 27 March 2017.

ASSUNÇÃO ML, FERREIRA HS, DOS SANTOS AF, CABRAL CR JR, FLORÊNCIO TM. 2009. Effects of dietary coconut oil on the biochemical and anthropometric profiles of women presenting abdominal obesity. *Lipids* 44(7):593-601.

BACH AC, BABAYAN VK. 1982. Medium-chain triglycerides: an update. *Am J Clin Nutr* 36: 950-962.

BRAY GA, NIELSEN SJ, POPKIN BM. 2004. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr* 79: 537-543.

CODEX ALIMENTARIUS. 2015. Standard for named vegetable oils, Codex Stan 210-1999, amended 2015. Food and Agriculture Organization and World Health Organization.

COHEN E, CRAGG M, DE FONSEKA J, HITE A, ROSENBERG M, ZHOU B. 2015. Statistical review of US macronutrient consumption data, 1965-2011: Americans have been following dietary guidelines, coincident with the rise in obesity. *Nutrition* 31:727-732.

DANIEL CR, CROSS AJ, KOEBNICK C, SINHA R. 2011. Trends in meat consumption in the United States. *Public Health Nutr* 14(4):575-583.

DEOL P, EVANS JR, DHAHBI J, CHELLAPPA K, HAN DS, SPINDLER S, SLADEK FM. 2015. Soybean oil is more obesogenic and diabetogenic than coconut oil and fructose in mouse: Potential role for the liver. *PLoS ONE* 10(7):e0132672.

DE VOS LC, LEFRANDT JD, DULLAART RPF, ZEEBREGTS CJ, SMIT AJ. 2016. Advanced glycation end products: An emerging biomarker for adverse outcome in patients with peripheral artery disease. *Atherosclerosis* 254: 305-313.

DWYER JT, AUSMAN L, KENNEDY E. 2010. D. Mark Hegsted, Ph.D. (Biographical Article). *J Nutr* 140(8): 1402-1403.

FLORENTINO RF, AGUINALDO AR. 1987. Diet and cardiovascular disease in the Philippines. *Philipp J Coconut Studies* 13(2): 56-70.

[FAO] Food and Agriculture Organization. 2008. Fats and Fatty Acids in Human Nutrition. *Report of an Expert Consultation*. p.9.

FRANTZ ID JR., DAWSON EA, ASHMAN PL, GATEWOOD LC, BARTSCH GE, KUBA K, ELIZABETH R. BREWER ER. 1989. *Arteriosclerosis* 9:129-135.

GALANIS DJ, MCGARVEY ST, QUESTED C, SIO B, AFELE-FA'AMULI S. 1999. Dietary intake of modernizing Samoans: Implications for risk of cardiovascular disease. *J Amer Dietetic Assoc* 99(2):184-190.

GOLDIN A, BECKMAN JA, SCHMIDT AM, CREAGER MA. 2006. Advanced glycation end products. Sparking the development of diabetic vascular injury. *Circulation* 114:597-605.

GORAN MI, ULIJASZEK SJ, VENTURA EE. 2012. High fructose corn syrup and diabetes prevalence: A global perspective. *Global Public Health* 1-10.

HEGAB Z, GIBBONS S, NEYSES L, MAMAS MA. 2012. Role of advanced glycation end products in cardiovascular disease. *World J Cardiol* 4(4):90-102.

HEGSTED DM, MCGANDY RB, MYERS ML, STARE FJ. 1965. Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr* 17(5):281-295.

HEGSTED DM. 2004. Fredrick John Stare (1910-2002) (Biographical Article). *J. Nutr* 134: 1007-1009.

INDEX MUNDI. 2016. http:// www.indexmundi.com/

JOHNSTON PV. 1957. Occurrence of trans fatty acids in human tissue. *Science* 126(3276): 698-699.

KEARNS CE, SCHMIDT LA, GLANTZ SA. 2016. Sugar industry and coronary heart disease research. A historical analysis of internal industry documents. *JAMA Intern Med* E1-E6.

KEYS A. 1952. The age trend of serum concentrations of cholesterol and of Sf 10-20 ("G") substances in Adults. *J Gerontol* 7(2): 201-206.

KEYS A. 1955. Atherosclerosis and the diet. *S.A. Med J* 332-334.

KEYS A, ANDERSON JT, GRANDE F. 1957a. Prediction of serum-cholesterol responses of man to changes in fats in the diet. *Lancet* 959-965.

KEYS A, BUZINA R, GRANDE F, ANDERSON JT. 1957b. Effects of meals of different fats on blood coagulation. Circulation 15:274-279.

KEYS A. 1957c. Epidemiologic aspects of coronary artery disease (editorial). *J Chron Dis* 6(5): 552-559.

KEYS A, PARLIN RW. 1966. The Serum Cholesterol Response to Changes in Dietary Lipids. *Am J Clin Nutr* 19:175-181.

KEYS A, MENOTTI A, KARVONEN MJ, ARAVANIS C, BLACKBURN H, BUZINA R, DJORDJEVIC BS,

DONTAS AS, FIDANZA F, KEYS MH, et al. 1986. The diet and 15-year death rate in the Seven Countries Study. *Am J Epidemiol* 124(6):903-915.

KEYS A. 1971. Sucrose in the diet and coronary heart disease. *Atherosclerosis* 14(2):193–202.

KO S-Y, KO H-A, CHU K-H, SHIEH T-M, CHI T-C, CHEN H-I, CHANG W-C, CHANG S-S. 2015. The possible mechanism of Advanced Glycation End Products (AGEs) for Alzheimer's disease. *PLoS ONE* 10(11):e0143345.

KRIS-ETHERTON PM. 2010. Trans-fat and coronary heart disease. *Crit Rev Food Sci Nutr* 50:29-30.

KRITCHEVSKY D, MOYER AW, TESAR WC, LOGAN JB, BROWN RA, DAVIES MC, COX HR. 1954. Effect of cholesterol vehicle in experimental atherosclerosis. *Am J Physiol* 178:30-32.

LAWRENCE GD, MAVI A, MERAL K. 2008. Promotion by phosphate of Fe(III)- and Cu(II)-catalyzed autoxidation of fructose. *Carbohydrate Res* 343(4):626-635.

LIU T-X, NIU H-T, ZHANG S-Y. 2015. Intestinal microbiota metabolism and atherosclerosis. *Chin Med J* 128(20):2805-2811.

MATTHAN NR, AUSMAN LM, LICHTENSTEIN AH, JONES PJH. 2000. Hydrogenated fat consumption affects cholesterol synthesis in moderately hypercholesterolemic women. *J Lipid Res* 41:834-839.

MAYES PA. 1993. Intermediary metabolism of fructose. *Am J Clin Nutr* 58(suppl): 754S-765S.

MCGANDY RB, HEGSTED DM, STARE FJ. 1967a. Dietary fats, carbohydrates and atherosclerotic vascular disease. *NEJM* 277(4): 186-192.

MCGANDY RB, HEGSTED DM, STARE FJ. 1967b. Dietary fats, carbohydrates and atherosclerotic vascular disease (Concluded). *NEJM* 277(5):242-247.

MENSINK RP, KATAN MB. 1990. Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *NEJM* 323(7):439-445.

[NCI] National Cancer Institute. 2017. Cancer statistics. https://www.cancer.gov/about-cancer/understanding/statistics.

[NIDDK] National Institute of Diabetes and Digestive and Kidney Diseases. 2012. Trends in overweight and obesity among adults, United States, 1962-2010. Retrieved from https://www.niddk.nih.gov/health-information/health-statistics/Pages/overweight-obesity-statistics.aspx on 25 March 2017.

NG CH, YAO XQ, HUANG Y, CHEN Z-Y. 2008. Oxidised cholesterol is more hypercholesterolaemic and atherogenic than non-oxidised cholesterol in hamsters. *Br J Nutr* 99(4):749-755.

O'CONNOR A. 2016. A decades-old study, rediscovered, challenges advice on saturated fat. *New York Times*, April 13, 2016. http://well.blogs.nytimes.com/2016/04/13/a-decades-old-study-rediscovered-challenges-advice-on-saturated-fat/?_r=0.

PAGE IH, ALLEN EV, CHAMBERLAIN FL, KEYS A, STAMLER J, STARE FJ. 1961. Dietary fat and its relation to heart attacks and strokes. Report by the Central Committee for Medical and Community Program of the American Heart Association. Circulation 23:133-136.

PRIOR IA, DAVIDSON F, SALMOND CE, CZOCHANSKA Z. 1981. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau Island studies. Am *J Clin Nutr* 34:1552-1561.

RAMSDEN CE, ZAMORA D, MAJCHRZAK-HONG S, FAUROT KR, BROSTE SK, FRANTZ RP, DAVIS JM, RINGEL A, SUCHINDRAN CM, HIBBELN JR. 2016. Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968-73). *BMJ* 353:i1246.

SCRIMSHAW NS. 2014. D. Mark Hegsted 1914-2009. National Academy of Sciences Biographical Memoirs. http://www.nasonline.org/memoirs.

SIMOPOULOS AP. 2002. The importance of the ratio of omega-6/omega-3 essential fatty acids. *Biomed Pharmacother* 56(8):365-379.

SIMOPOULOS AP. 2008. The importance of the omega-6/omega-3 fatty acid ratio in cardiovascular disease and other chronic diseases. *Exp Biol Med* 233(6):674-688.

SIMOPOULOS AP. 2010. Genetic variants in the metabolism of omega-6 and omega-3 fatty acids: their role in the determination of nutritional requirements and chronic disease risk. *Exp Biol Med* 235: 785-795.

SUN SZ, EMPIE MW. 2012. Fructose metabolism in humans – what isotopic tracer studies tell us. *Nutr Metab* 9:89.

[USDA] U.S. Department of Agriculture, Economic Research Service. 2014. ERS *Oilseed Yearbook*.

[WHO] World Health Organization. 2003. Diet Food Supply and Obesity in the Pacific. https://apps.who.int/infobase/Comparisons.aspx.

[WHO] World Health Organization. 2015. Healthy diet Fact sheet No. 394. http://www.who.int/mediacentre/factsheets/fs394/en/.

YERUSHALMY J, HILLEBOE HE. 1957. Fat in the diet and mortality from heart disease; a methodologic note. *NY State J Med* 57(14): 2343-2354.

YUDKIN J. 1965. Letter to the editor, *BMJ* 874.

Do you have friends who would like this newsletter? If so, please feel free to share this newsletter with them.

If this newsletter was forwarded to you by a friend and you would like to subscribe, click here.