Is there a Science of Healthy Eating?

Donald Gillies*

Abstract: There is a great deal of talk about healthy eating, but also considerable differences of opinion about what constitutes healthy eating, which are illustrated by comparing the Paleo diet with the advice on healthy eating given on the UK's National Health Service website. This raises the question as to whether recommendations about healthy eating are arbitrary, or whether some of them have a genuine scientific basis. The paper argues for the second alternative. It considers the hypothesis H that a diet high in saturated fat causes atherosclerosis and argues that H is well-confirmed empirically by observational and experimental evidence and so should be considered scientific. H is indeed accepted by mainstream science. However, a strange phenomenon has emerged which could be called 'media science'. This is run by journalists rather than medical researchers, but these journalists imitate the procedures of mainstream science. They argue that H is false, and that there is no harm in eating a diet high in saturated fat. The paper gives an analysis of the work of some of these media scientists and concludes that their arguments are largely bogus.

Keywords: saturated fat, confounding, media science.

Disagreements about What Constitutes Healthy Eating

The phrase 'healthy eating' is widely used, and yet there is considerable disagreement about what actually constitutes healthy eating. This can be seen by examining some websites which purport to give a healthy diet. Let us start with thepaleodiet.com[1], which advocates what is called the Paleo diet. This is based on the argument that humans evolved during the hunting-gathering period (the paleolithic), and so we are best adapted to eating the diet which was common at that period. It is this diet, which will keep us healthy. Since

* University College London.
donald.gillies@ucl.ac.uk

1 Consulted 19 October 2022.

hunters killed many animals, it follows that meat cannot be bad for humans. Further, it is desirable to eat a large quantity of fruit, nuts and vegetables, since, for the Paleo supporters, these were gathered in the hunter-gatherer period of human evolution. However, the Paleo diet eliminates bread, pasta and rice. The argument for so doing is the following. Such carbohydrates only appeared in the neolithic period with the invention of settled agriculture, which occurred after the hunter-gatherer period, the paleolithic. So, we did not evolve to eat such things as bread, pasta and rice which therefore can only do us harm. The Paleo diet also excludes legumes, such as peas, beans, and chickpeas, presumably for the same reason. On the website (thepaleodiet.com), the Paleo Diet is described as: "Healthy eating, based on science" and the following is a quotation: "Don't Eat: Grains, including oats, pasta, and cereal; Legumes, including beans, soy & peanuts".

Let us turn to another website nhs.uk[2]. This is the website of the UK National Health Service (NHS), and, among a great deal of information about diseases and treatments, it gives some advice about healthy eating. Here are some quotations from this website: "Starchy carbohydrates should make up just over a third of the food you eat. They include potatoes, bread, rice, pasta and cereals". Clearly this completely contradicts the Paleo diet which bans all cereals, as we have seen, and also bans potatoes, though curiously it allows sweet potatoes. What then about the question of meat? The NHS website does not advocate banning meat altogether but advises cutting down the amount of meat consumed. The website states that meat contains saturated fat, and it says: "Try to cut down on your saturated fat intake". It also says: "If you eat a lot of red […] meat, it is recommended that you cut down as there is likely to be a link between red […] meat and bowel cancer". This goes against the Paleo diet, which puts no limit on the amount of meat eaten provided it comes from grass, fed animals and is not processed.

The Paleo diet bans the consumption of beans and other legumes, but the NHS website says: "Pulses, such as beans, peas and lentils, are good alternatives to meat because they are lower in fat and higher in fibre and protein, too". It actually recommends substituting meat with pulses and starchy foods in stews etc, saying: "try using smaller quantities of meat and replacing some of the meat with vegetables, pulses and starchy food in such dishes such as stews, curries and casseroles". This is clearly the opposite of the recommendations of the Paleo diet which bans pulses and starchy foods, while putting no limit on meat consumption. I have given two accounts of what constitutes healthy eating from internet websites and clearly, they differ very considerably. There are several other accounts of what constitutes healthy eating to be found.

[2] Consulted 19 October 2022.

So, what are we to make of all this? It might be concluded that what constitutes healthy eating is just a matter of opinion, and the various opinions are based on speculative theories and anecdotal evidence. However, I do not agree with such a conclusion. My own view is that some theories of healthy eating are scientific in the sense that their claims have been rigorously tested using carefully collected observations and well-designed experiments, and that these claims have been well-confirmed empirically by this evidence. In the next section, I will give an example of such a theory concerning healthy eating.

How to Avoid Atherosclerosis

My example is that of atherosclerosis (from the Greek words: athera = gruel, and sclerosis = hardening), which consists in plaques forming in the arteries. These atherosclerotic plaques have a variety of undesirable effects. They restrict the blood flow in an artery, and, when strongly developed, can lead to thrombosis, that is the formation of a blood clot which may close the artery completely. If this happens in an artery supplying blood to the brain the result is a stroke. If the arteries supplying the legs and feet are affected by atherosclerosis, the result is peripheral vascular disease. A typical symptom when blood flow is restricted without being cut off altogether is intermittent claudication, which consists of pains in the legs when walking which disappear after a short rest. The arteries are no longer able to supply sufficient oxygenated blood to fuel the muscles needed in walking. In more extreme cases, perhaps after a thrombosis, gangrene can set in.

Coronary heart disease is produced by atherosclerosis of the coronary arteries which supply the heart with blood. If these arteries become partially blocked, and blood flow to the heart is restricted, the result is angina pectoris (acute coronary syndrome). This consists of chest pains which arise from exertion but disappear with rest. It is similar to intermittent claudication but affects the heart rather than the legs. A thrombosis in one of the arteries supplying the heart leads to a myocardial infarction (or heart attack). If the blood clot closes off the blood supply from the artery, a portion of the cardiac muscle (myocardium) dies. In 25 to 35 per cent of cases, a first heart attack is fatal.

Atherosclerosis was discovered in the 19th century as a result of autopsies carried out on patients who had died from coronary heart disease and other conditions. These autopsies showed that the disease symptoms in life were associated with atherosclerotic plaques in a section of the arterial system. A further important discovery was made in 1910 when a German chemist (Windaus) showed that atherosclerotic plaques were composed of calcified connective tissue and cholesterol.

So, by the second decade of the twentieth century, atherosclerosis in its various forms had become a well-known disease. However, the causes of atherosclerosis remained obscure. Then in the 1950s, an American nutritionist, Ancel Keys (1904-2004) who was studying coronary heart disease (or CHD), came up with the hypothesis that CHD was caused by eating a diet which was high in saturated fat. It is important here to distinguish saturated fat, which is mainly found in animal fats such as meat, butter, cheese, cream, from monounsaturated fat, found in olive oil, and polyunsaturated fat found in corn oil. Ancel Keys regarded saturated fat as potentially dangerous, but he did not regard monounsaturated fat or polyunsaturated fat as dangerous. In fact, he considered them as on the whole beneficial.

Ancel Keys' work was concerned with CHD, but it largely applies to atherosclerosis in general. If Keys' hypothesis is correct, then atherosclerosis can be avoided by reducing the amount of saturated fat in one's diet. But is the underlying hypothesis H: "A diet high in saturated fat causes atherosclerosis"[3], an arbitrary conjecture or a scientific claim which has been strongly empirically corroborated by the evidence of observations and experiments? In the next section, I will argue that the second alternative is the correct one.

Mainstream Science on Saturated Fat

Ancel Keys organised a large-scale international study to test out his hypothesis H, as applied to coronary heart disease (CHD). This became known as the seven countries study. It was a 'cohort study' or 'prospective study', and so purely observational in character. 16 different cohorts were chosen – at least one in each of the seven countries. The investigations were started between 1958 and 1964, with the plan of examining the participants at the beginning and then every five years[4].

The seven countries were: Finland, Greece, Italy, Japan, the Netherlands, USA, and Yugoslavia. The cohorts chosen consisted of men aged between 40 and 59, since this was the group most at risk of CHD. In the USA, there was one large cohort of 2,571 men who were employees of the American railroad industry. The participants in the 15 remaining cohorts numbered 10,199, giving an average of 680 per cohort. The smallest of these cohorts was 504, and the largest 993.

[3] I will refer to this as 'causal hypothesis H' in what follows.

[4] By 1969, the five-year results were in, and in 1970, the results were published in Ancel Keys (ed.), *Coronary Heart Disease in Seven Countries*, "Circulation", Vol. XLI-XLII, Supplement I, 1970, pp. 1-198.

The diet of the participants was carefully recorded. Samples from the cohorts were taken, and the foods they ate in a week were noted and weighed. This was repeated at different seasons of the year. The foods consumed were analysed to find their constituents in terms of fats, carbohydrates, proteins, etc. This analysis was carried out in different ways, and it was checked that the different estimates agreed[5]. The number of those in each cohort who died of coronary heart disease (CHD) was also recorded.

At the end of the study, for each cohort, the percentage of the diet calories which came from saturated fat, on the x-axis, was plotted against the number per hundred of the cohort who had died of CHD, on the y-axis. The result (see figure 1) shows a close approximation to a linear regression model with a correlation of 0.84[6]. This evidence undoubtedly confirms the causal hypothesis H. But the evidence has weaknesses as well as strengths. Its weakness can be summed up in the well-known maxim that *correlation is not causation*. It is perfectly possible to have a very strong correlation between two variables, which are not causally connected at all. A simple example is provided by Freudenheim[7], who report a series of studies of alcoholics carried out between 1974 and 1981, which showed that the high consumption of alcohol by these individuals was accompanied by high levels of morbidity and mortality from lung cancer. So, we have a strong correlation between heavy

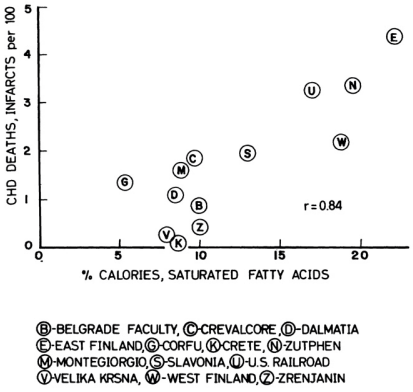

Fig. 1. Source Keys, 1970, Figure XVII.6, p. 174.

5 Id., *Coronary Heart Disease*, cit., pp. 162-9.

6 For further details about the seven countries study, see Donald Gillies, *Causality, Probability, and Medicine*, Routledge, London and New York 2019, pp. 108-119.

7 Jo L. Freudenheim, John Ritz, Stephanie A. Smith-Warner, Demetrius Albanes, Elisa V. Bandera, Piet A. van den Brandt, Graham Colditz, Diane Feskanich, R. Alexandra Goldbohm, Lisa Harnack, Anthony B. Miller, Eric Rimm, Thomas E. Rohan, Thomas A. Sellers, Jarmo Virtamo, Walter C. Willett, and David J. Hunter, *Alcohol consumption and risk of lung cancer: a pooled analysis of cohort studies*, "American Journal of Clinical Nutrition", 82, 2005, pp. 657-667.

drinking and lung cancer. However, few would accept that there is a significant causal link here. Instead, the correlation would be attributed largely to the confounding factor of smoking.

A large percentage of alcoholics are also heavy smokers, and most researchers would judge that it was the smoking of alcoholics rather than their drinking which increased the risk of lung cancer. In such an example the correlation does not show causality, because of the existence of a confounder, which, in this case, is smoking. So, the weakness of the kind of observational statistical evidence which Keys presents in his seven countries study is that it is liable to confounding. However, there are three strategies for dealing with confounding, and, in the case of our causal hypothesis H, they were all applied successfully, thereby showing that Keys' correlation really is causal in character.

The first strategy consists in considering possible confounders and then testing whether they hold or not. As far as the seven countries study is concerned, this can be most conveniently illustrated by confining ourselves to the results from just two countries, namely Japan and the USA There were two cohorts from Japan. One was from the farming village of Tanushimaru and had 509 members. The other was from the fishing village of Ushibuka and had 504 members. There was only one large cohort from the USA consisting of 2,571 railroad men. The various statistics for the 5-year period were as follows. The percentage of calories from saturated fats was 3% for Japan and 17-18% for USA The USA figure was thus between 5.7 and 6 times that of Japan[8]. Among the Japanese cohorts there were 5 deaths from CHD, giving a death rate per 1,000 of 4.9. In the USA cohort, there were 62 deaths from CHD, giving a death rate per 1,000 of 24. The U.S.A death rate from CHD was thus 4.9 times that of Japan.

These figures certainly suggest a causal link between dietary saturated fat and coronary heart disease, but could there be an alternative explanation? The USA railroad men were all white (Caucasian). Could the Japanese have a group of genes which protected them from CHD, and which was lacking among Caucasians? The lower death rates from CHD among the Japanese might be nothing to do with their diet and be the result rather of their genes. To put it another way, there might be a genetic confounder of the hypothetical causal link between dietary saturated fat and CHD.

In fact, it was quite easy in the CHD case to test out the hypothesis of a genetic confounder against data, and this was done by Keys and his colleagues. If there is a genetic confounder, then groups of Japanese who start eating a typical American diet will have an incidence of CHD much lower than that of white Americans, because they will be protected by their anti-CHD genes.

[8] A. Keys (ed.), *Coronary Heart Disease*, cit. p. 168.

If on the other hand, there is no genetic confounder, then groups of Japanese who eat a typical American diet will have an incidence of CHD much the same as that of white Americans. All that needs to be done is to compare data on incidence of CHD among Japanese living in Japan and eating the traditional Japanese diet with that among Japanese who have emigrated to the USA and started eating a typical American diet. In 1958, Keys and colleagues carried out an investigation of this sort by comparing Japanese in Japan with Japanese in Hawaii and in California[9]. The overall finding was that death rate from CHD among the Japanese in California was more or less the same as that of white Californians, and much higher than that of Japanese in Japan. The death rate from CHD for Japanese in Hawaii was intermediate between the two values. The observations indicated that as Japanese, who had emigrated, became increasingly Americanised and adopted the American diet, so their death rate from CHD rose to become more or less the same as that of white Americans. There was no indication that the Japanese had any special genetic protection against CHD. These findings were confirmed by a later investigation along similar lines[10]. In fact, the hypothesis that there might be a genetic confounder was strongly disconfirmed.

The second strategy for dealing with confounding is the use of randomization. Suppose that A is strongly correlated to B, and we think A may cause B, but are doubtful whether there may instead be a confounder C which causes B. The first strategy for dealing with this doubt was to think of all the possible confounders C that we can, and then test them out against the data to see if we can refute the hypothesis that C causes B. If we can indeed refute all such hypotheses, this provides good evidence that A causes B. However, it could now be objected that this is all very well, but that there may still be a confounder we have not thought of. Randomization provides a technique for dealing with unknown confounders. Suppose there is a confounder C, which, unknown to us, causes both A and B, and that A does not really have any causal influence on B. In our trial, let us divide the participants randomly by tossing a fair coin, into the control group, who do not receive A, and the intervention group, who do receive A. Then if a particular participant has C, he or she is just as likely to be in the control group as the intervention group. Since C is a cause of B, and A is not a cause of B, then the difference between

[9] Ancel Keys, Noboru Kimura, Akira Kusukawa, Brian Bronte-Stewart, Nils Larsen, and Margaret Keys, *Lessons from Serum Cholesterol Studies in Japan, Hawaii and Los Angeles*, "Annals of Internal Medicine", 48, 1958, pp. 83-94.

[10] Thomas L. Robertson, Hiroo Kato, George G. Rhoads, Abraham Kagan, Michael Marmot, S. Leonard Syme, Tavia Gordon, Robert M. Worth, Joseph L. Belsky, Donald S. Dock, Michihiro Miyanishi, and Sadahisa Kawamoto, *Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii and California. Incidence of Myocardial Infarction and Death from Coronary Heart Disease*, "The American Journal of Cardiology", 39, 1977, pp. 239-243.

the intervention group and the control group will disappear. If there continues to be a difference between the control group and the intervention group, we can conclude that the intervention variable (A) really is a cause and that confounders have been dealt with. Of course, this argument is not entirely conclusive, since random choice by tossing a fair coin only produces an equal distribution in the limit. In a particular trial, an unbalanced distribution can occur. Despite this problem, randomization still remains a useful technique for the reasons given earlier in this paragraph, and it is widely employed by medical statisticians as a strategy for dealing with confounding.

We can illustrate these points by considering a randomized controlled trial designed to investigate the relationship between the dietary intake of saturated versus unsaturated fat and the development of atherosclerosis. This clinical trial was carried out by Seymour Dayton and his colleagues as follows:

> Male veterans, aged 54-88 years (mean 65.5 years), living in the domiciliary unit of the Los Angeles Veterans Administration Center, were asked to volunteer for this study. A total of 1095 men volunteered, but exclusions and dropouts during the preliminary phases reduced this number so that 846 men were ultimately randomised. Of these, 422 received the conventional control diet, and 424 the experimental diet[11].

The control diet was a conventional American diet containing 40% fat calories, mostly of animal origin. The experimental diet was designed to be as similar to the conventional diet as possible, except that about two thirds of the animal fat calories were replaced by vegetable oils, such as corn, cottonseed, safflower, and soybean. In effect, the total fat content of the diet was about the same, but two thirds of the saturated fat had been replaced by unsaturated fat.

In addition to being randomized, the trial was double blind. The participants did not know whether they were having the experimental diet or the control diet. They were examined at regular intervals, and the values of various variables recorded. However, the physicians carrying out these examinations were also 'blinded' by not being told which diet the participant was on.

Recruitment started in the summer of 1959, and the trial was concluded with a final re-examination of a participant eight years after he had been recruited. No further recordings for the purposes of the trial were made after 15 January 1968. A feature of this trial was that it recorded all atherosclerotic events and not just coronary heart disease. The events recorded were those established by hard criteria, and, in addition to myocardial infarction, and sudden death due to coronary heart disease, they included definite cerebral

[11] Seymour Dayton, Morton Lee Pearce, Hubert Goldman, Alan Harnish, David Plotkin, Martin Shickman, Mark Winfield, Albert Zager, and Wilfrid Dixon, *Controlled Trial for a Diet High in Unsaturated Fat for Prevention of Atherosclerotic Complications*, "The Lancet", 2, 1968, p. 1060.

infarction (i.e. stroke), ruptured aneurysm, and even amputation necessitated by atherosclerosis of arteries in the leg. This choice of outcomes is more logical than focussing exclusively on coronary heart disease, since the other outcomes have the same underlying cause, namely atherosclerosis. The results were as follows. In the control group 70 men died of an atherosclerotic event, as opposed to 48 in the experimental group. The difference was significant at the 5% level[12]. In addition, there were some nonfatal atherosclerotic events established by hard criteria. 96 men in the control group were affected by a fatal or nonfatal atherosclerotic event, as opposed to 66 in the experimental group. The difference was significant at the 1% level.

These results clearly give empirical confirmation to the causal hypothesis H. The seven countries study was observational in character, and so strictly yielded only correlation not causation. The study of the Los Angeles veterans, by contrast, was a randomized, and even double blind, clinical trial, and so had a built-in remedy against confounding. This is the great strength of the Los Angeles veterans trial. In favour of the seven countries study, however, it should be added that Keys and colleagues did consider at least one possible confounder (racial/genetic factors) and showed that it was refuted by further observational evidence.

If we take the evidence of the veterans study together with that of the seven countries study, then the weakness of one piece of evidence is to a large extent cancelled out by the strength of the other. The weakness of the seven countries study was concerned with the problem of confounding, while the randomization used in the veterans study is one of the standard ways of dealing with the problem. The weaknesses of the veterans trial were those characteristic of clinical trials in general, namely sample limitations. The sample was relatively small, and the participants were too elderly to be representative of the target population. However, both these weaknesses were overcome in the seven countries study. The results of the two studies empirically confirmed broadly the same causal hypothesis. So, taken together, they give strong evidence for this hypothesis. This is an instance of the principle that combining evidence of different types all of which support a given hypothesis, greatly strengthens the confirmation of that hypothesis. This could be called the principle of *strength through combining*[13].

[12] Seymour Dayton, Morton Lee Pearce, Sam Hashimoto, Wilfrid Dixon, and Uwamie Tomiyasu, *A Controlled Clinical Trial of a Diet High in Unsaturated Fat in Preventing Complications of Atherosclerosis*, "Circulation", 40(1), 1969, Supplement II, pp. 26-27.

[13] Phyllis Illari, *Mechanistic Evidence: Disambiguating the Russo-Williamson Thesis*, "International Studies in the Philosophy of Science", 25(2), 2011, pp. 139-157. For further details see D. Gillies, *Causality,* cit., pp. 129-132.

The third strategy for dealing with confounding is a consideration of mechanisms. Let us take our usual case in which A and B are strongly correlated, and we are wondering whether this means that A causes B, or whether the correlation is due to confounding. Suppose further that B is some physical illness. Then it may be possible to discover a bodily mechanism M which links A to B. Such mechanisms are usually physiological/biochemical processes which go on in the body. Their existence and nature can be confirmed, or disconfirmed, by laboratory experiments on animals, tissues, cells, and so on. Suppose that the existence of a mechanism M linking A and B is established in this way. Such a result provides some evidence that the correlation is indeed a causal connection, and not the result of confounding. This strategy works very well in the case of causal hypothesis H. In fact, a mechanism has been established by which a diet high in saturated fat leads to atherosclerosis. It can be stated as follows:

1. Diet high in saturated fats *causes.*
2. Raised level of LDL cholesterol in the blood *causes.*
3. Infiltration of some parts of the arterial wall by lipids (namely cholesterol esters) *causes.*
4. Lipids to be absorbed by macrophages, which become foam cells *causes.*
5. Full development of atherosclerotic plaques.

The step from 1 to 2 is very easy to establish and has been confirmed by a variety of observational studies and randomized controlled trials. The steps from 2 to 5 were confirmed by autopsies on humans and by experimental work on animals. The first such experimental work was carried out by a Russian scientist, Anitschkow, who found a method of raising the LDL cholesterol level of rabbits by feeding them a special diet. These rabbits then developed atherosclerosis which is unknown in rabbits eating their usual diet. It could be objected that the experimental production of atherosclerosis in rabbits might be different from what occurs in humans, but Anitschkow replied that the two processes are very similar[14]. The atherosclerotic plaques develop in the experimental rabbits in the same order and in the same places in the arterial system as those in humans which are revealed by autopsies[15]. Taking all this together, we can see that there is a mass of evidence of different kinds which support causal hypothesis H. Applying the principle of strength through combining, we have to conclude that H is very well confirmed empirically and so should be accepted by scientific medicine[16]. Indeed, it has been accepted by

[14] Nikolaj Anitschkow, *Experimental Arteriosclerosis in Animals*, In E. V. Cowdry (ed.), *Arteriosclerosis*, Macmillan, New York 1933, pp. 271-322 (in particular p. 305).

[15] For further details of Anitschkow's work see Gillies, *Causality,* cit., pp. 95-101.

[16] An anonymous referee pointed out, however, that the studies by A. Keys and S. Dayton are

the medical mainstream. We saw this from the NHS website which says: "Try to cut down on your saturated fat intake". This is confirmed by Mike Laker. The idea of this series is to give the standard mainstream views regarding a variety of illnesses. Laker writes: "The main objective of changing your diet is to reduce your saturated fat intake"[17]. Yet despite this consensus of the medical mainstream, not everyone accepts causal hypothesis H, as we will see in the next section.

Media Science on Saturated Fat

The main opponents of the views of the medical mainstream on nutrition are journalists rather than medical researchers. The two most prominent are Gary Taubes, and his follower Nina Teicholz, who published in 2014 a book of 479 pages (*The Big Fat Surprise*) most of which are taken up with criticizing the work of Ancel Keys et al and proposing alternative hypotheses. It is curious that this opposition comes from journalists rather than medical researchers, but Nina Teicholz rather ingeniously uses some of Kuhn's philosophy to explain this. She argues that the ideas of Keys and colleagues on cholesterol constitute the dominant paradigm in this branch of medicine, and so are accepted by nearly all medical researchers who are normal scientists. What is needed however is a "paradigm shift on cholesterol". Moreover Teicholz says: "given the stranglehold that Keys's ideas have held on nutrition researchers for so many decades, it is perhaps inevitable that an alternative hypothesis had to come from an outsider"[18].

So, is there going to be a Kuhnian scientific revolution in nutrition started by two journalists? I will briefly consider this question by examining the arguments presented by Nina Teicholz in her 2014 book. It should be added that arguments of this type are often to be found in newspaper and magazine articles and on the internet. Let me begin by considering a phrase often used by Nina Teicholz and other journalists and bloggers, namely 'low-fat diet'. Thus, for example, Teicholz claims:

of men only, so that strictly we should not consider the results as necessarily applying to women. As we might expect, subsequent studies have shown that causal hypothesis H applies to women as well, but there is in fact a difference between the sexes. Before the menopause, women have lower cholesterol levels than men of the same age eating a similar diet, and correspondingly lower rates of heart disease. Some researchers think that is part of the explanation of why women on average live longer than men.

17 Mike Laker, *Understanding Cholesterol*, Family Doctor Publications in association with the British Medical Association, 2012, p. 85.

18 Nina Teicholz, *The Big Fat Lie*, Scribe, London and Melbourne 2014, pp. 315-316.

A review in 2008 of all studies of the low-fat diet by the United Nation's Food and Agriculture Organization concluded that there is "no probable or convincing evidence" that a high level of fat in the diet causes heart disease …[19]

An unwary reader might suppose that this contradicts Ancel Keys' ideas, but such is not the case. Ancel Keys not only never claimed that a high level of fat in the diet caused heart disease, but he also pointed out that the cohort in his seven countries' study which had the lowest level of heart disease had the highest percentage of dietary calories in the form of fat. This was the cohort from Crete, 40% of whose dietary calories came from fat[20]. The cohort which had the highest level of coronary heart disease was that from East Finland, 39% of whose dietary calories came from fat. What is crucial is not the intake of fat in general, but the intake of *saturated fat*. The Cretans' fat intake came mainly from olive oil, which is monounsaturated fat and does not raise LDL level in the blood. The Cretans' intake of saturated fat was only 8%. By contrast, the fat intake of the East Finland cohort came mainly from butter, cheese, and meat, and their intake of saturated fat was 22% – the highest of any of the cohorts. The East Finland cohort also had the highest blood cholesterol level, and the highest rate of CHD. The important point is that Ancel Keys did not advocate a low-fat diet, but a diet low in saturated fat.

There are also some confusions concerning the Atkins diet which Teicholz discusses from p. 287 on. This diet consists mainly of animal foods such as meat, cheese and eggs, while the intake of bread and other carbohydrates is severely restricted. It turns out that this diet is very effective in achieving weight loss, the reason being that after a few pork chops, the desire for more pork chops declines. So, eating too much on a meat-centred diet is nearly impossible[21]. The effectiveness of the Atkins diet in producing weight loss might seem to contradict Keys' ideas, but really it does not. Keys stresses that it is possible to be slim and fit on a diet which is high in saturated fat and gives the East Finland cohort as an example of this. The problem is that someone can be slim and fit and yet be developing atherosclerotic plaques in their arteries. Thus, the criticism of the Atkins diet, which would be made by those supporting the ideas of Ancel Keys and colleagues is that, although it may be effective in reducing weight, it is likely at the same time to increase LDL level in the blood, and so increase the risk of CHD and other atherosclerotic events. Teicholz discusses an Israeli study conducted in 2008[22]. This compared the effects of three different diets, one of which was assigned to each member of a group of 322 moderately obese middle-aged people, mostly

19 Ivi, p. 172.
20 A. Keys (ed.), *Coronary Heart Disease*, p. 168.
21 N. Teicholz, *The Big Fat Lie*, cit., p. 293.
22 Ivi, pp. 213-214.

men. One of these diets was the Atkins diet, while another was the Mediterranean diet. Those on the Mediterranean diet lost 10 pounds, while those on the Atkins diet lost 12 pounds. Teicholz comments: "Only LDL-cholesterol looked better for Mediterranean dieters"[23]. In other words, the result is just what Ancel Keys et al. would have predicted. The Atkins dieters lost more weight, but their LDL cholesterol level was higher than that of the Mediterranean dieters. As regards the low-fat and Atkins diets, Teicholz may appear to contradict Ancel Keys' ideas, but does not really do so. In other passages of her book, however, she does indeed put forward claims which contradict those of Keys.

> Eat butter; drink milk whole, and feed it to the whole family. Stock up on creamy cheeses, offal, and sausage, and yes, bacon. None of these foods have been demonstrated to cause obesity, diabetes, or heart disease[24].

Even this passage does not engage very directly with Ancel Keys. Keys writes little about obesity (which was not a problem in his day), or diabetes, and does not put forward any hypotheses about the causes of obesity or diabetes. Moreover, he is careful to point out that there are several different types of heart disease, of which coronary heart disease is only one, albeit the largest category. Still, the passage from Teicholz can be taken as contradicting Keys' claim that a diet high in saturated fat causes coronary heart disease. Why therefore does Teicholz reject the evidence which Keys puts forward for his claim?

Teicholz begins by casting doubt on the accuracy of the data which Keys and his colleagues collected in the seven countries study. They took samples of the diet of each of the cohorts at different seasons and averaged the results. However, in the case of Crete one of the three samples was in Lent, when meat and other animal products were forbidden foods[25]. This sample would therefore have underestimated the amount of saturated fat consumed by the Cretans. Keys mentions this point, but thinks it is not a very serious one since, so he claims, Lent was not very strictly observed in Crete. Of course, obtaining accurate figures on diet is difficult, and problems like that of Lent may introduce some errors. However, given the careful procedures followed by Keys and his fellow researchers, these errors are unlikely to have distorted the general picture to any great degree. Teicholz now goes on to say:

> […] there was also a huge structural limitation to the Seven Countries study; it was an epidemiological investigation and therefore could show only an association, not causation[26].

[23] Ivi, p. 214.
[24] Ivi, p. 335.
[25] Ivi, p. 40.
[26] Ivi, p. 42.

It would be more accurate to speak here of an "observational epidemiological investigation", but with this proviso, what Teicholz says here is quite correct. The correlations found by Keys may not be causal in character because of confounders. This problem has been discussed earlier in the present paper. Teichholz suggests that there may be an alternative explanation, namely sugar. Despite her earlier scepticism about the accuracy of the data produced by the seven countries study, she uses some of this data to argue for this alternative explanation.

> In 1999, when the Seven Countries study's lead Italian researcher, Alessandro Menotti, went back twenty-five years later and looked at data from the study's 12,770 subjects, he noticed an interesting fact: the category of foods that best correlated with coronary mortality was sweets. By "sweets," he meant sugar products and pastries, which had a correlation coefficient with coronary mortality of 0.821…[27]

Here we do have a striking correlation. So, the next step of course is to consider whether this is causal in character, or whether it is confounded by some other variable. Unfortunately, however, Teicholz does not take this step, and nowhere in her book does she consider whether there might be confounders of this correlation of 0.821. I will now undertake this task which she has omitted.

Might this correlation be at least partially confounded by saturated fat? "Sweets" include pastries, and these usually contain a lot of saturated fat. So at least part of the correlation between sweets and coronary mortality might be explained by the intake of saturated fat consequent on eating many types of sweet. It would be better therefore to consider the correlation between sugar intake and coronary mortality. Fortunately, Menotti and colleagues provide also the correlation between sugar and coronary heart disease, which was 0.600[28]. Although lower than 0.821, this is still positive and quite high which could indicate a causal connection, but once again it could be confounded by saturated fat intake because sugar consumption was higher in countries like the USA and Northern Europe where consumption of saturated fat was also higher. Menotti and colleagues argue that confounding of this type may have taken place in another case. There is one curious feature of their results: bread (-0.001) and cereals (-0.305) are both negatively correlated with coronary mortality, but potatoes (0.464) are positively correlated[29]. This difference is surprising since potatoes seem quite similar from a dietary point of view to bread and cereals. Menotti and colleagues claim this about the matter:

[27] *Ibidem.*

[28] Alessandro Menotti, Daan Kromhout, Henry Blackburn, Flaminio Fidanza, Ratko Buzina, and Aulikki Nissinen, *Food intake patterns and 25-year mortality from coronary heart disease: Cross-cultural correlations in the Seven Countries Study*, "European Journal of Epidemiology", 15, 1999, p. 509.

[29] *Ibidem.*

An exception is the consumption of potatoes which is highly positively correlated (univariately) to CHD, likely because their highest consumption is in Finland and The Netherlands where potatoes are accompanied by the highest intake of animal and mainly dairy products[30].

It is thus possible that the positive correlation between sugar intake and coronary mortality is confounded by saturated fat intake. The objection might, however, be raised that the situation could be the other way round, that is to say that the correlations between saturated fat intake and coronary heart disease are confounded by sugar. This situation shows the advantage of applying not just the first, but all three of the strategies for dealing with confounding which were described above. The use of randomization provides evidence against the existence of confounders not explicitly considered, and hence against sugar as a possible confounder. The mechanism linking high LDL levels in the blood to the formation of atherosclerotic plaques does not apply in the case of sugar, since sugar intake does not raise the level of LDL in the blood. So, the second and third strategies provide good evidence that sugar is not a confounder of the correlation between saturated fat and coronary heart disease. Teicholz does not consider sugar the sole cause of CHD. Her hypothesis is that it is sugar in conjunction with refined carbohydrates.

Recent scientific research and the historical record all lead to the conclusion that the consumption of refined carbohydrates leads to a higher risk of obesity, heart disease, and diabetes[31].

Unfortunately, as we have seen, the data in Menotti and colleagues show that consumption of bread and cereals is negatively correlated with coronary heart disease. This hardly lends much support to Teicholz' hypothesis. Teicholz might reply that much of the carbohydrates consumed by participants in the seven countries study was unrefined. This could be true of the Cretans, but certainly not of the Japanese who refined their rice in the period under consideration, and consumed a great deal of it, while having one of the lowest rates of coronary heart disease in the world. Teicholz is rather selective in the data she considers. While citing a correlation from Menotti and colleagues to support her case, she does not mention that the highest correlation on their list is between butter and coronary mortality at 0.887. Despite this, she urges her readers to "eat butter". Teicholz says: "Only carbohydrates have been shown, in clinical experiments, to be the likely principal cause of obesity, heart disease and diabetes"[32].

30 Ivi, p. 511.
31 N. Teicholz, *The Big Fat Lie*, cit., p. 335.
32 *Ibidem*.

This is contradicted by the clinical experiment of Dayton and colleagues which was described above. Here the two diets were identical as far as the intake of sugar and refined carbohydrates were concerned. Yet those who ate the diet with more saturated fat had higher rates of atherosclerosis and coronary heart disease.

We can now sum up as follows. Nina Teicholz, following Gary Taubes, does formulate a causal hypothesis which differs from that of Ancel Keys and colleagues. It is that sugar and refined carbohydrates, and not saturated fat, are the principal cause of coronary heart disease. However, the evidence she presents for this alternative hypothesis is not very convincing. It consists of some positive correlations, but she never considers whether these arise from confounding. In fact, they can all be explained quite plausibly as arising from confounders. Moreover, her selection of correlations to consider is very selective. She gives those, which support her case, but does not mention others, sometimes in the same paper, which go against her case. She claims that her hypothesis is the only one confirmed by clinical experiments, whereas at least one well-known clinical experiment gives strong evidence against it.

Perhaps for these reasons, Nina Teicholz' book has not found many supporters among medical researchers, and there is no sign of a scientific revolution beginning in the field of nutrition. Instead, we have a curious phenomenon, namely the development among journalists of a kind of alternative science based on a paradigm for nutrition different from that accepted by mainstream medical science. An example of this is Alice Hancock's 2019 article *Praise the lard*. Apparently, lard is becoming a fashionable food among celebrity chefs, and Hancock defends lard consumption against its critics: "And until the 1970s, lard was a regular part of the British diet. The average consumption was 55g per week in 1974 […] Today it is less than 5g. The trend is similar in the US"[33]. This would naturally seem a good thing for a follower of mainstream medical opinion on diet, but not for Hancock, who continues:

> Why did it all but vanish from our cupboards? Lard and lardo are victims of a fear of fat that started spreading in the 1950s. According to Nina Teicholz, who spent nine years investigating American dietary science for her book *The Big Fat Surprise*, it can be credited to one man: Ancel Benjamin Keys[34].

Hancock then summarises Teicholz's critique of Keys, concluding: "But the science is now being revised"[35]. The science is certainly being revised but by journalists rather than scientists. A journalist (Hancock) supports

[33] Alice Hancock, *Praise the lard*, "Financial Times Magazine", 12/13, 2019, pp. 42-43.
[34] *Ibidem*.
[35] *Ibidem*.

her defence of lard by citing another journalist (Teicholz), just as a typical research scientist might cite the work of another research scientist. So, the media are producing not just alternative facts, but also alternative science, which could be called 'media science'.

Conclusion

My conclusion is that there is a science of healthy eating. This contains results which are very well confirmed by carefully collected observations and well-designed experiments. It gives recommendations as to what diets are most likely to improve the general level of health, and these recommendations would have a very beneficial effect if they were generally adopted. Unfortunately, there is also a media science which advocates views opposed to those accepted by mainstream science. This media science imitates superficially the practices of mainstream science, but a closer inspection reveals that its arguments are largely bogus. Nonetheless because of its wide circulation, media science almost certainly has a considerable influence and one that is harmful as far of the health of the population is concerned.