# The 2012 University of Cape Town Faculty of Health Sciences centenary debate

## "Cholesterol is not an important risk factor for heart disease, and the current dietary recommendations do more harm than good"

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**Keywords:** diet heart hypothesis, lipid hypothesis, Ancel Keys, carbohydrate resistance, low-carbohydrate diet, Woman's Health Initiative, cholesterol, ancestral diet, dietary guidelines

**Abstract**

Our human ancestors thrived on a diet high in fat and protein of animal or fish origin for at least 2.5 million years. Foods with a high-energy content and nutritional density were required for the development of the large, energy-expensive human brain. A reduction in human height and deterioration in our health followed the introduction of agriculture 2 000-12 000 years ago. In 1977, the United States Department of Agriculture (USDA) introduced novel dietary guidelines based on an untested hypothesis of Keys that dietary fat, especially of animal origin, increases the blood cholesterol concentration, "clogging" the coronary arteries and causing heart attacks, i.e. the diet-heart hypothesis.

Ⓟ **Peer reviewed. (Submitted: 2014-12-19. Accepted: 2015-03-07.) © SAJCN** **S Afr J Clin Nutr 2015;28(1):19-33**

Here, I use five key arguments to show that those guidelines represent the single greatest error in the long history of medicine:

- Economic considerations drove the adoption of the 1977 USDA dietary guidelines in the absence of proper scientific proof.
- Within five years of their adoption, the rates of type 2 diabetes mellitus and obesity increased explosively, especially in the USA, subsequently spreading across the globe.
- The presence of insulin resistance (IR) explains why large numbers of persons in predisposed populations develop obesity and type 2 diabetes mellitus when following the high-carbohydrate, low-fat (HCLF) diet advocated by the USDA dietary guidelines.
- A low-carbohydrate, high-fat (LCHF) diet reverses all known coronary risk factors in persons with IR, whereas the HCLF diet may worsen many of those factors.
- The multi-million dollar 48 835 persons Woman's Health Initiative Randomized Controlled Dietary Modification Trial (WHIRCDMT), of which Rossouw was project leader, shows that the USDA dietary guidelines are associated with accelerated disease progression in persons with either established heart disease or diabetes. That study does not support Keys' diet-heart hypothesis, of which Rossouw continues to be a staunch advocate.

This paper shows why "cholesterol" is not an important risk factor for heart disease, and why the current dietary recommendations that promote a high-carbohydrate and low-fat intake, aimed at reducing blood cholesterol blood concentrations, raise blood glucose and insulin concentrations at the same time and stimulating hunger, have caused the global epidemic of obesity and type 2 diabetes mellitus that will bankrupt the world's medical services within the next two decades. Seldom have economically-driven "good" intentions produced such calamitous outcomes.

## Introduction

Our human ancestors evolved from the tiny *Australopithecus africanus* (~ 1 m tall, weighing 30 kg) to the substantially taller and heavier modern *Homo sapiens* over a period of 3.5 million years. This change occurred as hominins became more successful at increasing the quality of the foods they ate, changing from a predominantly vegetarian diet to one containing an increasing amount of animal fat and protein.[1-3] The greater consumption of meat occurred as early hominins became the most effective persistence hunters on the planet, able to run large antelope to their exhaustion within 4-6 hours in extreme heat.[4-6] The discovery of fire and the development of cooking[7] approximately 1.8 million years ago increased the energy delivery from meat, roots and shoots, reducing the time humans spent chewing their food. Stone-tipped throwing spears were added approximately 500 000 years ago,[8] further increasing our human ancestors' ability to capture large, fat-filled animals, including elephants,[9] and in Africa, rhinoceros and hippopotamus.

By the mid 1800s, the Plains Indians of North America, who existed on a diet of bison and little else, were the tallest[10] and perhaps the healthiest of all the peoples then populating the earth. In the 1830s, Catlin[11] travelled west of the Mississippi River, and painted hundreds of Plains Indians, including Black Dog and Tal-lee, two

Osage warriors, who ate mostly buffalo meat and were both over 1.98 m tall. The arrival of avaricious Europeans, disconnected from the land, foretold the massacre of 60 million bison and the demise of the Plains Indians' health. Forced to eat the standard American diet, the modern descendants of the Plains Indians are now among the least healthy populations in North America.[12] Annually, millions of dollars of public monies are spent trying to "discover" why these first peoples of North America are so unhealthy. Genius is not required to solve that particular riddle.

Xhosa- and Zulu-speaking South Africans experienced a not dissimilar fate. In 1896, the Rhinderpest virus decimated the cattle herds of East and South Africa, forcing our indigenous peoples to migrate to the cities, where they first encountered the standard American diet of highly processed foods, white flour, refined carbohydrates, sugar and processed "vegetable" (actually seed) oils. There was an epidemic increase in obesity and diabetes within 20 years.[13-15] Other immigrant populations have shown an identical response.[16,17] The increase in diabetes and ischaemic heart disease in Yemenite Jews settling in Israel is associated with an increased intake of dietary sugar and polyunsaturated, not saturated, fat, the so-called Israeli Paradox.[18]

The most recent assault on global health began in the 1950s, largely as a result of the determined endeavours of biochemist, Keys, aided after 1972 by USA President Nixon, that ultimately led to the 1977 USDA dietary guidelines. Political and economic forces, not science, drove the adoption of those dietary guidelines.[19-21] Since those forces will continue to intensify in the future, there is little hope that anything short of a people-led uprising will save us from the obesity and type 2 diabetes mellitus tsunami that those forces have unleashed.

I present five topics that explain the abysmal science and weak logic that has produced this predictable outcome.

**Economic considerations drove the adoption of the current dietary guidelines in the absence of any scientific proof**

*Keys and the origins of the diet-heart hypothesis*

The theory that fat in the diet raises the cholesterol blood concentration, which then causes coronary atherosclerosis, leading to coronary heart disease (CHD), is known as the diet-heart hypothesis. (The lipid hypothesis excludes the dietary component, and postulates only that elevated cholesterol blood concentrations cause heart disease. Therefore, they are separate hypotheses.) The diet-heart hypothesis owes its origin to the single-minded vigour of one American biochemist, Keys. In 1953, Keys wrote the following:[22]

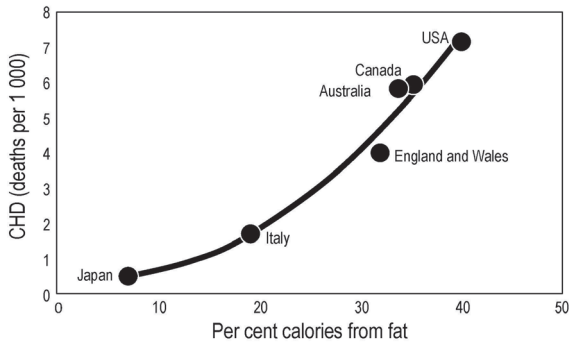
- "It is a fact that compared with healthy people of the same age, patients with definite angina pectoris or who those have survived a myocardial infarction tend to have blood serum characterised by high cholesterol and certain lipoprotein concentrations, a high cholesterol to phospholipid ratio, and a larger proportion of the total cholesterol in the beta 1-lipoprotein fraction.
- It is a fact that, on average, persons afflicted with diabetes, myxoedema and nephrosis tend to have high cholesterol and the other serum peculiarities mentioned. There is a high incidence of atherosclerosis and degenerative heart disease in those patients.
- It is a fact that in animal experiments, those measures, such as high-cholesterol diets and thyroid suppression, which produce high levels of cholesterol and allied substances in the serum, are also productive of atherosclerosis.
- It is a fact that a major characteristic of the atherosclerotic plaque is the presence of abnormal amounts of cholesterol in that artery. The atherosclerotic plaque consists of 40-70% cholesterol. It is extremely probable that most or all of this cholesterol is derived from the blood".[22]

In his paper, Keys also published the iconic figure that would define the future of the debate. He showed an apparently linear relationship between the (supposed) fat content of the diet and CHD rates in six countries (Figure 1). He concluded that this association proved that by raising blood cholesterol concentrations, dietary fat was the direct cause of CHD. So iconic is that paper, that 61 years after its publication, Keys' unproven hypothesis still forms the basis for the teaching of cardiology in the University of Cape Town's Faculty of Health Sciences.

Hence, a recent publication from the Faculty of Health Sciences at the University of Cape Town begins with the statement: "Their publication followed closely the 1957 Seven Countries Study by Keys, which established unequivocally the pathophysiological role of dietary saturated fats acting through serum cholesterol concentrations in the causation of atherosclerotic vascular disease".[23]

There are four substantive problems with Keys' "unequivocal proof".

Firstly, since those selected data provided the best visual representation of his theory, Keys included the data from only six of the 22 countries on which he had information. Secondly, he (and many since) failed to understand that the simple association of two observations does not prove causation.[21] Causation can only be proved by randomised controlled clinical trials (RCTs) in which all variables, except the one of interest, are held constant. Keys reported on observational studies, not RCTs, on the diet-heart hypothesis




CHD: coronary heart disease

**Figure 1:** The iconic figure of Keys showing an association between dietary fat intake and the incidence of coronary heart disease in six selected countries[22]

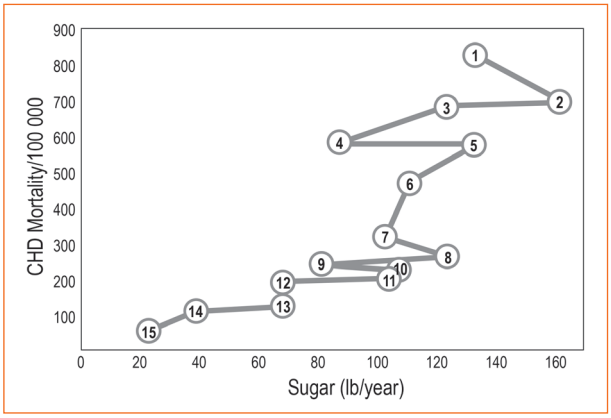
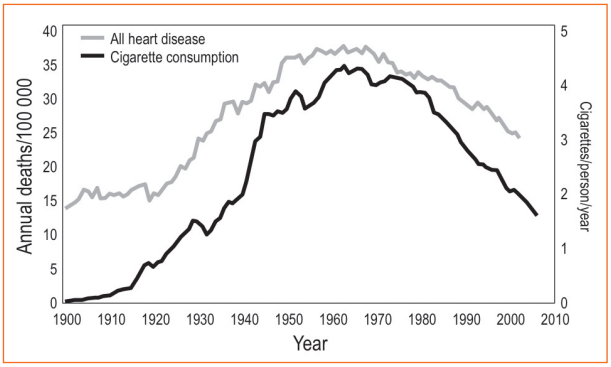



**Figure 2:** The relationship between sugar intake and coronary heart disease rates in 15 countries[25]




Source: Data from US National Vital Statistics System and the Centers for Disease Control and Prevention

**Figure 3:** Increases and subsequent falls in the rates of smoking and coronary heart disease are linked by time in the USA[29]

in his career. As a result, he could never prove that hypothesis "unequivocally". Thirdly, he spent much of his life defending his theory against the criticism that any number of confounding variables could also explain the associational relationships that he considered to be causal. Fourthly, his dubious research methods, exposed only recently, further undermine the likely validity of this hypothesis.[20] For example, Keys studied populations that had yet to recover from the economic hardships of World War II. He included Greek Orthodox populations during Lent when they abstained from all foods of animal origin. Also, he did not consider the potential health benefits of fasting, and most troublingly is that his conclusions on what constitutes the "Mediterranean diet" were based on an analysis of the dietary habits of just 33 Cretan men, and did not produce any consistent results.

Yerushalmy and Hilleboe[24] were the first to publish a careful rebuttal of Keys' associational studies. From the available data for all 22 countries, including the 16 ignored by Keys, they investigated a wide range of possible associational relationships, finally concluding that "the evidence from 22 countries, for which data are available, indicates that the association between the percentage of fat calories available for consumption in the national diets and mortality from arteriosclerotic and degenerative heart disease is not valid. The association is specific, neither for dietary fat nor for heart disease mortality. Clearly, this tenuous association cannot serve as much support for the hypothesis which implicates fat as an aetiological factor in arteriosclerotic and degenerative heart disease". As a result: "It is concluded that the suggested association between national death rates from heart disease and the percentage of fat in the diet available for consumption cannot at the present time be accepted as valid".

Similarly, Yudkin[25] argued that the relationship might be explained just as well by differences in affluence or in sugar intake between those countries. For example, an increasing number of radio and television licences was associated (not causally) with the rising CHD mortality in the UK, suggesting a role of increasing affluence. He also showed that patients with occlusive arterial disease ate nearly twice as much sugar as controls without this disease,[26] that there was a close relationship between dietary fat and sugar intake in 41 countries,[27] and that in 15 of the 22 countries studied by Yerushalmy and Hilleboe on which data were available, rising sugar intakes were associated with increased CHD rates (Figure 2). He questioned how a distinction could be made between sugar and fat as the key nutritional factor driving the CHD "epidemic" if consumption rates for both were high in countries with high CHD rates and vice versa. A subsequent 1974 study showed an almost perfect relationship between the amount of sugar in the diets of different nations and their CHD rates.[28]

Keys also conveniently ignored the evidence that the exponential growth, and then fall, in cigarette consumption, exactly matched changes in CHD incidence in the USA (Figure 3).[29]

Note that Harper argues that death rates from coronary heart disease were already high in 1900, but were under-reported due to "frequent reclassifications of heart disease during the past 75 years" so that no single risk factor has yet been discovered that tracks changes in coronary heart disease mortality since 1900[29]

Thus, already in the 1970s, associational evidence linking tobacco and sugar use with the rising incidence of CHD was at least as strong as any postulated link between saturated fat and that disease.

### *Further growth of the diet-heart hypothesis*

The manner in which the diet-heart hypothesis became an institutionalised dogma, immune to disinterested scientific enquiry, has been described in great detail by Taubes,[21] Minger[19] and Teicholz.[20] A few key points are presented here.

Firstly, USA President Eisenhower's heart attack in September 1955 was skilfully orchestrated to prove that Americans were in the midst of a CHD epidemic.[21] In fact, the incidence of CHD in the USA had begun to increase shortly after the end of World War I, and was already peaking in the 1950s at the time of Eisenhower's heart attack (Figure 3).Within a decade, and even before scientists had begun to institute preventive steps, CHD was already in retreat in the USA. Others[29] argue that the death rates from all causes of heart disease had been falling since the early 1900s, and that the cause of this "epidemic" is owing to "frequent reclassifications of heart disease during the past 75 years".[29]

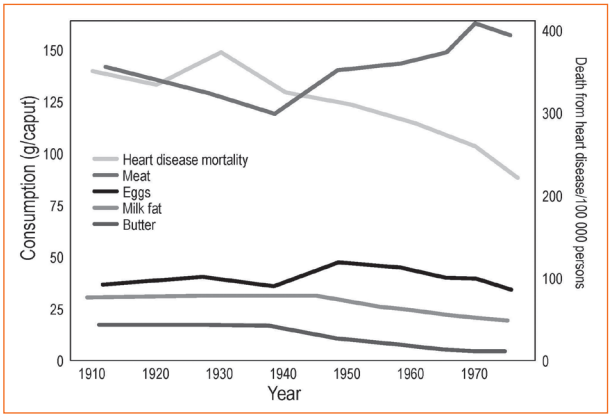



**Figure 4:** Changes in the age-adjusted death rates from heart disease and per person intake of meat, eggs, milk, fat and butter in the USA from 1910-1970[29]

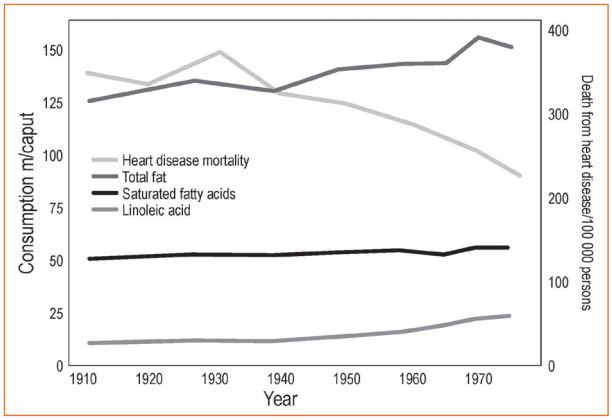



**Figure 5:** Changes in the age-adjusted death rates from heart disease and per person intake of fat and fatty acids from 1910-1970 in the USA[29]

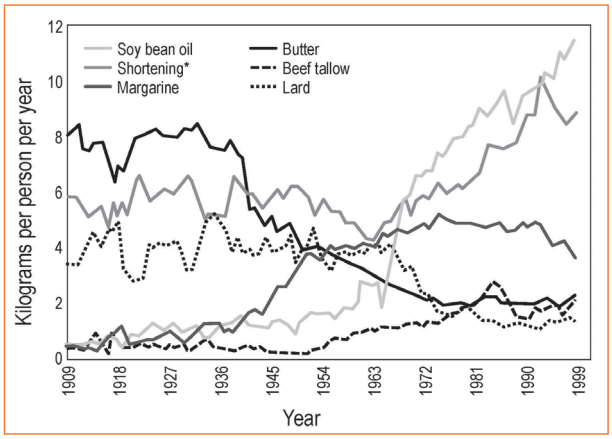



**Figure 6:** Changes in the per capita intake of soy bean oil, shortening, margarine, butter, beef tallow and lard between 1909 and 1999 in the USA[20]

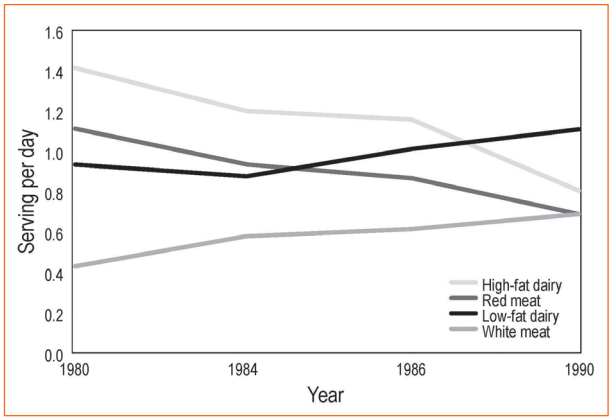



**Figure 7:** The age-adjusted intake of the major food groups (high-fat dairy, red meat, low-fat diary and white meat) between 1980 and 1990 by participants in the USA Nurses' Health Study[43]

Secondly, the American Heart Association (AHA) did not initially support Keys' speculative hypothesis based on associational data. In 1957, a 13-page review concluded that "the proposition that the character of the American diet has so changed during the past 50 years as to increase the incidence of coronary vascular disease cannot be supported".[30] Without the addition of any new information, in 1961, the AHA reversed its earlier conclusion, advising Americans at risk of CHD to reduce their consumption of dietary fat and cholesterol, concluding that "this recommendation is based on the best scientific information available at the present time".[31] The sole change between 1957 and 1961 was not the appearance of definitive new evidence. Rather, it occurred after Keys became a key member of the AHA committee.

Thirdly, Keys' speculative hypothesis could become the singular focus of USA commercial and political interests, only if the two other suspected culprits of the day, cigarettes and sugar, could be made to "disappear". The formation of the tobacco lobby, i.e. Tobacco Industry Research Committee, in the 1950s specifically to bury concerns about the health dangers of the tobacco habit explains why any possible link between the cigarette habit and CHD (and other diseases) was not pursued.[32] Similarly, the formation of the USA Sugar Research Foundation in 1943, subsequently re-named the Sugar Association in 1947, served an identical function.[33] As a result, the potential link between sugar and ill health, including CHD, proposed by a group of Afro-English scientists,[34,35] including Yudkin,[36,37] was effectively silenced. Instead, the Sugar Lobby applied its influence to squeeze Yudkin's funding sources, causing the premature termination of his research career.[37]

As a result, the possibility that rising sugar consumption might play a central role in the development of obesity, type 2 diabetes mellitus and CHD has been skilfully buried for the past 70 years. But the book that Yudkin wrote in 1972[37] survives as a work of genius. Forty years later, his campaign has been revived by Taubes,[33] Gillespie[38] and Lustig,[39-41] among others.[42]

With the cigarette and sugar hypotheses conveniently concealed from public scrutiny, the path was cleared for the diet-heart and lipid hypotheses to become the unchallenged winners. But while there was clear evidence that the "increase" in CHD incidence occurred at the same time that cigarette (Figure 3) and sugar consumption[35] had both increased dramatically, no such increase occurred in the intake of meat, eggs, milk fat or butter (Figure 4).[29] An increase in total fat intake began after 1940 (Figure 5),[29] but was due to an increased intake of polyunsaturated fat, including linoleic acid, not saturated fat. Figure 6 shows that the intake of lard and butter fell dramatically since 1909, whereas the intake of soy bean, shortening

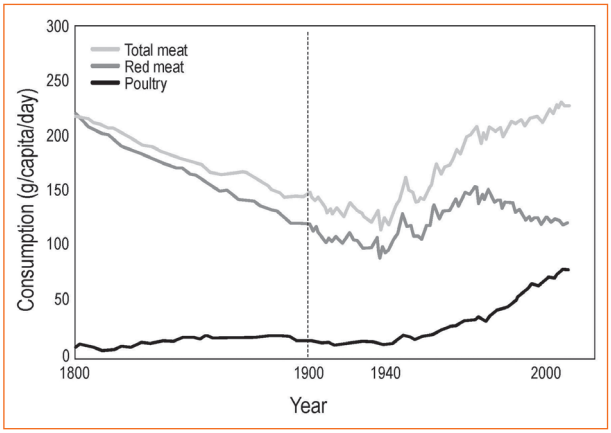



**Figure 8:** Changes in total meat and red and white meat intake from 1800-2000 in the USA[20]

and margarine increased steeply over the past century, with a much smaller increase in beef tallow. More recently, data from the USA Nurses' Health Study show that the intake of high-fat dairy and red meat fell from 1980 and 1990, to be replaced with an increased intake of low-fat dairy and white meat (fish and poultry) (Figure 7).[43] This change is associated with the rising incidence of obesity and type 2 diabetes mellitus after 1980 (next section).

Note that before 1936, shortening comprised mainly lard, but the lard was replaced with hydrogenated oils incrementally thereafter.

Instead, it seems probable that modern Americans began to eat substantially less meat in the 100 years immediately before CHD rates began to increase (Figure 8),[20,44] and especially in the 1930s after the Depression when CHD rates were increasing, according to the conventional interpretation.[29] Thus, "meat consumption in the USA dropped to unusually low levels in the Great Depression. Consumption rebounded in the 1940s, but it remained well below levels seen in the early 20th century, and was sharply distinguished by income".[44]

Importantly, meat intake has always been least in the lowest socio-economic classes, precisely those who suffer the greatest burden of chronic diseases, including CHD.[45] Subsequently, "meat consumption began to climb dramatically in the 1950s after the end of the Korean War's rationing programme.[44] By 1965, it had reached the highest levels in American history".

Thus, the conventionally described increase in CHD rates in the USA from 1910-1950 was associated with decreased meat, and hence saturated fat intake from meat, whereas declining rates after the 1960s occurred subsequent to a ~ 50% increase in meat consumption, especially in those in the lowest socio-economic groups. Most of this increase is explained by the rising consumption of white meat, especially poultry (Figure 8).

Therefore, from the outset, the validity of the diet-heart hypothesis, linking the conventionally described rising CHD incidence after 1910 to an increased saturated fat intake from meat especially, is not supported by the evidence, at least in the USA (Figures 4-8). Instead, it would seem to be disproven by this information that is conveniently ignored by advocates of the diet-heart hypothesis.

### *The industrialisation of corn production in the USA leads to the 1977 United States Department of Agriculture dietary guidelines*

The next key event that drove the global adoption of the diet-heart hypothesis was the 1972 USA presidential election, in which the incumbent, Nixon, was confronted by a losing war in Vietnam, rising food prices, unhappy housewives and a disgruntled farming community. He appointed Butz as Secretary of Agriculture with two orders, namely to increase the wealth of USA farmers, and to bring down the price of food.[46] Butz decided that the production of corn on an industrial scale by farmers receiving large government subsidies to cultivate all their available land was the solution to both "problems". His actions would have momentous effects on global health.

The industrialisation of corn production would be of little value if all of the newly grown corn was not eaten, either by USA citizens or the rest of the world. The challenge was to convince the world that grains and cereals were healthier than the foods high in animal fat and protein that Americans had always eaten.

The key player was Senator McGovern, whose Senate Select Committee on Nutrition and Human Needs developed the first USDA dietary guidelines in 1977. These novel guidelines were based specifically on Keys' unproven diet-heart hypothesis. They advised Americans to restrict their intake of saturated fats, especially, by eating 8-12 servings of grain and cereals per day. Grains and cereals replaced the butter, lard, cheese, eggs and meat that had been the American staples until then.[20]

The 1977 USDA dietary guidelines, compiled by a vegetarian, Mottern, who had no formal training in nutrition science,[20,21] were criticised by Handler, then President of the National Science Academy. Handler posed the question: "What right has the federal government to propose that the American people conduct a vast nutritional experiment, with themselves as subjects, on the strength of so very little evidence that it will do them any good?" He added: "The resolution of this dilemma turns on a value judgement. The dilemma so posed is not a scientific question; it is question of ethics, morals and politics. Those who argue either position strongly are expressing their values. They are not making scientific judgements".[21]

Similarly, one of the leading cholesterol researchers of the time, Ahrens,[47] noted that "a trial of the low-fat diet recommended by the McGovern committee and the AHA has never been carried out. It seems that the proponents of this dietary change are willing to advocate an untested diet to the nation on the basis of suggestive evidence obtained from tests of a different diet. This illogic is presumably justified by the belief that benefits will be obtained, *vis-à-vis* CHD prevention, by any diet that causes a reduction in plasma lipid levels".

Other dissenters included Corday: "New investigations indicate that the lipid hypothesis has not yet been proven to be completely correct,[48] so that before a change of dietary goals is applied to the nation to prevent arteriosclerosis, it behoves the health planners to

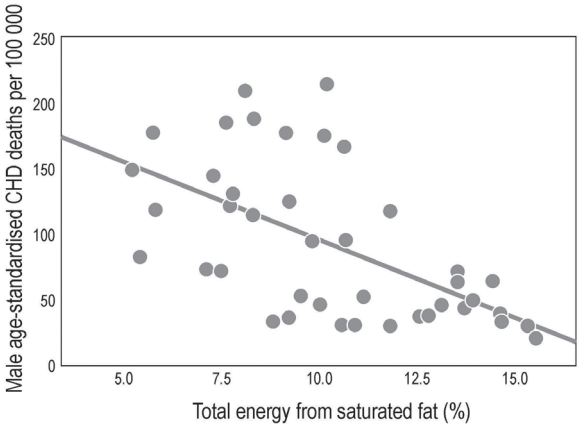
test the hypothesis of alterations in nutritional risk with the diets advocated by their health advisers". He also noted that "less than 20% of patients" with CHD have an "abnormal lipid pattern". Harper pointed out that "the assumption that cardiovascular diseases may be delayed by the adoption of the dietary goals is speculation" so that "proposals for action, that are based on speculation and conjecture, rather than on critical analysis, are distressing under any circumstances. They are especially distressing when they are proposed by a senate committee for adoption as the basis for federal policy". Leveille argued that the "committee's report failed to consider all of the available evidence" and that "there is equally compelling evidence suggesting that the recommended dietary changes would make little or no difference as to the incidence and severity of cardiovascular disease or cancer". Mann complained that "the level of fat in the diet has not been related causally to any disease, and in particular, not to either obesity or to cancer. Those who contend this are adventurists" because "the amount of saturated fat in the diet has not been shown to be causal for any disease". He concluded that "the release of this document is a nutritional debacle". Olson concurred: "I think the proposal is disastrous as there is no evidence at present to justify such a drastic change in the American diet". He argued that the new guidelines were "not based on scientific evidence" and that "there is no evidence from 10 intervention studies on coronary disease involving 5 000 men and 36 000 man-years of study on the effect of the prudent diet that diet modification will change the mortality rate of this disease". Schmidt warned: "For anyone to say, 'Let's change the nation's dietary habits, even though we don't know that doing so will do any good – it can't do any harm' is naïve. One doesn't know, and can't predict, what harm may result. But experience teaches us that we often learn late, even a generation late of harm, that no one could or did predict". Others argued that "the data demonstrating the concept that the risk of coronary heart disease is a function of serum lipids at any level is no longer valid".[49]

Finally, Hegsted, then Professor of Nutrition at the Harvard School of Public Health, presented a more optimistic opinion: "The question to be asked, therefore, is not why should we change our diet, but why not? What are the risks associated with eating less meat, less fat, less saturated fat, less cholesterol, less salt and more fruit, vegetables, unsaturated fat and cereal products, especially wholegrain cereals. There are none that can be identified and important benefits can be expected". One wonders whether Hegsted would still hold this position.

These guidelines continue to be modified every five years. However, the advice to eat less animal fat and protein and more carbohydrate in the form of grains and cereals remains immutable.[50] Instead, the guidelines appear to be driven by the political decision that only the increased production of cereals and grains, and not animal products, would provide sufficient food to sustain the global population explosion.

### *Recent epidemiological evidence does not support the diet-heart hypothesis*

Modern epidemiological data show an inverse relationship between the percentage of saturated fat in the diet and the incidence of heart disease in European countries (Figure 9).[51] In fact, Western European countries have among the lowest rates of heart disease in the world, despite high rates of saturated fat intake and higher blood cholesterol concentrations, a phenomenon that has been termed the "European Paradox". This term replaces the incorrect "French Paradox". Thus, in 2015, Keys would be unable to produce epidemiological evidence to support his 1957 "unequivocal proof".



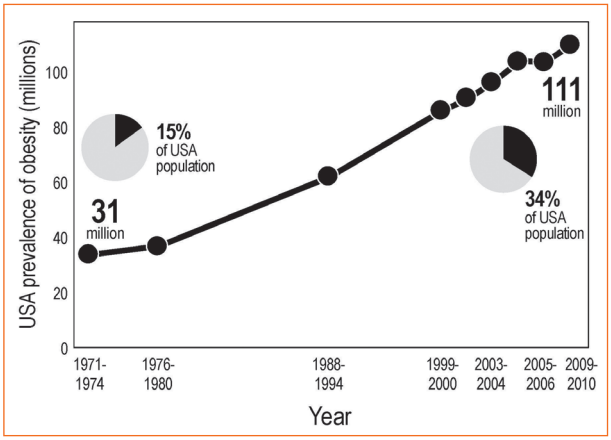

CHD: coronary heart disease

**Figure 9:** The inverse relationship between the percentage of dietary energy from saturated fat and age-adjusted death rates from coronary heart disease in 41 European countries[51]

Furthermore, meta-analyses show that the amount of fat in the diet does not relate to heart disease risk in individuals,[52-56] and that reducing dietary fat intake has not been proven to reduce heart attack risk,[20,57] although there is some suggestion from associational studies that replacing saturated fat in the diet with omega-3 polyunsaturated fat may provide some benefit,[58] whereas replacement with carbohydrates or omega-6 polyunsaturated fat is more likely to be detrimental.[59-61] It is perhaps important to remember that it has never been shown that persons with heart disease eat more saturated fat than those without the disease.[52] Even in the revered Framingham study, it was found that "there is a considerable range of serum cholesterol levels within the Framingham study group. Something explains this inter-individual variation, but it is not the diet".[62] Interestingly, this information is from an unpublished manuscript from the Framingham study group discovered by Mann in a basement in Washington DC. Another quotation is: "In Framingham, for example, we found that the people who ate the most cholesterol ate the most saturated fat, ate the most calories, weighed the least, and were the most physically active".[63]

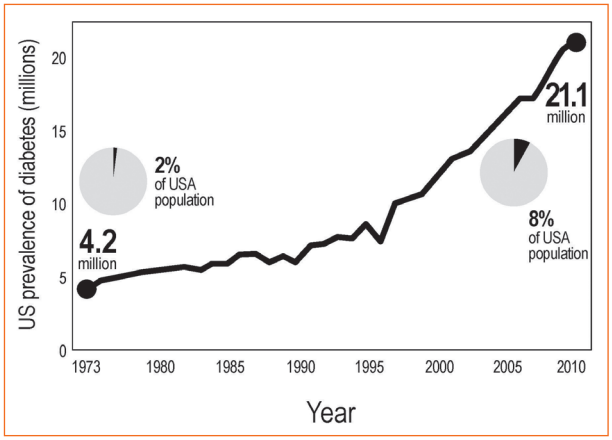
The Harvard School of Public Health has recently written:[64] "Well, it's time to end the low-fat myth. The low-fat approach to eating may have made a difference to the occasional individual, but as a nation, it hasn't helped us to control weight or become healthier".

In summary, the original data on which the diet-heart hypothesis were constructed are purely associational and cannot ever prove causation. That almost all subsequent studies of this hypothesis are fatally flawed has been exposed by careful analysis in three recent books.[19-21] Teicholz[20] concluded that "the advice that comes out of this book is that a higher-fat diet is almost assuredly healthier than




Note that the exponential rise in obesity rates began after 1980, three years after the promotion of the 1977 United States Department of Agriculture dietary guidelines

**Figure 10:** The increase in the prevalence of obesity in the USA from 1971-2010[67]




Note that the exponential rise in type 2 diabetes mellitus rates began in 1995, 18 years after the promotion of the 1977 United States Department of Agriculture dietary guidelines and in keeping with the 20-year rule[13,35]

**Figure 11:** The increase in the prevalence of type 2 diabetes mellitus in the USA from 1973-2010[67]

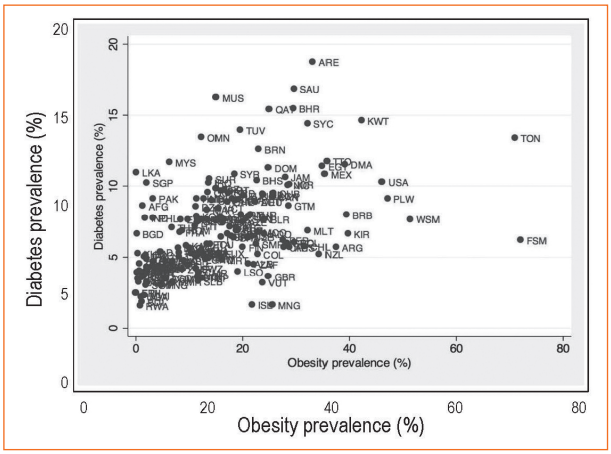
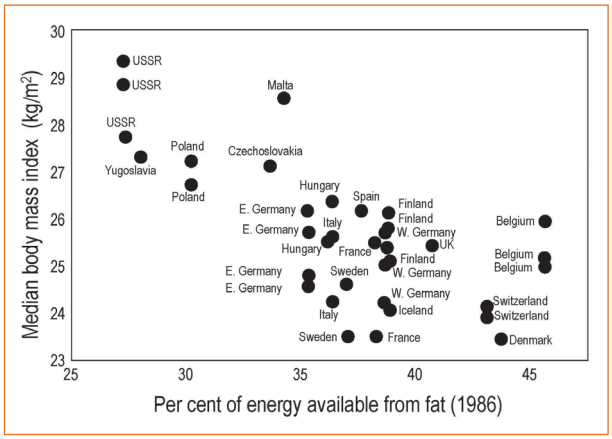



**Figure 12**: The relationship between obesity and diabetes prevalence rates worldwide[41]




E. Germany: East Germany, W. Germany: West Germany, UK: United Kingdom, USSR: Union of Soviet Socialist Republics

**Figure 13:** Median body mass index in women in 18 European countries and the percentage of energy provided by fat in their national diets[66]

one low in fat and high in carbohydrates. The most rigorous science now supports this statement".

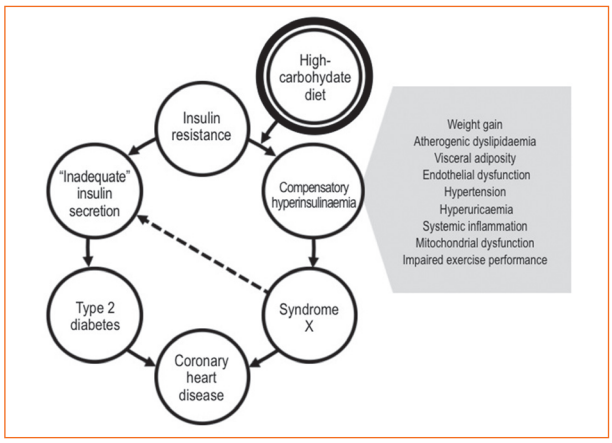
The power of this evidence led *Time* magazine to conclude in its 23 June 2014 issue: "Don't blame fat. For decades it has been the most vilified nutrient in the American diet. But new science reveals fat isn't what's hurting our health". Instead, the author concludes that it is dietary carbohydrates that are the villain.

**Within five years of adoption of the 1977 United States Department of Agriculture dietary guidelines, rates of type 2 diabetes mellitus and obesity increased explosively, especially in Britain and the USA**

The damage caused by the adoption of the 1977 USDA dietary guidelines did not take long to surface. Already by 1994, the adoption of these guidelines was followed by a 6% increase in daily energy intake in men and a 22% increase in women.[50] This is predictable since carbohydrates drive hunger. They do not satiate. This information has been known since at least 1970.[65]After 1980, there was also a dramatic increase in the rates of obesity (Figure 10) and type 2 diabetes mellitus (Figure 11) in the USA, perfectly matching the temporal change in this increased carbohydrate and energy consumption.[50] Interestingly, there is a strong relationship between obesity and diabetes prevalence rates across the world (Figure 12).[41] Furthermore, there was an inverse relationship between the median body mass index and the percentage of fat in the diet (Figure 13),[66] at least in women, in 18 European countries, leading Willett to conclude that "diets high in fat do not appear to be the primary cause of the high prevalence of excess body fat in our society, and a reduction in fat will not be a solution".

This increased calorie intake by persons with IR (next section) would be more than sufficient in explaining the explosive rise in the USA obesity and diabetes rates.

That these two events are linked by both time and plausible biological mechanisms, proves, in my opinion, that "current dietary recommendations do more harm than good". This also disproves Hegsted's optimistic predictions of the health benefits that would follow the adoption of the 1977 USDA dietary guidelines.




Note that in the absence of a high-carbohydrate diet, insulin resistance does not "cause" the hyperinsulinaemia needed to produce these pathological changes

**Figure 14:** The mechanism by which insulin resistance leads to coronary heart diease[71]

**The presence of insulin resistance explains why large numbers of persons in predisposed populations become obese and develop type 2 diabetes mellitus, when following the high-carbohydrate, low-fat diet advocated by the United States Department of Agriculture dietary guidelines**

Not all humans are created with an equal capacity to metabolise carbohydrate. Instead, a sizable proportion of humans are unable to store the glucose derived from ingested carbohydrate normally in their liver and muscles[68-71] and have the condition of IR. Persons with IR convert excess ingested carbohydrate into triglycerides (TGs) in the liver,[72] producing non-alcoholic fatty liver disease,[73] or export the fat in the blood, raising blood TG concentrations.[74] Excess production of hepatic TG reduces high-density lipoprotein (HDL) cholesterol blood concentrations,[69,75-79] and increases the production of small, dense low-density lipoprotein (LDL) cholesterol particles. Persons with an increased number of small, dense LDL cholesterol particles are at threefold greater risk of suffering a heart attack. This risk may,[80] or may not,[81] be explained by related changes in HDL cholesterol and TG blood concentrations.[82] Small, dense LDL cholesterol particles are more damaging because of an increased susceptibility to oxidation.[83] Interestingly, the higher rates of CHD in men are not predicted by higher total cholesterol concentrations, but rather by increased numbers of small, dense LDL cholesterol particles, compared to women.[84] In addition, ultra-sensitive C-reactive protein concentrations in the blood plasma, a marker of systemic inflammation, are associated with an increasing dietary carbohydrate intake.[85] The immediate consequence is that persons with IR show each of these acute abnormalities in response to any meal containing carbohydrates.[68,86]

With time, the excessive production of insulin produces pancreatic beta-cell failure and the onset of type 2 diabetes mellitus. Accumulating evidence implicates high-carbohydrate diets and continual hyperinsulinaemia[71] in the causation of :

- Arterial damage in diabetes.[87-90]
- Gout.[71,91]
- Hypertension.[71,92]
- Mental deterioration with age.[93,94]
- Alzheimer's disease.[95]
- A central component of the ageing process.[96]
- In the causation of at least two common cancers, of the colon[97] and breast.[98]

Increased cancer risk is also associated with hyperglycaemia.[99] The manner in which repetitive hyperinsulinaemia contributes to these diseases in response to a high-carbohydrate diet is depicted in Figure 14.

The diagnostic features of IR are listed in Table I. Increased girth and hypertension are the additional features of this condition. These are also the features of metabolic syndrome. Thus, the only logical conclusion must be that metabolic syndrome is simply the long-term result of a high-carbohydrate diet eaten by those with IR (Figure 14).

**A high-fat diet reverses all known coronary risk factors in persons with insulin resistance, whereas a high-carbohydrate diet may worsen those risk factors**

The idea that one can predict one's future risk of heart attack or other illness on the basis of "risk factors" measured many years before is of quite recent origin, beginning with the iconic Framingham study in 1948.[20]

But risk factors are not diseases, nor does their presence mean that they cause disease. Risk factors are simply a range of measured variables found to be associated more frequently with particular diseases; that is, the relationship is purely associational. Fear of the risk factors has great commercial potential since it converts a population of healthy people into fee-paying patients.[100] Table II provides a detailed list of the range of identified risk factors for CHD.

**Table I:** The metabolic characteristics of persons with insulin resistance exposed to a high-carbohydrate diet for a prolonged period (decades)

| Blood parameters |
|---|
| • Elevated blood glucose concentrations |
| • Elevated blood insulin concentrations |
| • Elevated haemoglobin $A_{1c}$ concentrations |
| • Elevated blood triglyceride concentrations |
| • Reduced blood high-density lipoprotein cholesterol concentrations |
| • Increased, small, low-density lipoprotien particles |
| • Increased blood uric acid concentrations |
| • Increased blood ultra-sensitive C-reactive protein concentrations |

**Table II:** Blood risk factors for the development of coronary heart disease

| Blood parameters |
|---|
| • Total blood cholesterol concentrations |
| • Blood ultra-sensitive C-reactive protein concentrations |
| • Bloof fibrinogen concentrations |
| • Fasting or random blood glucose concentrations |
| • Blood-glycosylated haemoglobin concentrations |
| • Blood homocysteine concentrations |
| • Blood high-density lipoprotein cholesterol concentrations |
| • Blood low-density lipoprotein cholesterol concentrations |
| • Blood low-density lipoprotein particle size or number |
| • Blood lipoprotein (a) concentrations |
| • Blood-fasting insulin concentrations |
| • Blood omega 6 to omega 3 ratio |
| • Blood triglyceride concentrations |
| • Blood uric acid concentrations |

Seldom are these risk factors measured in individual patients for reasons of cost. Rather, the total cholesterol, HDL cholesterol and TG concentrations in the blood are measured. Persons with elevated total cholesterol and TG concentrations, and reduced HDL cholesterol concentrations, are labelled as suffering from atherogenic dyslipidaemia (AD). According to the diet-heart hypothesis, patients with AD must be treated with a low-fat diet and cholesterol-lowering medications (statins) in the belief that this prevents any further progression of coronary atherosclerosis. But this is an unproven theory.[101]

A recent publication evaluated the ability of some of these risk factors to predict the future risk of a heart attack or stroke[102] in a population of 165 544 individuals, of whom 10 132 developed heart disease and 4 994 suffered a stroke. Table III lists those risk factors in order of their predictive power, measured as the hazard ratio (HR). The higher the HR, the greater the ability of that factor to predict future risk in that study.

**Table III:** The relative importance, based on the hazard ratio, of the different risk factors for coronary heart disease[102]

| Risk factor | Hazard ratio (95% confidence interval) |
|---|---|
| Diabetes | 2.04 (1.76-2.35) |
| Age | 1.87 (1.73-2.02) |
| Current smoking | 1.79 (1.66-1.94) |
| Systolic blood pressure | 1.31 (1.26-1.37) |
| Total cholesterol concentration | 1.22 (1.17-1.27) |
| Triglyceride concentration | 1.19 (1.15-1.23) |
| High-density lipoprotein cholesterol concentration | 0.83 (0.78-0.87) |

Total blood cholesterol concentration was among the least important risk predictors since an elevated blood cholesterol concentration increases the risk of a future heart attack by only 21%, little better than the risk associated with elevated blood TG concentrations (19%), or low HDL cholesterol concentrations (17%). In contrast, age alone predicts an increased risk by 87%, whereas the presence of diabetes increases the risk by 104%. Other studies show that the majority of persons in the USA with a heart attack have normal blood cholesterol concentrations, but elevated blood TG concentrations[103] (in fact, the Framingham study was the first to show this),[104] and that blood TG concentrations, not total or LDL cholesterol concentrations, in persons with type 2 diabetes mellitus, are the best predictors of CHD risk.[82] It was found in the Framingham study that "90% of the total cholesterol levels measured were ineffectual (by themselves) in predicting the risk of CHD in a general population. Indeed, twice as many individuals who had a lifetime total cholesterol level of less than 200 mg/dl (5.2 mmol/l) had CHD, compared with those who had a total cholesterol level greater than 300 mg/dl (7.8 mmol/l)".[105]

Additional published findings that conflict with the lipid hypothesis are that a study has not yet shown a significant relationship between pre-morbid blood cholesterol concentrations and the extent of coronary atherosclerosis at autopsy.[106,107] Nor do blood cholesterol concentrations predict the coronary artery calcium volume (CACV) score, by far the best predictor of future risk of a cardiac event,[108] because groups with the highest and the lowest CACV scores have identical ranges of blood cholesterol concentrations.[108] This latter study is perhaps the strongest evidence that disproves the lipid hypothesis, although the authors fastidiously avoid drawing that conclusion.

Thus, the question: "If total blood cholesterol concentration is such a relatively poor predictor of heart attack risk, is it likely to be the sole important factor causing heart disease, as is the focus of this debate?"

In summary, these data show that the total blood cholesterol concentration is a poor predictor of future heart attack risk (Table III), confirming that "cholesterol is not an important risk factor for heart disease".

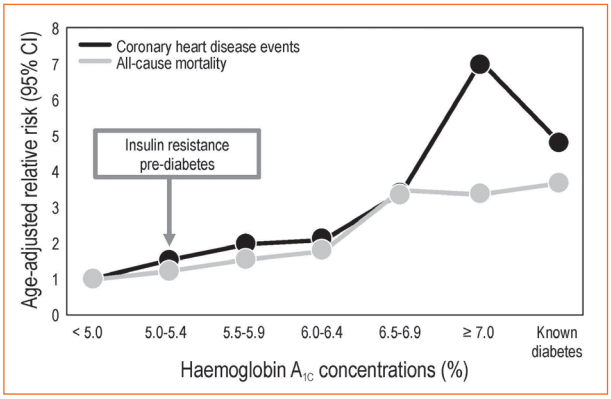
Instead, both diabetes and metabolic syndrome, which are disorders of carbohydrate, not fat, metabolism, are the single most important risk factors for a heart attack. What is it about diabetes and metabolic syndrome that makes them so dangerous?

### *The concept of hyperglycaemic hyperinsulinaemic atherogenic dyslipidaemia*

In contrast to what Keys wrote in 1953, diabetics do not have higher blood cholesterol concentrations than those without the disease.[109] Thus, higher rates of arterial damage in diabetics cannot be due to higher blood total cholesterol concentrations. Something else must be involved.

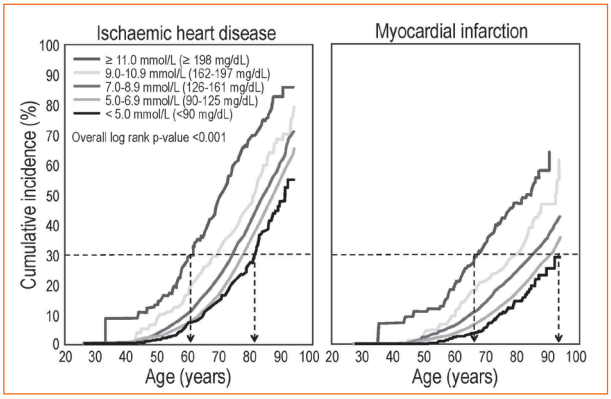
The best predictors of heart attack risk in those with an abnormal carbohydrate metabolism are the blood TG concentrations,[82] and the blood glycosylated haemoglobin ($HbA_{1c}$) concentrations,[110,111] a measure of the average 24-hour blood glucose concentrations over the previous three months. Similarly, blood TG, but not other lipid markers, are associated with magnetic resonance imaging markers of cerebral small vessel disease.[112]

Thus, the Norfolk arm of the European Investigation into Cancer and Nutrition Study (EPIC-Norfolk study)[110] found an essentially linear increase in the relative risk of developing a heart attack with increasing $HbA_{1c}$ concentration (Figure 15), such that an $HbA_{1c}$ concentration greater than 7% (a value considered to be acceptable in persons with type 2 diabetes mellitus eating a high-carbohydrate diet, according to the current guidelines for treating diabetes) increases the risk of a heart attack more than sevenfold, that is, by more than 700%. Compare this to the ~ 20% increased risk associated with an elevated blood cholesterol concentration (Table III).This relationship was "independent of age, body mass index, waist to hip ratio, systolic blood pressure, serum cholesterol concentration, cigarette smoking, and a history of cardiovascular disease".[111] The authors concluded that their findings "support the need for randomised trials of interventions to reduce haemoglobin $A_{1c}$ concentrations in persons without diabetes". Restricting dietary carbohydrate intake is currently the only proven physiological method of reducing the $HbA_{1c}$ concentration, especially in those with IR and diabetes.[113]



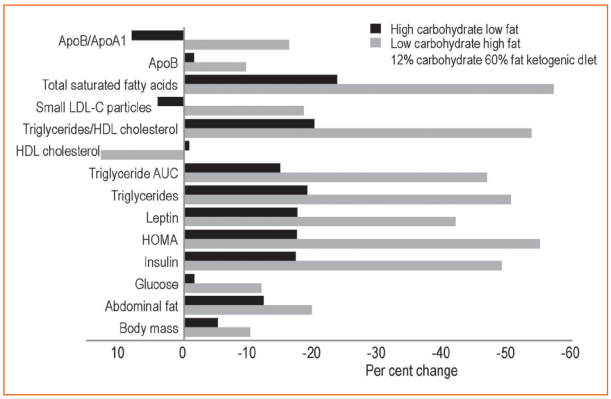

CI: confidence interval, EPIC-Norfolk study: Norfolk arm of the European Investigation into Cancer and Nutrition Study
All-cause mortality also rose with increasing haemoglobin $A_{1c}$ concentration

**Figure 15:** The EPIC-Norfolk study found an essentially linear increase in the relative risk of developing coronary heart disease with increasing haemoglobin $A_{1c}$ concentration[110]




Note that 30% of persons with a random blood glucose concentration > 11 mmol will suffer ischaemic heart disease by age 60, whereas the same outcome will occur at age 80 years in those with random glucose concentrations < 5 mmol/l. The same applies to myocardial infarction

**Figure 16:** Cumulative incidence with age of ischaemic heart disease (left panel) and myocardial infarction (right panel) in persons with different random blood glucose concentrations[115]




ApoB/ApoA1: apolipoprotein B/apolipoprotein A1, AUC: area under the curve, HOMA: homeostatic model assessment, LDL: low-density lipoprotein, HDL: high-density lipoprotein
Note that the high-fat diet produces superior changes in all these variables

**Figure 17:** Changes in coronary risk factors in persons with metabolic syndrome in response to either a high-fat or high-carbohydrate diet[143]

The manner in which elevated blood glucose concentrations cause arterial damage was fully described by Brownlee in his 2004 Banting lecture.[114]

The ages at which persons with different random blood glucose concentrations are likely to develop ischaemic heart disease or myocardial infarction was determined in another publication from the EPIC study.[115] It was shown in the study that there is a graded effect of the blood glucose concentration, so that the lower the glucose concentration, the older the age at which the risk of a heart attack begins to rise (Figure 16).

Even within the normal range of blood glucose concentrations (5.0-5.5 mmol/l), heart attack risk rises with increasing blood glucose concentration, so that persons with fasting blood glucose concentrations at the upper limit of the normal range have a 50% higher risk of suffering a heart attack than those with blood glucose concentrations of 5.0 mmol/l.[116] However, absolute risk at these low $HbA_{1c}$ values is extremely low.

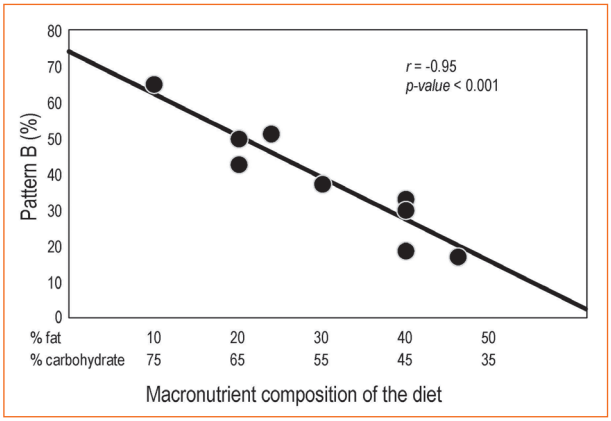
Other markers of metabolic syndrome and IR (Table I) include elevated blood insulin and TG concentrations, and reduced HDL cholesterol concentrations, with an increased number of small, dense LDL cholesterol particles. The key recent finding described in the next section is that all these risk factors are modified in the same direction by a diet rich in carbohydrates, and in the reverse direction, by a diet low in carbohydrates and higher in fat and protein.

### *All the metabolic features of the hyperglycaemic hyperinsulinaemic atherogenic dyslipidaemia alter together, worsening on a high-carbohydrate diet and improving on a high-fat diet*

According to the traditional model, a high-fat diet promotes arterial damage by causing AD. But according to the hyperglycaemic hyperinsulinaemic atherogenic dyslipidaemia model, a high-carbohydrate diet causes arterial damage by producing changes in almost all of the risk factors listed in Table III.

The suppressed[20] work of Phinney and Volek[117-119] has clearly established that all the known risk factors alter in the same direction in response to a dietary change, improving on the LCHF diet, or either worsening or improving less on the high-carbohydrate diet promoted by the USDA dietary guidelines. Twenty-five RCTs[75,120-144] have now established the superiority of the LCHF over the HCLF diet.

Figure 17 provides a summary of the findings from the most complete study by Volek et al,[143] in which patients with metabolic syndrome were studied while eating either a high-fat or high-carbohydrate diet. All risk factors improved on the high-fat diet, whereas they changed less, or worsened, on the high-carbohydrate "prudent" diet. Importantly, blood glucose, insulin and TG concentrations, and the number of small, dense LDL cholesterol particles increased on a low-fat diet, but were reduced on the LCHF diet, as were apolipoprotein B blood concentrations. Similarly, blood HDL cholesterol concentrations increased on the low-carbohydrate diet, but were reduced on the low-fat diet. Other studies have reported similar findings,[145,146] especially the effect of high-carbohydrate diets in increasing the atherogenic small, dense LDL particles.[146-148] As a result, the prevalence of the atherogenic pattern B, comprising increasing numbers of these atherogenic particles, is an inverse function of the percentage of fat in the diet (Figure 18).[147]




Note that a higher fat intake is associated with a reduction in the proportion of subjects with this atherogenic pattern B profile

**Figure 18:** Relationship between the percentages of dietary fat and carbohydrate and the prevalence of the atherogenic pattern B low-density lipoprotein particle sizes in men[147]

More recently, Volek's group[149] showed that "dietary- and plasma-saturated fat are not related, and that increasing dietary carbohydrates across a range of intakes promotes incremental increases in plasma palmitoleic acid, consistently associated with adverse health outcomes".

To prove that these findings are not specific to a single research group, a recent meta-analysis has concluded that "data obtained on 1 141 obese patients showed the low-carbohydrate diet to be associated with significant decreases in body weight, body mass index, abdominal circumference, systolic blood pressure, diastolic blood pressure, plasma TGs, fasting plasma glucose, glycated haemoglobin, plasma insulin and plasma C-reactive protein, as well as an increase in HDL cholesterol. LDL cholesterol and creatinine did not change significantly, whereas limited data exist concerning plasma uric acid".[150] The finding that the LCHF diet did not cause a significant increase in blood total cholesterol concentrations, as also reported in a second meta-analysis,[151] is especially interesting, and disproves the contention that an increased dietary fat intake will inevitably cause blood total or LDL cholesterol concentrations to rise.

Thus, these studies establish a common aetiological mechanism for the chronic diseases, i.e. obesity, type 2 diabetes mellitus, metabolic syndrome and heart disease, as already shown by Reaven.[71,152] These diseases occur in persons with IR who are exposed to high-carbohydrate diets for 10 or more years.

**The multi-million dollar 48 835-persons Woman's Health Initiative Randomized Controlled Dietary Modification Trial, of which Rossouw was project leader, shows that the United States Department of Agriculture dietary guidelines are associated with accelerated disease progression in persons with either established heart disease or type 2 diabetes mellitus. This study does not support Keys' diet-heart hypothesis, of which Rossouw continues as a staunch advocate**

The goal of the WHIRCDMT[153] was to determine whether or not a population of older women who followed the USDA dietary guidelines would reduce their risks of colorectal and breast cancers, as well as heart disease. For the study, 48 835 postmenopausal women were encouraged to either adopt the USDA dietary guidelines by reducing their fat intake, and eating more vegetables and grains, or to continue eating their usual diet. Women in the intervention group also received an "intensive behavioural modification programme" comprising 18 group sessions in the first year, followed by quarterly maintenance sessions for the next seven years. The control group only received a copy of dietary guidelines for Americans. As a result, any positive outcomes in the intervention group could not be ascribed purely to dietary change, since the intervention group received additional interventions not shared by the control group.

The study found that eating according to the USDA dietary guidelines did not reduce the risk of cancers of either the colorectum or breast. This is predictable if both cancers are associated with low-fat, high-carbohydrate diets, or rich in omega 6 polyunsaturated fats[154] of the type promoted by the 1977 USDA dietary guidelines. The authors concluded that the avoidance of weight gain reduced the risk for developing these cancers.[155] Given that high-carbohydrate diets cause weight gain in persons with IR, it would have been more logical for the authors to postulate that high-carbohydrate diets explain the co-existence of obesity with colorectal and breast cancers in persons with IR.

The main reported finding of the WHIRCDMT study on heart disease[153] was that "a reduced total fat intake and increased intake of vegetables, fruit and grains did not significantly reduce the risk of CHD, a stroke or CVD in postmenopausal women, and achieved only modest effects on CVD risk factors". In reality, this study, published after the USDA dietary guidelines were first released, found that these eating guidelines were without any health benefits in postmenopausal women. The warnings expressed by the experts in 1977 quoted earlier, have proven to be correct.

In fact, the only significant finding in that study escaped the attention of the authors until quite recently.[156] Evidence is presented on the seventh page of the published manuscript, where the following is stated: "The HR for 3.4% of the women with CVD at baseline was 1.26 (95% confidence interval: 1.03-1.54)".

Properly interpreted, this finding indicates that women with established heart disease at the start of the trial had a 26% increased risk of developing further cardiac complications if they adopted the USDA dietary guidelines. By showing that postmenopausal women with heart disease were at a lower risk of developing subsequent cardiac complications if they continued to eat more fat and fewer vegetables, fruit and grains, the study essentially disproves the diet-heart hypothesis. For how can a diet designed to prevent heart disease be associated with a worsening of the condition in those who are the most vulnerable because they already have the disease?

As I have described in detail elsewhere,[156,157] this finding was not discussed further in the abstract, the discussion or the conclusions of that paper. In addition, a key line of text was missing from a table.

When challenged to explain these errors and omissions, the authors[158] dismissed the only significant finding in their study as "likely to be a chance finding" because "there is no biological basis for expecting a different outcome in this (ill) subgroup, as shown in cholesterol-lowering trials on women with prior disease". Thus, an inconvenient outcome that the authors disliked was ignored because of their certainty that this adverse result had no currently known biological basis. But this explanation is unacceptable.

For example, the authors failed to reference the Estrogenic Replacement and Atherosclerosis (ERA) Trial, which found that coronary atherosclerosis progressed significantly more rapidly over a three-year period in postmenopausal women eating the equivalent of the WHIDMT low-fat "prudent" diet.[159] A higher carbohydrate intake accelerated coronary artery disease progression, as did the substitution of dietary saturated fat with polyunsaturated fat. In contrast, postmenopausal women eating the most saturated fat, and the least carbohydrates and polyunsaturated fat, showed no progression of coronary atherosclerosis, even though that group included a significantly higher proportion of current smokers. As expected, women eating the most saturated fat also had significantly higher serum HDL cholesterol and lower serum TG concentrations, as well as lower total cholesterol to HDL cholesterol ratios.

These findings, the subject of an accompanying editorial,[160] predict that the clinical manifestation of coronary heart disease should increase in participants in the WHIDMT eating the low-fat "prudent" diet. When Howard et al found this,[153] their responsibility was to explain why the conclusions from the Estrogenic Replacement and Atherosclerosis (ERA) trial were not relevant to their discovery. Instead, they ignored that research, choosing rather to advance their deceptive "biologically implausible" argument.

Eminently plausible biological explanations for this inconvenient finding in the WHIRCDMT would include the favourable changes in blood HDL cholesterol and TG concentrations measured in the ERA trial, together with the evidence that a HFLC diet reduces the blood concentration of small, dense LDL cholesterol particles,[144,161] which, when oxidised[83] or glycated,[88] are considered particularly atherogenic.[80,81,162-165]

The WHIRCDMT also found that although the higher carbohydrate intake of the intervention diet did not influence blood glucose control in women without diabetes, it caused a progressive worsening of control in those with type 2 diabetes mellitus.[166] This finding "agrees with some, but not all, previous studies evaluating the effects of high- and low-carbohydrate diets in persons with diabetes", forcing the authors to conclude that "caution should be exercised in recommending a reduction in overall dietary fat in women with diabetes, unless accompanied by additional recommendations to guide carbohydrate intake". That diets with a high glycaemic load are associated with an increased risk of the development of type 2 diabetes mellitus is well established in the literature.[41,167]

In truth, the authors of both papers should have stated the obvious, namely that their findings indicate that persons with established heart disease or diabetes should be mandated to eat a higher-, not lower-fat, diet, in order to limit further progression of their disease.

A final study confirms this interpretation, at least in type 2 diabetes mellitus. In October 2012, an 11.5-year study on the combined effects of regular exercise and the USDA dietary guidelines in persons with type 2 diabetes mellitus,[168] was terminated as "pointless" when it was established that these interventions were no more effective in slowing the progression of arterial damage than doing nothing. This confirms that type 2 diabetes mellitus is a determined disease that will not be beaten by simple measures, and certainly not by a diet rich in blood glucose and insulin-raising carbohydrates.

In summary, the WHIRCDMT, of which Rossouw was the "project director", has clearly established that eating according to the USDA dietary guidelines is associated with an increased risk of the development of the complications of heart disease and of type 2 diabetes mellitus. Rossouw's findings from the most expensive low-fat diet RCT yet undertaken, fatally damage his favoured diet-heart hypothesis.

## Conclusion

The diet-heart hypothesis has its origins in an associational epidemiological study, and was driven by commercial interests in the absence of evidence from properly designed randomised controlled clinical trials. However, associational studies cannot ever prove causation,[21] regardless of how frequently they are advanced as "definite" evidence. Today, the evidence is clear. Fat in the diet does not relate to the risk of heart disease.[20,53-56,64] Rather, there is accumulating evidence that it is the exposure of susceptible individuals with IR to a high-carbohydrate diet for 10 or more years that produces obesity, diabetes and metabolic syndrome, and through these diseases, to an increased susceptibility to CHD.[71]

It follows that the only way to counter the epidemic increases in all these diseases is to promote the consumption of diets with a reduced carbohydrate content, most especially in those with IR and metabolic syndrome.[113]

On the evidence presented in this article, those who continue to prescribe or to promote "balanced" high- carbohydrate diets to such individuals are guilty of at best, ignorance; at worst, medical negligence. It is only a matter of time before a major class action will be instigated by patients with IR whose health has suffered as a result of following this wholly inappropriate advice.

## Declaration

The personal work on which this review is based is funded by Discovery Health, the University of Cape Town, the Medical Research Council and The National Research Foundation.

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